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Contents

List of Contributors, viii

Foreword, xii

Preface, xiii

Part 1 The Underpinning Science

1 Human Nutrition, 3
DAVID A. BENDER

2 Exercise Physiology, 20
W. LARRY KENNEY, ROBERT MURRAY

3 Biochemistry of Exercise, 36
MICHAEL GLEESON

Part 2 Energy and Macronutrients

4 How to Assess the Energy Costs of Exercise and Sport, 61
BARBARA E. AINSWORTH

5 Energy Balance and Energy Availability, 72
ANNE B. LOUCKS

6 Assessing Body Composition, 88
TIMOTHY R. ACKLAND, ARTHUR D. STEWART

7 Carbohydrate Needs of Athletes in Training, 102
LOUISE M. BURKE

8 The Regulation and Synthesis of Muscle Glycogen by Means of Nutrient Intervention, 113
JOHN L. IVY

9 Carbohydrate Ingestion During Exercise, 126
ASKER JEUKENDRUP

10 Defining Optimum Protein Intakes for Athletes, 136
STUART M. PHILLIPS

11 Dietary Protein as a Trigger for Metabolic Adaptation, 147
LUC J.C. VAN LOON

12 Fat Metabolism During and After Exercise, 156
BENTE KIENS, JACOB JEPPESEN

13 Metabolic Adaptations to a High-Fat Diet, 166
JOHN A. HAWLEY, WEE KIAN YEO

14 Water and Electrolyte Loss and Replacement in Training and Competition, 174
RONALD J. MAUGHAN

15 Performance Effects of Dehydration, 185
ERIC D.B. GOULET

16 Rehydration and Recovery After Exercise, 199
SUSAN M. SHIRREFFS

17 Nutritional Effects on Central Fatigue, 206
BART ROELANDS, ROMAIN MEEUSEN

Part 3 Micronutrients and Dietary Supplements

18 Vitamins, Minerals, and Sport Performance, 217
STELLA L. VOLPE, HA NGUYEN

19 Iron Requirements and Iron Status of Athletes, 229
GIOVANNI LOMBARDI, GIUSEPPE LIPPI, GIUSEPPE BANFI

20 Calcium and Vitamin D, 242
ENETTE LARSON-MEYER

21 Exercise-Induced Oxidative Stress: Are Supplemental Antioxidants Warranted?, 263
JOHN C. QUINDRY, ANDREAS KAVAZIS, SCOTT K. POWERS

22 Dietary Phytochemicals, 277
J. MARK DAVIS, BENJAMIN GORDON, E. ANGELA MURPHY, MARTIN D. CARMICHAEL

23 Risks and Rewards of Dietary Supplement Use by Athletes, 291
RONALD J. MAUGHAN

24 Creatine, 301
FRANCIS B. STEPHENS, PAUL L. GREENHAFF

25 Caffeine and Exercise Performance, 313
LAWRENCE L. SPRIET

26 Buffering Agents, 324
CRAIG SALE, ROGER C. HARRIS

27 Alcohol, Exercise, and Sport, 336
RONALD J. MAUGHAN, SUSAN M. SHIRREFFS

Part 4 Practical Issues

28 The Female Athlete, 347
SUSAN I. BARR

29 The Young Athlete, 359
FLAVIA MEYER, BRIAN W. TIMMONS

30 The Aging Athlete, 369
CHRISTINE A. ROSENBLOOM

31 The Vegetarian Athlete, 382
JACQUELINE R. BERNING

32 The Special Needs Athlete, 392
ELIZABETH BROAD

33 Overreaching and Unexplained Underperformance Syndrome: Nutritional Interventions, 404
PAULA ROBSON-ANSLEY, RICARDO COSTA

34 The Traveling Athlete, 415
SUSIE PARKER-SIMMONS, KYLIE ANDREW

35 Environment and Exercise, 425
SAMUEL N. CHEUVRONT, BRETT R. ELY, RANDALL L. WILBER

36 Food and Nutrition Considerations at Major Competitions, 439
FIONA PELLY

Part 5 Health-Related and Clinical Sports Nutrition

37 Nutrition, Physical Activity, and Health, 455
BARRY BRAUN, BENJAMIN F. MILLER

38 Exercise, Nutrition, and Inflammation, 466
MICHAEL J. KRAAKMAN, MARTIN WHITHAM, MARK A. FEBBRAIO

39 Exercise, Nutrition, and Immune Function, 478
DAVID C. NIEMAN

40 The Diabetic Athlete, 490
GURJIT BHOGAL, NICHOLAS PEIRCE

41 The Overweight Athlete, 503
HELEN O'CONNOR

42 Eating Disorders in Male and Female Athletes, 513
MONICA K. TORSTVEIT, JORUNN SUNDGOT-BORGEN

43 Importance of Gastrointestinal Function to Athletic Performance and Health, 526
NANCY J. REHRER, JOHN MCLAUGHLIN, LUCY K. WASSE

44 Hyponatremia of Exercise, 539
TIMOTHY D. NOAKES

Part 6 Sport-Specific Nutrition: Practical Issues

45 Strength and Power Events, 551
ERIC S. RAWSON, CHARLES E. BRIGHTBILL, MICHAEL J. STEC

46 Sprinting: Optimizing Dietary Intake, 561
GARY SLATER, HELEN O'CONNOR, BETHANIE ALLANSON

47 Distance Running, 572
TRENT STELLINGWERFF

48 Cycling, 584
PETER HESPEL

49 Gymnastics, 596
DAN BENARDOT

50 Swimming, 607
LOUISE M. BURKE, GREGORY SHAW

51 Winter Sports, 619
NANNA L. MEYER

52 Team Sports, 629
FRANCIS HOLWAY

53 Weight-Category Sports, 639
HATTIE H. WRIGHT, INA GARTHE

Index, 651

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Foreword

The publication of the *Encyclopaedia of Sports Medicine*, Volume VII *Nutrition in Sport*, by the IOC Medical Commission in 2000 constituted a milestone in the rapid growth of research on sports nutrition during the last quarter century. Concomitant with this growth has been the increasing body of evidence concerning the important interactive roles of proper nutrition and a balanced program of physical exercise for each person's health and welfare.

Since the appearance of that volume, a large amount of research has appeared in scientific journals concerning the role of nutrition in the training programs of athletes and in their preparation for competition. It is, therefore, timely that this new volume appears in this rapidly expanding field of research and practice.

A highly qualified team of international authorities present a comprehensive coverage of macronutrients, micronutrients, and dietary supplements for the athlete. Extensive coverage is given both to general practical issues and to sports-specific issues.

Professor Ronald J. Maughan has been a frequent contributor to both the *Encyclopaedia of Sports Medicine* series and the *Handbook of Sports Medicine and Science* series as published by the IOC Medical Commission. We welcome with great appreciation his latest publication project related to the science and medicine of sport.

Dr Jacques Rogge
IOC President

Preface

Most of us eat every day, indeed several times every day. What we eat will affect how we feel and how we perform, both in the short term and in the long term. The immediate effects are often small and easily dismissed. However, after only a short period—a few days at most—without food, performance in most tests of physical and mental performance will inevitably decline. Similar effects are seen if some food is allowed but the intake of carbohydrate or water is restricted. It is easy, therefore, to demonstrate the impact of nutrition on athletic performance. Nutrition, though, has many far more subtle effects on the athlete's well-being and performance. A whole range of essential nutrients must be supplied in the right amounts and at the right times if health and performance are to be optimized. Food, and the pleasures as well as the nutrients it gives to the consumer, is a vital part of everyday life. Sports nutrition must therefore be concerned not only with the identification of the athlete's nutritional goals but also with the translation of these goals into an eating strategy that takes account of personal preferences, social and cultural issues, and a whole range of other factors.

The Medical Commission of the International Olympic Committee (IOC) has consistently recognized the importance of nutrition in every aspect of the elite athlete's life. In choosing to commission a new encyclopedia volume on the relationships between diet and performance, the IOC has recognized the great changes that have taken place in our understanding since the publication of the earlier version that appeared three Olympic cycles previously.

A comparison of the content of the two volumes will reveal some constants and some changes. Perhaps the most important change that has taken place in recent years is the recognition that the primary role of nutrition in the athlete's life is to support consistent training and to enhance the process of adaptation that takes place in every tissue of the body in response to each individual training session. It is the sum of these vanishingly small incremental changes that translates into an enhanced performance. Nutrition support is more about promoting those changes rather than simply allowing the athlete to recover more effectively between training sessions and, therefore, to train harder. Training harder undoubtedly brings some benefits in terms of performance improvement, but it also brings increased risks of illness and injury. Training more effectively, rather than just training harder, is surely a better option.

Those who work at the molecular level have given us an understanding of the signaling pathways within cells that modulate gene expression in response to training and diet. This understanding was almost completely absent until very recently, and these new approaches have great promise for identifying strategies that might allow even better performances than those of today's athletes. At the same time, however, there has been a renewed interest in the adaptations that take place at the whole body level, including perhaps especially the links between the brain and the peripheral tissues. The two approaches, the molecular and the whole body, have emphasized the individuality of the response to both diet and exercise and of the need for

individualization and periodization of nutrition strategies to allow athletes to reach their genetic potential.

The contributors to this book are, without exception, world leaders in their fields. Each has given unstintingly of their knowledge and experience in preparing their chapters. The result is a substantial volume, but such is the scope of the science and practice of sports nutrition that even this can be no more than an introduction to the field and an overview of the key issues. Those who seek to advise athletes or whose aspiration is to make the next great advance in our understanding must be prepared to dig much deeper, but perhaps this volume will provide a framework and a reference point for further study.

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PART 1

THE UNDERPINNING SCIENCE

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Chapter 1

Human Nutrition

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The first requirement in human nutrition is for an energy source; the metabolic fuels that provide this are carbohydrates, fats, protein, and alcohol. There is also a need for protein, not only in growth when the total amount of protein in the body is increasing but also throughout life to permit turnover of tissue proteins. In addition, there is a need for some essential fatty acids and for relatively small amounts (milligrams or micrograms per day) of vitamins and minerals.

Energy Nutrition

Even when completely at rest there is a requirement for energy to maintain nerve and muscle tone, circulation and breathing, and metabolic homeostasis. When measured under controlled conditions of thermal neutrality (so that energy is not being expended in keeping warm or cooling down) and completely at rest (but not asleep, since some people increase their metabolic rate when asleep, while others reduce it), this is the basal metabolic rate (BMR). When the measurement is made under less strictly controlled conditions, the result is termed the resting metabolic rate (RMR). BMR depends on body weight, age, and gender and reflects mainly the metabolically more active lean tissues of the body, although adipose tissue makes a modest contribution to BMR. The effect of age on BMR reflects the replacement of muscle tissue by adipose tissue with increasing age (even when body weight remains constant). The gender difference is because women have a greater percentage of body weight as essential and storage adipose tissue than do men. Table 1.1 shows equations for calculating BMR from age, gender, body weight, and height.

The energy cost of different activities is most commonly expressed as a multiple of BMR—the physical activity ratio (PAR) for any given activity. As shown in Table 1.2, PAR ranges from about 1.2× BMR for sedentary activities up to 6× or more times BMR for vigorous exercise, and significantly higher for some sports. Table 1.3 shows the classification of occupational work by PAR over the 8-hour working day, excluding leisure activities.

Summing the PAR for different activities throughout the day, multiplied by the time spent in each activity as a fraction of 24 hours, allows the calculation of a person's physical activity level (PAL), again as a multiple of BMR. A person's total energy expenditure is then (PAL × BMR) + an allowance for diet-induced thermogenesis (DIT)—the energy cost of digestion and absorption, plus the cost of synthesizing glycogen, fat, and protein after a meal. DIT is about 10–15% of the energy yield of a meal. For people with a markedly sedentary lifestyle BMR may represent 80–90% of total energy expenditure.

Measurement of BMR and Energy Expenditure in Activity

The gold standard method of measuring BMR and energy expenditure in an activity is by measurement of heat output from the body. This is done using a calorimeter—an insulated chamber in which a

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Table 1.1 Equations for estimating basal metabolic rate from weight or weight and height, at different ages

	Males		Females	
Age (years)	MJ/day	kcal/day	MJ/day	kcal/day
0–3	$0.2548w - 0.226$ $0.007w + 6.349h - 2.584$	$60.9w - 54$ $1.673w + 1517h - 617$	$0.255w - 0.213$ $0.068w + 4.281h - 1.730$	$61.0w - 51$ $16.252w + 1023h - 413$
3–10	$0.0949w + 2.07$ $0.082w + 0.545h + 1.736$	$22.7w + 495$ $19.59w + 130h + 415$	$0.0941w + 2.09$ $0.071w + 0.677h + 1.5453$	$22.5w + 499$ $16.97w + 161h + 531$
10–17	$0.0732w + 2.72$ $0.068w + 0.574h + 2.157$	$17.5w + 651$ $16.25w + 137h + 516$	$0.0510w + 3.12$ $0.035w + 1.948h + 0.837$	$12.2w + 746$ $8.365w + 465h + 200$
18–29	$0.0640w + 2.84$ $0.063w - 0.042h + 2.953$	$15.3w + 679$ $15.06w + 10.04h + 705$	$0.0615w + 2.08$ $0.057w + 1.184h + 0.411$	$14.7w + 496$ $13.62w + 283h + 98$
30–59	$0.0485w + 3.67$ $0.048w - 0.011h + 3.670$	$11.6w + 879$ $11.47w + 2.629h + 877$	$0.0364w + 3.47$ $0.034w + 0.006h + 3.530$	$8.7w + 829$ $8.126w + 4.434h + 843$
>60	$0.0565w + 2.04$	$13.5w + 487$	$0.0439w + 2.49$	$10.5w + 596$

Source: Data reported by Schofield (1985a, 1985b); recalculated for estimation of BMR in kcal. w, body weight (kg); h, height (m).

Table 1.2 Energy cost of activity, by Physical Activity Ratio (PAR) or multiple of BMR

PAR	
1.0–1.4	Lying, standing, or sitting at rest, e.g., watching TV, reading, writing, eating, playing cards, and board games
1.5–1.8	*Sitting*: sewing, knitting, playing piano, driving *Standing*: preparing vegetables, washing dishes, ironing, general office and laboratory work
1.9–2.4	*Standing*: mixed household chores, cooking, playing snooker or bowls
2.5–3.3	*Standing*: dressing, undressing, showering, making beds, vacuum cleaning *Walking*: 3–4 km/h, playing cricket *Occupational*: tailoring, shoemaking, electrical and machine tool industry, painting and decorating
3.4–4.4	*Standing*: mopping floors, gardening, cleaning windows, table tennis, sailing *Walking*: 4–6 km/h, playing golf *Occupational*: motor vehicle repairs, carpentry and joinery, chemical industry, bricklaying
4.5–5.9	*Standing*: polishing furniture, chopping wood, heavy gardening, volley ball *Walking*: 6–7 km/h *Exercise*: dancing, moderate swimming, gentle cycling, slow jogging *Occupational*: laboring, hoeing, road construction, digging and shoveling, felling trees
6.0–7.9	*Walking*: uphill with load or cross-country, climbing stairs *Exercise*: jogging, cycling, energetic swimming, skiing, tennis, football

Source: Department of Health (1991) and WHO (1985).

Table 1.3 Classification of types of occupational work by PAR (average PAR through 8-hour working day, excluding leisure activities)

	PAR		
	Men	Women	
Light	1.7	1.7	Professional, clerical, and technical workers, administrative and managerial staff, sales representatives, housewives
Moderate	2.7	2.2	Sales staff, domestic service, students, transport workers, joiners, roofing workers
Moderately heavy	3.0	2.3	Machine operators, laborers, agricultural workers, forestry, hunting and fishing, bricklaying, masonry
Heavy	3.8	2.8	Laborers, agricultural workers, bricklaying, masonry where there is little or no mechanization

Source: Department of Health (1991).

constant temperature is maintained by running cold water through pipes and measuring the increase in the temperature of the water. Obviously, the range of activities that can be achieved in a small chamber is limited, and rather than direct calorimetry, most studies use indirect calorimetry, i.e., the measurement of the rate of oxygen consumption (Levine, 2005). As shown in Table 1.4, to first approximation, 1 liter of oxygen consumed is equivalent to 20 kJ of energy expenditure. This means that measurement of oxygen consumption can be used to estimate energy expenditure in a wide range of activities, not confined to the laboratory. If the production of carbon dioxide is also measured then it is possible to calculate the relative amounts of fat, carbohydrate, and protein being metabolized from the respiratory quotient (RQ)—the ratio of carbon dioxide produced to oxygen consumed. When carbohydrate is being oxidized the RQ = 1, while for fat oxidation RQ = 0.707. The amount of protein being metabolized can be calculated separately from the excretion of urea, the end product of amino acid metabolism.

If respirometry includes the measurement of carbon dioxide and oxygen, as well as urinary nitrogen, then it is possible to estimate both energy expenditure and the proportions of different fuels being utilized, from the following formulae (Weir, 1949):

- Energy expenditure (kJ) = 16.849 × ml oxygen consumed + 4.628 × ml carbon dioxide produced – 9.079 × g N excreted
- Energy expenditure (kcal) = 4.025 × ml oxygen consumed + 1.106 × ml carbon dioxide produced – 2.168 × g N excreted

If urinary nitrogen is not determined, and it is assumed that protein provides 15% of energy, then

- Energy expenditure (kJ) = 16.318 × ml oxygen consumed + 4.602 × ml carbon dioxide produced
- Energy expenditure (kcal) = 3.898 × ml oxygen consumed + 1.099 × ml carbon dioxide produced

The amount of each fuel being utilized can be calculated from

- Grams carbohydrate oxidized = 4.706 × ml carbon dioxide produced – 3.340 × ml oxygen consumed – 2.714 × g N excreted

Table 1.4 Oxygen consumption and carbon dioxide production in the oxidation of metabolic fuels

	Energy yield (kJ/g)	Oxygen consumed (l/g)	Carbon dioxide produced (l/g)	Respiratory quotient (CO_2/O_2)	Energy/oxygen consumption (kJ/l oxygen)
Carbohydrate	16	0.829	0.829	1.0	19.3
Protein	17	0.966	0.782	0.809	17.5
Fat	37	2.016	1.427	0.707	18.35

- Grams fat oxidized = 1.768 × ml oxygen consumed − 1.778 × ml carbon dioxide produced − 2.021 × g N excreted
- Grams protein oxidized = 6.25 × g N excreted

Although classical respirometer studies have allowed the measurement of energy expenditure in many activities (and indeed provided the data for PAR shown in Table 1.2), such studies are, of necessity, short term. A more recent development has been the use of double stable isotopically labeled water ($^2H_2{}^{18}O$), which provides a noninvasive method of measuring energy expenditure over a period of 1–3 weeks. As shown in Figure 1.1, the label from ^{18}O is lost from the body faster than that from ^2H. This is because the labeled hydrogen is lost from the body only as water, while the oxygen is lost in both water and carbon dioxide, because of the rapid equilibrium between carbon dioxide and bicarbonate (Heymsfield et al., 2006; Ritz & Coward, 1995; Speakman, 1997).

Following an oral dose of $^2H_2{}^{18}O$, the isotopic enrichment of water in plasma, saliva, or urine is determined at intervals over a period of 10–21 days. The rate of carbon dioxide production is then calculated from the greater rate of loss of ^{18}O than ^2H, as

- Carbon dioxide production rate = (0.5 × total body water) × (rate constant for ^{18}O disappearance − rate constant for ^2H disappearance)

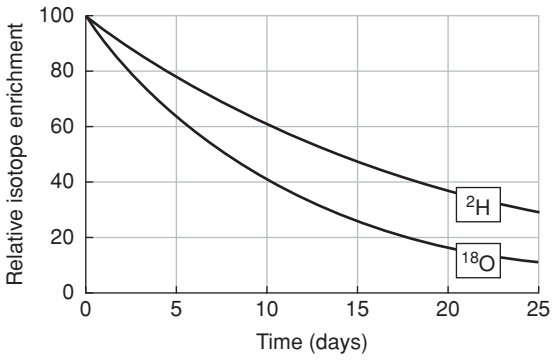

Figure 1.1 Loss of label from $^2H_2{}^{18}O$—the basis of the dual isotopically labeled water method for estimating total energy expenditure.

From records of food eaten, the average RQ over the period can be estimated and hence, allowing for any changes in body weight, the total oxygen consumption and energy expenditure can be calculated.

Recommendations and Reference Levels for Energy Intake

Unlike reference intakes for protein and micronutrients which allow a margin of 2× standard deviation above the observed average requirement, so as to allow for individual variation and cover almost all of the population, reference levels for energy intake (see Table 1.5) are based on average requirements, since adding 2× standard deviation would result in half the population being over-provided with energy, and hence contribute to the development of obesity. At its simplest, energy intake should match energy expenditure and hence, assuming that body weight is within the desirable range (a body mass index of 20–25 kg/m^2), should be such that a constant body weight is achieved.

Table 1.5 Estimated average requirements for energy

	Males		Females	
Age	MJ/day	kcal/day	MJ/day	kcal/day
0–3 months	2.28	545	2.06	515
4–6 months	2.89	690	2.69	645
7–9 months	3.44	825	3.20	765
10–12 months	3.85	920	3.61	865
1–3 years	5.15	1230	4.86	1165
4–6 years	7.16	1715	6.46	1545
7–10 years	8.24	1970	7.28	1740
11–14 years	9.27	2220	7.92	1845
15–18 years	11.51	2755	8.83	2110
19–50 years	10.6	2550	8.10	1940
51–59 years	10.6	2550	8.00	1900
60–64 years	9.93	2380	7.99	1900
65–74 years	9.71	2330	7.96	1900
>75 years	8.77	2100	7.61	1810

Source: Department of Health (1991).

Carbohydrate, Fat, and Protein as Metabolic Fuels

There is no absolute requirement for a dietary source of carbohydrate or fat (apart from essential fatty acids, see below), but there is a need to maintain an adequate supply of glucose for the brain (which is largely dependent on glucose) and red blood cells (which are completely dependent on glucose).

The current consensus (Prentice, 2005) is that carbohydrates should provide about 55% of energy intake for the general population, largely as starches and other complex carbohydrates, with sugars providing no more than about 10% of energy intake. For athletes, it is more common to express daily carbohydrate requirements in absolute terms, as grams of carbohydrate per kilogram of body mass, as this is independent of energy intake which may vary widely. Sugars are divided into intrinsic sugars, contained within the cells of plant foods, and extrinsic sugars in free solution; it is these extrinsic sugars that should be limited, mainly to reduce the risk of dental caries and also because it is easy to overconsume sugars in beverages, etc., leading to obesity. In the United Kingdom, lactose in milk is considered separately from other extrinsic sugars, since it does not contribute to the development of dental caries and milk is an important source of calcium and riboflavin in most diets.

Glucose needs for the brain and red blood cells can be met by gluconeogenesis from amino acids and glycerol in fasting (or when the diet is low in carbohydrate and high in protein), albeit at a relatively high energy cost. The increase in metabolic rate associated with gluconeogenesis explains much of the weight loss associated with very low carbohydrate diets that permit more or less unlimited consumption of protein-rich foods.

When the diet provides less than about 15% of energy from fat, it is difficult to eat a sufficient volume of food to meet energy requirements. Conversely, when the diet provides more than about 35% of energy from fat it is easy to overconsume food, leading to obesity. More importantly, a high fat intake leads to persistence of atherogenic chylomicron remnants in the bloodstream, a contributory factor in atherosclerosis and coronary artery disease. The current consensus is that fat should provide 30% of energy intake for the general population (Prentice, 2005), though the fraction may be higher or lower for athletes, depending on the training load and therefore on the energy demand. At very low fat intakes, it is difficult to absorb the fat-soluble vitamins A, D, E, and K, which are absorbed in lipid micelles together with the products of dietary fat digestion.

The type of dietary fat is also important. Figure 1.2 shows the families of fatty acids and Table 1.6, the main dietary fatty acids. There is a convenient shorthand notation for fatty acids showing the number of carbon atoms, and the number of double bonds and the position of the first double bond from the methyl group as either *n*–3, 6, or 9 or ω3, 6, or 9.

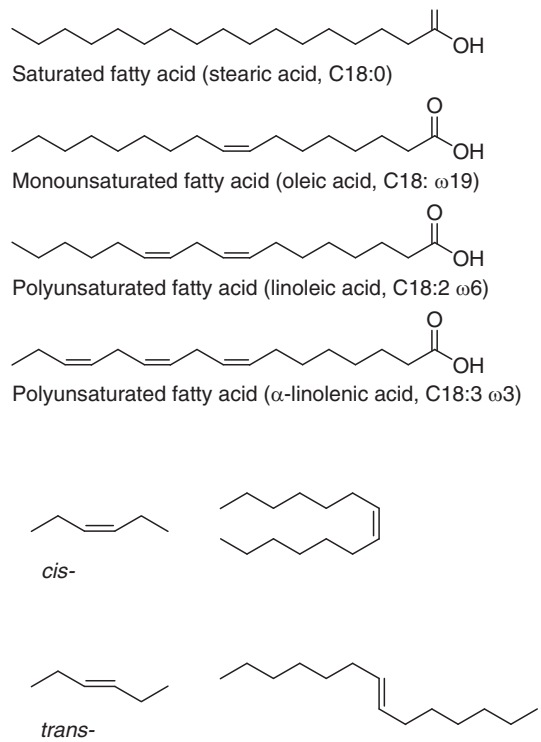

Figure 1.2 The families of fatty acids and *cis–trans* isomerism in unsaturated fatty acids.

Table 1.6 Fatty acid nomenclature

		Double bonds		
	C atoms	Number	First	Shorthand
Saturated				
Butyric	4	0	–	C4:0
Caproic	6	0	–	C6:0
Caprylic	8	0	–	C8:0
Capric	10	0	–	C10:0
Lauric	12	0	–	C12:0
Myristic	14	0	–	C14:0
Palmitic	16	0	–	C16:0
Stearic	18	0	–	C18:0
Arachidic	20	0	–	C20:0
Behenic	22	0	–	C22:0
Lignoceric	24	0	–	C24:0
Monounsaturated				
Palmitoleic	16	1	7	C16:1 *ω7*
Oleic	18	1	9	C18:1 *ω9*
Nervonic	24	1	9	C24:1 *ω9*
Polyunsaturated				
Linoleic	18	2	6	C18:2 *ω6*
α-Linolenic	18	3	3	C18:3 *ω3*
γ-Linolenic	18	3	6	C18:3 *ω6*
Arachidonic	20	4	6	C20:4 *ω6*
Eicosapentaenoic	20	5	3	C20:5 *ω3*
Docosatetraenoic	22	4	6	C22:4 *ω6*
Docosapentaenoic	22	5	3	C22:5 *ω3*
Docosapentaenoic	22	5	6	C22:5 *ω6*
Docosahexaenoic	22	6	3	C22:6 *ω3*

High intakes of saturated fatty acids lead to an increase in low-density lipoprotein (LDL) cholesterol and are therefore a major factor in atherogenesis. Compared with monounsaturated fatty acids, saturated fatty acids lead to an increase in LDL cholesterol proportional to twice the intake. Polyunsaturated fatty acids lead to a decrease in LDL cholesterol proportional to their intake (Anderson et al., 1957; Armstrong et al., 1957; Hegsted et al., 1965, 1993; Keys et al., 1957). This is because saturated fatty acids are poor substrates for esterification of cholesterol, while monounsaturated are better, and polyunsaturated are the best substrates. Stearic acid (C18:0), although saturated, has less adverse effect on LDL cholesterol than other saturated fatty acids because it is readily unsaturated to oleic acid (C18:1 *ω9*). It is recommended that no more than one-third of fat intake (10% of energy intake) should be from saturated fatty acids, with 6% from polyunsaturated fatty acids.

Trans-isomers of unsaturated fatty acids arise during the catalytic hydrogenation of vegetable oils to yield spreadable fats, and also occur in modest amounts in fats from ruminants. They do not have the same beneficial effect on LDL cholesterol as do the *cis*-isomers, but are atherogenic, and may adversely affect the fluidity of cell membranes. It is therefore recommended that *trans*-fatty acids should provide less than 2% of energy intake (British Nutrition Foundation, 1995).

As shown in Figure 1.2, there are three families of unsaturated fatty acids, with the first double bond at the *ω3*, *ω6*, or *ω9* position. Human beings have an enzyme that can introduce a double bond into a saturated fatty acid at the *ω9* position and enzymes that can introduce double bonds between *ω3* or *ω6* and the carboxyl group, but not between *ω9* and the methyl group. This means that there is a requirement for a dietary source of both *ω3* and *ω6* polyunsaturated fatty acids, which are precursors of prostaglandins and other eicosanoids that acts as signaling molecules. These are the essential fatty acids—linoleic and linolenic acids, which can undergo chain elongation and further desaturation in the body. The same enzymes are involved in the chain elongation, desaturation, and onward metabolism to eicosanoids for both *ω3* and *ω6* polyunsaturated fatty acids, and the balance between the two families of fatty acids in the diet, is important.

In addition to the requirement for protein *per se*, protein must also be considered as a metabolic fuel. For an adult in nitrogen balance, whose total body protein content is constant, an amount of amino acids equivalent to the dietary intake of protein will be metabolized each day, as an energy source. The metabolism of amino acids is less efficient than that of carbohydrates, since there are a number of

thermogenic steps in which ATP is synthesized and then consumed (Bender, 2012) but, nevertheless, protein can be a factor in the development of obesity if total energy intake is greater than expenditure. If carbohydrate is to provide 55% of energy and fat 30%, the recommendation is that protein should provide 14–15% of energy (with alcohol, if consumed, providing about 1%). This is almost twice the requirement for protein turnover. Athletes with a high energy intake can achieve an adequate protein intake even if protein accounts for even a much lower fraction of total energy intake.

Protein Requirements

For an adult whose total body protein content is constant, the intake of nitrogenous compounds (mainly protein) in the diet will be equal to the urinary and fecal losses of nitrogenous compounds. This is nitrogen (N) balance or equilibrium. If the dietary protein intake is inadequate then the losses of N will be greater than the intake—negative N balance and a net loss of body protein. This also occurs in response to a variety of pathological conditions. In growth, pregnancy, and recovery from losses there will be positive N balance—excretion is less than intake and there is an increase in total body protein.

The determination of protein requirements remains controversial. Apart from times of growth and recovery from losses, N balance can be maintained at any level of protein intake above the requirement. In principle, it is easy to measure N balance in people maintained at different levels of protein intake and so determine their requirement. The problem is that the rates of protein turnover and catabolism of amino acids change with protein intake, so that apparent protein requirements are influenced by prior protein intake. This means that studies of N balance require a long period of adaptation at each level of intake.

Reexamination of the results of N balance studies led to the 2007 WHO/FAO/UNU report (WHO, 2007) that increased the average requirement for protein by 10%, from the previously accepted figure of 0.6 g of protein/kg body weight/day to 0.66. Allowing for individual variation in requirements, a safe level of protein intake is set at 0.8 g/kg body weight or 56 g/day for a 70 kg adult. Safe here means safe and (more than) adequate to prevent deficiency and does not imply that higher levels of intake are unsafe, although there is some evidence that exceptionally high levels of habitual protein intake are associated with bone and kidney disease.

The safe level of protein intake is equivalent to 8–9% of energy from protein, so that people in western countries consuming the recommended 14–15% of energy from protein are more than adequately supplied. However, the 10% increase in the estimated average requirement of the 2007 report greatly increases the number of people in developing countries whose protein intake is deemed marginal or inadequate.

Protein Quality and Amino Acid Requirements

The need for protein is not just for total protein, but for an intake of amino acids in the amounts required for body protein synthesis and turnover. Classical studies of the amounts of individual amino acids required to maintain N balance in the 1950s and 1960s established that 8 of the 20 amino acids found in body proteins are dietary essentials and cannot be synthesized in the body. These essential or indispensable amino acids are isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine. It was not until 1975 that histidine was also recognized to be an essential amino acid; for reasons that are unclear, it is possible to maintain N balance on a histidine-free diet for at least a week. These early studies permitted determination of the amounts of each essential amino acid needed to maintain N balance, as shown in Table 1.7.

More recent studies have attempted to determine the requirements for essential amino acids using amino acids labeled with the stable isotopes ^{15}N or ^{13}C. One approach is to measure the incorporation of the labeled amino acid of interest into body proteins, and its subsequent catabolism and the excretion of $^{13}CO_2$ or ^{15}N urea. The problem with this direct approach is that the amount of stable isotopically labeled amino acid that has to be administered in order to achieve adequate

Table 1.7 Reference patterns of essential amino acids

	From N balance studies		From stable isotope studies	
	mg/kg bw/day	mg/g protein	mg/kg bw/day	mg/g protein
Histidine	–	–	10	15
Isoleucine	10	15	20	30
Leucine	14	21	39	59
Lysine	12	18	30	45
Methionine + cysteine	13	20	15	22
Methionine	–	–	10	16
Cysteine	–	–	4	6
Phenylalanine + tyrosine	14	21	25	38
Threonine	7	11	15	23
Tryptophan	3.5	5	4	6
Valine	10	15	26	39
Total essential amino acids	93.5	141	184	277

Source: WHO (2007).

sensitivity is so large that it distorts the body pool of the amino acid and so overestimates the apparent requirement.

The alternative approach is the indicator amino acid method, in which varying amounts of the amino acid of interest are fed and the catabolism of a different essential amino acid (the indicator amino acid) is measured. The principle here is that when the amino acid of interest has been depleted then all of the remaining indicator amino acid will be catabolized, since it cannot be used for further protein synthesis. Estimates of essential amino acid requirements by isotope tracer methods are shown in Table 1.7.

The essential amino acid that is present in dietary protein in the least amount compared with the requirement for body protein synthesis is termed the limiting amino acid. Once supplies of this amino acid have been exhausted, protein synthesis comes to a halt and the remaining amino acids are catabolized as metabolic fuel.

Two of the amino acids can only be synthesized in the body from essential precursors: tyrosine from phenylalanine and cysteine from methionine. Providing these in the diet thus spares the requirement for the parent amino acid. This is especially important in the case of cysteine and methionine since, in many diets, it is the sum of these two amino acids that is limiting.

The remaining amino acids are generally considered to be nonessential or dispensable, since they can be synthesized in the body from more or less common metabolic intermediates. However, only three amino acids can be considered to be completely dispensable since they are synthesized from ubiquitous intermediates of carbohydrate metabolism: alanine from pyruvate, glutamate from 2-oxoglutarate, and aspartate from oxaloacetate. The remaining amino acids (arginine, asparagine, glutamine, glycine, proline, and serine) must all be considered to be semi-essential, in that under conditions of metabolic stress or rapid growth the capacity for their synthesis may be inadequate to meet requirements.

The nutritional value or quality of individual proteins depends on whether or not they contain the essential amino acids in the amounts that are required. A number of different ways of determining protein quality have been developed:

- Biological value (BV) is the proportion of absorbed protein that is retained in the body. A

protein that is completely useable (e.g., egg and human milk) has a BV of 0.9–1; meat and fish have a BV of 0.75–0.8; wheat protein has a BV of 0.5; gelatin (which completely lacks tryptophan) has a BV of 0.

- Net protein utilization (NPU) is the proportion of dietary protein that is retained in the body (i.e., it takes account of the digestibility of the protein). By convention, it is measured at 10% dietary protein, at which level the experimental animal can utilize all of the protein as long as the balance of essential amino acids is correct.
- Protein efficiency ratio (PER) is the gain in weight of growing animals per gram of protein eaten.
- Relative protein value (RPV) is the ability of a test protein, fed at various levels of intake, to support nitrogen balance, compared with a standard protein.
- Chemical score is based on chemical analysis of the amino acids present in the protein; it is the amount of the limiting amino acid compared with the amount of the same amino acid in egg protein (which is completely useable for tissue protein synthesis).
- Protein score (or amino acid score) is again based on chemical analysis, but uses a reference pattern of amino acid requirements as the standard. This provides the basis of the legally required way of expressing protein quality in the United States—the protein digestibility-corrected amino acid score (PDCAAS).

All of these measures of protein quality suffer from two problems in practical nutrition:

1. No one eats a single food as their only protein source. While individual vegetable proteins may have a low BV, cereals are generally limited by lysine (and hence have a relative excess of the sulfur amino acids methionine + cysteine), while legume proteins are limited by the sulfur amino acids and hence have a relative excess of lysine. There is complementation between the amino acids in different proteins in a meal, and a judicious mixture of cereals and legumes can have a BV as high as that of meat. Worldwide there is very little difference in protein quality between the "best" diets in developed countries and the "worst" in developing countries.
2. The quality of the dietary protein is only important when the total protein intake is marginal. If the total amount of protein consumed is significantly greater than requirements then the quality of that protein is irrelevant.

Micronutrients: Vitamins and Minerals

Minerals

Any chemical element that has a metabolic or other function in the body is obviously a dietary essential, since elements cannot be interconverted. Table 1.8 shows the minerals that are known to be dietary essentials, classified by their functions. Some minerals appear under more than one heading, since they have multiple functions in the body. There is a small group of minerals (silicon, vanadium, nickel, and tin) that are known to be dietary

Table 1.8 Essential minerals classified by their function

Structural function	Calcium, magnesium, phosphate
Involved in membrane function	Sodium, potassium
Function as prosthetic groups in enzymes	Cobalt, copper, iron, molybdenum, selenium, zinc
Regulatory role or role in hormone action	Calcium, chromium, iodine, magnesium, manganese, sodium, potassium
Known to be essential, but function unknown	Silicon, vanadium, nickel, tin
Have effects in the body, but essentiality is not established	Fluoride, lithium
May occur in foods and known to be toxic in excess	Aluminum, arsenic, antimony, boron, bromine, cadmium, cesium, germanium, lead, mercury, silver, strontium

essentials for experimental animals maintained on highly purified diets, but whose metabolic function is unknown. These ultra-trace minerals are not of practical importance in human nutrition. Lithium salts are known to have a pharmacological effect in the treatment of bipolar psychiatric disease, and fluoride is known to improve bone health and reduce dental caries, but neither can be considered to be a dietary essential.

Most minerals are required in milligram or microgram amounts daily to match losses from the body. In principle, it is easy to determine mineral requirements by balance studies—how much is required to replace urinary and fecal losses? In practice, it is less simple. Negative calcium balance may be the result of bone loss (osteoporosis) in old age, and increasing calcium intake may not restore balance, but simply lead to higher intake and output, but still with negative balance.

Iron deficiency anemia is a major problem of public health worldwide and, together with iodine and vitamin A, is one of the WHO's three micronutrient priorities. The absorption of dietary iron is strictly controlled by the state of body iron reserves, because there is no mechanism for iron excretion. However, excessive blood losses (as in menstruation or as a result of intestinal parasites) lead to requirements for replacement that cannot readily be met from the diet. As a result of menstrual blood losses, most women between menarche and menopause have negligible body iron reserves, while men have relatively large reserves. Postmenopausally, women's iron reserves increase, approaching those of men. A further problem of iron nutrition is that perhaps 10% of the population (and more in some ethnic groups) are at risk of iron overload because of genetic polymorphisms in the various enzymes and proteins involved in iron homeostasis. Iron overload (hemochromatosis) leads to liver cirrhosis, cardiomyopathy, and pancreatic damage (bronze diabetes) and can also lead to depletion of vitamin C as a result of nonenzymic reactions, and hence the development of scurvy (Institute of Medicine, 2001).

Iodine is required for the synthesis of the thyroid hormones, thyroxine and tri-iodothyronine. Deficiency, leading to goiter (a visible enlargement of the thyroid gland), is widespread in inland upland areas over limestone soil. This is because the soil over limestone is thin, and minerals, including iodine, readily leach out, so that locally grown plants are deficient in iodine. Near the coast, sea spray contains enough iodine to replace these losses. Worldwide, many millions of people are at risk of deficiency, and in parts of central Brazil, the Himalayas, and central Africa, goiter may affect more than 90% of the population. A contributory problem, in addition to low dietary iodine, may be the presence of goitrogens (compounds that interfere with iodine metabolism) in some foods. Thyroid hormones regulate metabolic activity, and people with thyroid deficiency have a low metabolic rate, and hence gain weight readily. They tend to be lethargic and have a dull mental apathy. Children born to iodine-deficient mothers are especially at risk, and more so if they are then weaned onto an iodine-deficient diet. They may suffer from very severe mental retardation (goitrous cretinism) and congenital deafness (Institute of Medicine, 2001).

Apart from iron and iodine, mineral deficiencies are likely to be a problem only for people whose food comes entirely or largely from a limited region where the soil may be deficient. For people whose food comes from a number of different regions of the world, mineral deficiencies are relatively uncommon. Selenium intake in the United Kingdom has fallen over the last three decades as a result of increasing use of wheat grown in Europe, where soils are relatively poor in selenium, compared with earlier use of wheat from Australia and North America, where soils contain more selenium. Indeed, in some parts of the United States soils contain so much selenium that grazing livestock suffer from selenium poisoning (Rayman, 1997, 2000).

The availability of minerals from foods also presents a problem. Requirements are estimated from balance and other studies, using crystalline mineral salts. However, while chemical analysis reveals the content of a mineral in a food, much of this may not be available for absorption. Interactions between different foods can also affect the availability of minerals for absorption. Tannins in tea chelate iron

and phytates in unleavened breads chelate calcium and zinc, reducing their absorption.

Vitamins

Vitamins are organic compounds (and hence distinct from minerals) that are required in the diet in small amounts (milligrams or micrograms daily, as opposed to essential amino and fatty acids, which are required in gram amounts) for the maintenance of normal metabolic integrity and homeostasis. In order to be considered a vitamin, a compound must be shown to be a dietary essential and have a metabolic function; deprivation must lead to more or less specific deficiency signs that are reversed by restoring the vitamin to the diet. Table 1.9 shows the vitamins and their principal metabolic functions and deficiency signs.

For a vitamin or any other nutrient, there is a range of intakes between that which is clearly

Table 1.9 The vitamins

Vitamin		Functions	Deficiency disease
A	Retinol, β-carotene	Visual pigments in the retina, regulation of gene expression, and cell differentiation	Night blindness, xerophthalmia, keratinization of skin
D	Calciferol	Maintenance of calcium balance, enhances intestinal absorption of Ca^{2+}, and mobilizes bone mineral	Rickets = poor mineralization of bone, osteomalacia = bone demineralization
E	Tocopherols, tocotrienols	Antioxidant, especially in cell membranes	Extremely rare—serious neurological dysfunction
K	Phylloquinone, menaquinones	Coenzyme in the formation of γ-carboxyglutamate in proteins of blood clotting and bone matrix	Impaired blood clotting, hemorrhagic disease
B_1	Thiamin	Coenzyme in pyruvate and 2-oxoglutarate dehydrogenases and transketolase, role in nerve conduction	Peripheral nerve damage (beriberi) or central nervous system lesions (Wernicke–Korsakoff syndrome)
B_2	Riboflavin	Coenzyme in oxidation and reduction reactions, prosthetic group of flavoproteins	Lesions of corner of mouth, lips, and tongue, seborrheic dermatitis
Niacin	Nicotinic acid, nicotinamide	Coenzyme in oxidation and reduction reactions, functional part of NAD and NADP	Pellagra—photosensitive dermatitis, depressive psychosis
B_6	Pyridoxine, pyridoxal, pyridoxamine	Coenzyme in transamination and decarboxylation of amino acids and glycogen phosphorylase, role in steroid hormone action	Disorders of amino acid metabolism, convulsions
	Folic acid	Coenzyme in transfer of one-carbon fragments	Megaloblastic anemia
B_{12}	Cobalamin	Coenzyme in transfer of one-carbon fragments and metabolism of folate	Pernicious anemia = megaloblastic anemia with degeneration of the spinal cord
	Pantothenic acid	Functional moiety of CoA and acyl carrier protein in fatty acid metabolism and synthesis	Peripheral nerve damage (burning foot syndrome)
H	Biotin	Coenzyme in carboxylation reactions in gluconeogenesis and fatty acid synthesis	Impaired fat and carbohydrate metabolism, dermatitis
C	Ascorbic acid	Coenzyme in hydroxylation of proline and lysine in collagen synthesis, antioxidant, enhances absorption of iron	Scurvy—impaired wound healing, loss of dental cement, subcutaneous hemorrhage

inadequate, leading to clinical deficiency disease, and that which is so much in excess of the body's metabolic capacity that there may be signs of toxicity. Any excess of the water-soluble vitamins is generally excreted in the urine, but the fat-soluble vitamins may accumulate in tissues with harmful consequences. Between these two extremes is a level of intake that is adequate for normal health and the maintenance of metabolic integrity, and a series of more precisely definable levels of intake that are adequate to meet specific criteria, and may be used to determine requirements and appropriate levels of intake. In order of decreasing severity, or increasing sensitivity as markers of adequacy, these are

- Clinical deficiency disease, with clear anatomical and functional lesions, and severe metabolic disturbances, possibly proving fatal. Prevention of deficiency disease is a minimal goal in determining requirements.
- Covert deficiency, where there are no signs of deficiency under normal conditions, but any trauma or stress reveals the precarious state of the body reserves and may precipitate clinical signs. For example, an intake of 10 mg of vitamin C per day is adequate to prevent clinical deficiency, but at least 20 mg per day is required for healing of wounds.
- Metabolic abnormalities under normal conditions, such as impaired carbohydrate metabolism in thiamin deficiency or excretion of methylmalonic acid in vitamin B_{12} deficiency.
- Abnormal response to a metabolic load, such as the inability to metabolize a test dose of histidine in folate deficiency or tryptophan in vitamin B_6 deficiency, although at normal levels of intake there may be no metabolic impairment.
- Inadequate saturation of enzymes with (vitamin-derived) coenzymes—this can be tested for three vitamins, using red blood cell enzymes: thiamin, riboflavin, and vitamin B_6.
- Low plasma concentration of the nutrient, indicating that there is an inadequate amount in tissue reserves to permit normal transport between tissues. For some nutrients, such as vitamin A, this may reflect failure to synthesize a transport protein rather than deficiency of the nutrient itself.
- Low urinary excretion of the nutrient, reflecting low intake and changes in metabolic turnover.
- Incomplete saturation of body reserves.
- Adequate body reserves and normal metabolic integrity.
- Possibly beneficial effects of intakes that are more than adequate to meet requirements—the promotion of optimum health and life expectancy.

Having decided on an appropriate criterion of adequacy, requirements are determined by feeding volunteers an otherwise adequate diet, but lacking the nutrient under investigation, until there is a detectable metabolic or other abnormality. They are then repleted with graded intakes of the nutrient until the abnormality is just corrected. Problems arise in interpreting the results, and therefore defining requirements, when different markers of adequacy respond to different levels of intake. This explains the difference in the tables of reference intakes published by different national and international authorities (see Tables 1.10, 1.11, 1.12, and 1.13).

Dietary Reference Values

Individuals do not all have the same requirement for nutrients, even when calculated on the basis of body size or energy expenditure. There is a range of individual requirements of up to 25% around the mean. Therefore, in order to set population goals and assess the adequacy of diets, it is necessary to set a reference level of intake that is high enough to ensure that no one will either suffer from deficiency or be at risk of toxicity.

As shown in the upper graph in Figure 1.3, if it is assumed that individual requirements are normally distributed around the observed average requirement, then a range of $\pm 2 \times$ the standard deviation (SD) around the mean will include the requirements of 95% of the population. This 95% range is conventionally used as the "normal" or reference range (e.g., in clinical chemistry to assess the normality or otherwise of a test result) and is used to define three levels of nutrient intake:

Table 1.10 Reference nutrient intakes of vitamins and minerals, United Kingdom, 1991

Age	Vit B_1 (mg)	Vit B_2 (mg)	Niacin (mg)	Vit B_6 (mg)	Vit B_{12} (μg)	Folate (μg)	Vit C (mg)	Vit A (μg)	Vit D (μg)	Ca (mg)	P (mg)	Mg (mg)	Fe (mg)	Zn (mg)	Cu (mg)	Se (μg)	I (μg)
0–3 months	0.2	0.4	3	0.2	0.3	50	25	350	8.5	525	400	55	1.7	4.0	0.2	10	50
4–6 months	0.2	0.4	3	0.2	0.3	50	25	350	8.5	525	400	60	4.3	4.0	0.3	13	60
7–9 months	0.2	0.4	4	0.3	0.4	50	25	350	7	525	400	75	7.8	5.0	0.3	10	60
10–12 months	0.3	0.4	5	0.4	0.4	50	25	350	7	525	400	80	7.8	5.0	0.3	10	60
1–3 years	0.5	0.6	8	0.7	0.5	70	30	400	7	350	270	85	6.9	5.0	0.4	15	70
4–6 years	0.7	0.8	11	0.9	0.8	100	30	500	–	450	350	120	6.1	6.5	0.6	20	100
7–10 years	0.7	1.0	12	1.0	1.0	150	30	500	–	550	450	200	8.7	7.0	0.7	30	110
Males																	
11–14 years	0.9	1.2	15	1.2	1.2	200	35	600	–	1000	775	280	11.3	9.0	0.8	45	130
15–18 years	1.1	1.3	18	1.5	1.5	200	40	700	–	1000	775	300	11.3	9.5	1.0	70	140
19–50 years	1.0	1.3	17	1.4	1.5	200	40	700	–	700	550	300	8.7	9.5	1.2	75	140
50+ years	0.9	1.3	16	1.4	1.5	200	40	700	10	700	550	300	8.7	9.5	1.2	75	140
Females																	
11–14 years	0.7	1.1	12	1.0	1.2	200	35	600	–	800	625	280	14.8	9.0	0.8	45	130
15–18 years	0.8	1.1	14	1.2	1.5	200	40	600	–	800	6254	300	14.8	7.0	1.0	60	140
19–50 years	0.8	1.1	13	1.2	1.5	200	40	600	–	700	550	270	14.8	7.0	1.2	60	140
50+ years	0.8	1.1	12	1.2	1.5	200	40	600	10	700	550	270	8.7	7.0	1.2	60	140
Pregnant	+0.1	+0.3	–	–	–	+100	+10	+100	10	–	–	–					
Lactating	+0.1	+0.5	+2	–	+0.5	+60	+30	+350	10	+550	+440	+50		+6.0	+0.3	+15	

Source: Department of Health (1991).

Table 1.11 Population reference intakes of vitamins and minerals, European Union, 1993

Age	Vit A (μg)	Vit B_1 (mg)	Vit B_2 (mg)	Niacin (mg)	Vit B_6 (mg)	Folate (μg)	Vit B_{12} (μg)	Vit C (mg)	Ca (mg)	P (mg)	Fe (mg)	Zn (mg)	Cu (mg)	Se (μg)	I (μg)
6–12 months	350	0.3	0.4	5	0.4	50	0.5	20	400	300	6	4	0.3	8	50
1–3 years	400	0.5	0.8	9	0.7	100	0.7	25	400	300	4	4	0.4	10	70
4–6 years	400	0.7	1.0	11	0.9	130	0.9	25	450	350	4	6	0.6	15	90
7–10 years	500	0.8	1.2	13	1.1	150	1.0	30	550	450	6	7	0.7	25	100
Males															
11–14 years	600	1.0	1.4	15	1.3	180	1.3	35	1000	775	10	9	0.8	35	120
15–17 years	700	1.2	1.6	18	1.5	200	1.4	40	1000	775	13	9	1.0	45	130
18+ years	700	1.1	1.6	18	1.5	200	1.4	45	700	550	9	9.5	1.1	55	130
Females															
11–14 years	600	0.9	1.2	14	1.1	180	1.3	35	800	625	18	9	0.8	35	120
15–17 years	600	0.9	1.3	14	1.1	200	1.4	40	800	625	17	7	1.0	45	130
18+ years	600	0.9	1.3	14	1.1	200	1.4	45	700	550	16[a]	7	1.1	55	130
Pregnant	700	1.0	1.6	14	1.3	400	1.6	55	700	550	–[a]	7	1.1	55	130
Lactating	950	1.1	1.7	16	1.4	350	1.9	70	1200	950	16	12	1.4	70	160

Source: Scientific Committee for Food (1993).
[a]8 mg Fe postmenopausally; supplements required in second half of pregnancy.

Table 1.12 Recommended dietary allowances and acceptable intakes for vitamins and minerals, United States and Canada, 1997–2011

Age	Vit A (μg)	Vit D (μg)	Vit E (mg)	Vit K (μg)	Vit B_1 (mg)	Vit B_2 (mg)	Niacin (mg)	Vit B_6 (mg)	Folate (μg)	Vit B_{12} (μg)	Vit C (mg)	Ca (mg)	P (mg)	Fe (mg)	Zn (mg)	Cu (mg)	Se (μg)	I (μg)
0–6 months	400	–	4	2.0	0.2	0.3	2	0.1	65	0.4	40	–	100	–	2.0	200	15	110
7–12 months	500	–	5	2.5	0.3	0.4	4	0.3	80	0.5	50	–	275	11	3	220	20	130
1–3 years	300	15	6	30	0.5	0.5	6	0.5	150	0.9	15	700	460	7	3	340	20	90
4–8 years	400	15	7	55	0.5	0.6	8	0.6	200	1.2	25	1000	500	10	5	440	30	90
Males																		
9–13 years	600	15	11	60	0.9	0.9	12	1.0	300	1.8	45	1300	1250	8	8	700	40	120
14–18 years	900	15	15	75	1.2	1.3	16	1.3	400	2.4	75	1300	1250	11	11	890	55	150
19–30 years	900	15	15	120	1.2	1.3	16	1.3	400	2.4	90	1000	700	8	11	900	55	150
31–50 years	900	15	15	120	1.2	1.3	16	1.3	400	2.4	90	1000	700	8	11	900	55	150
51–70 years	900	15	15	120	1.2	1.3	16	1.7	400	2.4	90	1000	700	8	11	900	55	150
>70 years	900	20	15	120	1.2	1.3	16	1.7	400	2.4	90	1200	700	8	11	900	55	150
Females																		
9–13 years	600	15	11	60	0.9	0.9	12	1.0	300	1.8	45	1300	1250	8	8	700	40	120
14–18 years	700	15	15	75	1.0	1.0	14	1.2	400	2.4	65	1300	1250	15	9	890	55	150
19–30 years	700	15	15	90	1.1	1.1	14	1.3	400	2.4	75	1000	700	18	8	900	55	150
31–50 years	700	15	15	90	1.1	1.1	14	1.3	400	2.4	75	1000	700	18	8	900	55	150
51–70 years	700	15	15	90	1.1	1.1	14	1.5	400	2.4	75	1200	700	8	8	900	55	150
>70 years	700	20	15	90	1.1	1.1	14	1.5	400	2.4	75	1200	700	8	8	900	55	150
Pregnant	770	15	15	90	1.4	1.4	18	1.9	600	2.6	85	1000–1300	700	27	11	1000	60	220
Lactating	900	15	16	90	1.4	1.6	17	2.0	500	2.8	120	1000–1300	700	9	12	1300	70	290

Source: Institute of Medicine (1997, 1998, 2000, 2001, 2011).
Figures for infants under 12 months are adequate intakes, based on the observed mean intake of infants fed principally on breast milk; for nutrients other than vitamin K, figures are RDA, based on estimated average requirement +2 sd; figures for vitamin K are adequate intakes, based on observed average intakes.

Table 1.13 Recommended nutrient intakes for vitamins

Age	Vit A (μg)	Vit D (μg)	Vit K (μg)	Vit B_1 (mg)	Vit B_2 (mg)	Niacin (mg)	Vit B_6 (mg)	Folate (μg)	Vit B_{12} (μg)	Vit C (mg)	Panto (mg)	Biotin (μg)
0–6 months	375	5	5	0.2	0.3	2	0.1	80	0.4	25	1.7	5
7–12 months	400	5	10	0.3	0.4	4	0.3	80	0.5	30	1.8	6
1–3 years	400	5	15	0.5	0.5	6	0.5	160	0.9	30	2.0	8
4–6 years	450	5	20	0.6	0.6	8	0.6	200	1.2	30	3.0	12
7–9 years	500	5	25	0.9	0.9	12	1.0	300	1.8	35	4.0	20
Males												
10–18 years	600	5	35–55	1.2	1.3	16	1.3	400	2.4	40	5.0	30
19–50 years	600	5	65	1.2	1.3	16	1.3	400	2.4	45	5.0	30
50–65 years	600	10	65	1.2	1.3	16	1.7	400	2.4	45	5.0	30
>65 years	600	15	65	1.2	1.3	16	1.7	400	2.4	45	5.0	30

Age	Vit A (μg)	Vit D (μg)	Vit K (μg)	Vit B_1 (mg)	Vit B_2 (mg)	Niacin (mg)	Vit B_6 (mg)	Folate (μg)	Vit B_{12} (μg)	Vit C (mg)	Panto (mg)	Biotin (μg)
Females												
10–18 years	600	5	35–55	1.1	1.0	16	1.2	400	2.4	40	5.0	25
19–50 years	600	5	55	1.1	1.1	14	1.3	400	2.4	45	5.0	30
50–65 years	600	10	55	1.1	1.1	14	1.5	400	2.4	45	5.0	30
>65 years	600	15	55	1.1	1.1	14	1.5	400	2.4	45	5.0	30
Pregnant	800	5	55	1.4	1.4	18	1.9	600	2.6	55	6.0	30
Lactating	850	5	55	1.5	1.6	17	2.0	500	2.8	70	7.0	35

Source: FAO/WHO (2001).

- Estimated average requirement (EAR)—This is the observed mean requirement to meet the chosen criterion of adequacy in experimental studies.
- Reference nutrient intake (RNI)—This is 2× SD above the observed mean requirement and is therefore more than adequate to meet the individual requirements of 97.5% of the population. This is the goal for planning diets, e.g., in institutional feeding, and the standard against which the intake of a population can be assessed. In the European Union tables (Table 1.11), this is called the population reference intake (PRI); in the United States, it is called the recommended dietary allowance (RDA, Table 1.12).
- Lower reference nutrient intake (LNRI)—This is 2× sd below the observed mean requirement and is therefore adequate to meet the requirements of only 2.5% of the population. In the European Union tables, this is called the lower threshold intake, to stress that it is a level of intake at or below which it is extremely unlikely that normal metabolic integrity could be maintained.

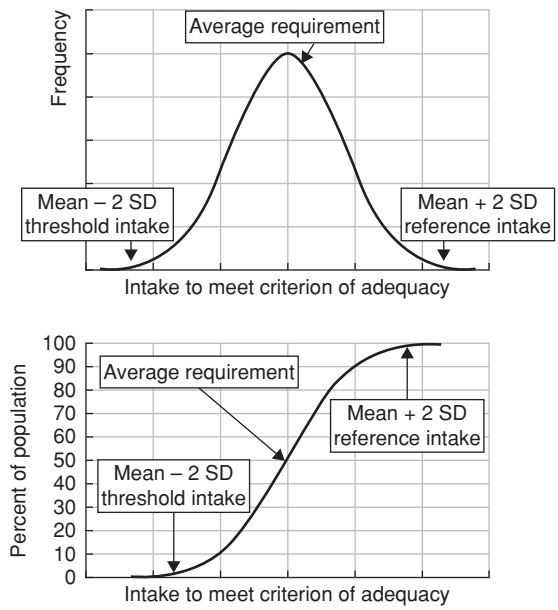

Figure 1.3 The determination of reference nutrient intakes.

The lower graph in Figure 1.3 shows the distribution of requirements plotted as the cumulative percentage of the population whose requirements have been met at each level of intake. This can therefore be used to estimate the probability that a given level of intake is adequate to meet an individual's requirements.

For some nutrients, deficiency is unknown except under experimental conditions, and there are no estimates of average requirements, and therefore no reference intakes. Since deficiency does not occur, it is obvious that average intakes are more than adequate to meet requirements, and for these nutrients, there is a range of intakes that is defined as safe and adequate, based on the observed range of intakes.

The reference intakes of vitamins and minerals shown in Tables 1.10, 1.11, 1.12, and 1.13 are age and gender specific. Apart from foods for infants and children, the highest requirement for any population group is used to provide the basis for nutritional labeling of foods.

Other Compounds in Foods

Three other groups of compounds in foods are not considered to be nutrients, in that they are not dietary essentials, but need consideration:

- Compounds that have a metabolic function in the body, but, as far as is known, can be synthesized in adequate amounts to meet requirements. These include carnitine, choline, inositol, and ubiquinone (coenzyme Q).
- Complex carbohydrates (polysaccharides) such as cellulose in plant cell walls that are not digested, but provide bulk in the gut, improving intestinal transit time, and also provide a substrate for intestinal bacterial fermentation and so promote intestinal health—collectively these are known as non-starch polysaccharides, or sometimes as dietary fiber (although fiber includes compounds such as lignin that are not carbohydrates and are not substrates for bacterial fermentation).
- A wide variety of plant metabolites that have potentially protective functions including
  - polyphenols, flavonoids, anthocyanins, and carotenoids that have antioxidant actions;
  - glucosinolates and glycosides that modify the metabolism of potential carcinogens;
  - phytoestrogens that have antiestrogenic actions;
  - polyterpenes and squalene that inhibit cholesterol synthesis.

In addition to these, an adequate intake of water is essential to maintain health and performance, though water is often not considered as a nutrient.

Further Reading

Ainslie, P., Reilly, T., & Westerterp, K. (2003) Estimating human energy expenditure: a review of techniques with particular reference to doubly labelled water. *Sports Medicine* 33, 683–698.

Bender, D.A. (2003) *Nutritional Biochemistry of the Vitamins*, 2nd edn., Cambridge University Press, New York and London.

Bender, D.A. (2008) *Introduction to Nutrition and Metabolism*, 4th edn. CRC Press, Boca Raton, FL.

Bender, D.A. & Bender, A.E. (1997) *Nutrition: A Reference Handbook*. Oxford University Press, Oxford.

Gibney, M.J., Lanham-New, S.A., Cassidy, A., et al. (2009) *Introduction to Human Nutrition*, 2nd edn. Wiley-Blackwell, Oxford.

Vaz, M., Karaolis, N., Draper, A., et al. (2005) A compilation of energy costs of physical activities. *Public Health Nutrition* 8, 1153–1183.

References

Anderson, J.T., Keys, A., & Grande, F. (1957) The effects of different food fats on serum cholesterol concentration in man. *Journal of Nutrition* 62, 421–424.

Armstrong, W.D., Van Pilsum, J., Keys, A., et al. (1957) Alteration of serum cholesterol by dietary fats. *Proceedings of the Society for Experimental Biology and Medicine* 96, 302–306.

Bender, D.A. (2012) The metabolism of "surplus" amino acids. *British Journal of Nutrition* 108, S113–S121.

British Nutrition Foundation. (1995) *Trans-fatty Acids: Report of a Task Force*. Blackwell Science, Oxford.

Department of Health. (1991) *Dietary Reference Values for Food Energy and Nutrients for the United Kingdom*. Her Majesty's Stationery Office, London.

FAO/WHO (2001) *Human Vitamin and Mineral Requirements: Report of a Joint FAO/WHO Expert Consultation*. Food and Nutrition Division of the United Nations Food and Agriculture Organization, Bangkok, Thailand, Rome.

Hegsted, D.M., McGandy, R.B., Myers, M.L., et al. (1965) Quantitative effects of dietary fat on serum cholesterol in man. *American Journal of Clinical Nutrition* 17, 281–295.

Hegsted, D.M., Ausman, L.M., Johnson, J.A., et al. (1993) Dietary fat and serum lipids: an evaluation of the experimental data. *American Journal of Clinical Nutrition* 57, 875–883.

Heymsfield, S.B., Harp, J.B., Rowell, P.N., et al. (2006) How much may I eat? Calorie estimates based upon energy expenditure prediction equations. *Obesity Reviews* 7, 361–370.

Institute of Medicine. (1997) *Dietary Reference Intakes for Calcium, Phosphorus, Magnesium, Vitamin D and Fluoride*. National Academy Press, Washington, DC.

Institute of Medicine (1998) *Dietary Reference Values for Thiamin, Riboflavin, Niacin, Vitamin B_6, Folate, Vitamin B_{12}, Pantothenic Acid, Biotin and Choline*. National Academy Press, Washington, DC.

Institute of Medicine. (2000) *Dietary Reference Values for Vitamin C, Vitamin E, Selenium and Carotenoids*. National Academy Press, Washington, DC.

Institute of Medicine. (2001) *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc*. National Academy Press, Washington, DC.

Institute of Medicine. (2011) *Dietary Reference Values for Calcium and Vitamin D*. National Academy Press, Washington, DC.

Keys, A., Anderson, J.T., & Grande, F. (1957) Prediction of serum-cholesterol responses of man to changes in fats in the diet. *Lancet* 273, 959–966.

Levine, J.A. (2005) Measurement of energy expenditure. *Public Health Nutrition* 8, 1123–1132.

Prentice, A.M. (2005) Macronutrients as sources of food energy. *Public Health Nutrition* 8, 932–939.

Rayman, M.P. (1997) Dietary selenium: time to act. *British Medical Journal* 314, 387–388.

Rayman, M.P. (2000) The importance of selenium to human health. *Lancet* 356, 233–241.

Ritz, P. & Coward, W.A. (1995) Doubly labelled water measurement of total energy expenditure. *Diabetes & Metabolisme* 21, 241–251.

Schofield, C. (1985a) An annotated bibliography of source material for basal metabolic rate data. *Human Nutrition. Clinical Nutrition* 39(Suppl. 1), 42–91.

Schofield, W.N. (1985b) Predicting basal metabolic rate, new standards and review of previous work. *Human Nutrition. Clinical Nutrition*, 39(Suppl. 1), 5–41.

Scientific Committee for Food. (1993) *Nutrient and Energy Intakes for the European Community,*. Commission of the European Communities, Luxembourg.

Speakman, J.R. (1997) *Doubly-Labelled Water: Theory and Practice.* Chapman & Hall, London.

Weir, J.B. (1949) New methods for calculating metabolic rate with special reference to protein metabolism. *Journal of Physiology* 109, 1–9.

WHO. (1985) *Energy and Protein Requirements. Report of a Joint FAO/WHO/UNU Expert Consultation*. WHO, Geneva.

WHO. (2007) *Protein and Amino Acid Requirements in Human Nutrition*. WHO, Geneva.

Chapter 2

Exercise Physiology

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Introduction

Exercise physiology is a relatively young scientific discipline that evolved from the sciences of anatomy—the study of body structure—and physiology—the study of body function. Physiologists study how the body functions to regulate *homeostasis*, the maintenance of a stable and sustainable internal environment. Exercise physiologists study how the body responds to the challenges to homeostasis imposed by exercise (i.e., physical activity, sports training and competition, physical work). The impact of muscle contraction on homeostasis is vast and invokes responses from virtually every physiological system (Kenney et al., 2012). The study of exercise physiology includes how the body responds to a single bout of exercise, i.e., the acute responses to exercise, and how the body adapts over time to repeated bouts of exercise, commonly referred to as training effects. In addition, exercise physiologists are interested in how physiological and metabolic functions respond to nutrient intake before, during, and after exercise.

Much has been written in recent years about the modern role of the parent discipline, physiology, in what has become an increasingly reductionist scientific community. In 1987, the directors of two of the National Institutes of Health's (NIH) divisions published editorials heralding the era of molecular biology and the genomic revolution (Hurd & Lenfant, 1987a, 1987b). The linkage of research funding to predominantly molecular and genetic approaches to science subsequently led to the demise or reorganization of many traditional departments of physiology (Wagner & Paterson, 2011). Dr. Michael Joyner (2011) has compared the genomics (and other similarly reductionist "-omics" disciplines) to physiology, claiming that the former has failed to "get satisfying and applicable large scale insights from their work." In other words, at this point in time, reductionism has not led to overtly promised disease cures and novel insights into how organisms truly function. On the other hand, physiology (what reductionists now oddly and redundantly call "systems biology") has continued to inform scientists about life at the macro level, while forming the foundation for advances in "epidemiology, population health, and even public policy" (Joyner, 2011).

In that context, *exercise* physiology is poised to play an even greater role as a subdiscipline of physiology. While there is inherent import in better understanding the resting organism, its true function can only truly be appreciated by examining its reaction to challenges to homeostasis, i.e., the organism under stress. For humans, that stress most often involves the body's integrated responses to the muscular actions that define exercise, as well as to environmental stresses of heat and cold, hypo- and hyperbaria, and gravitational/postural perturbations.

This chapter provides a broad overview of the primary physiological responses involved in

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maintaining homeostasis during exercise and when exposed to environmental stressors. In that regard, this chapter covers cardiorespiratory function, neuromuscular function, thermoregulatory function, fatigue, and principles of training. Within each of these topics, implications for sports nutrition are identified. The critical responses and adaptations that occur with energy metabolism and hormone release are covered extensively in other chapters and so will not be addressed here.

Cardiorespiratory Function

When a fit and seemingly healthy athlete of any age suddenly dies, their death is a stark and sobering reminder of our mortality and of the inherent frailty of human life. Life depends upon the continued beat of the heart muscle, a fist-sized organ of specialized cardiac muscle cells that, even in a sedentary person, is capable of well over 2 billion contractions in a normal life span. When the heart simply stops beating or becomes incapable of pumping sufficient blood to the brain, life is immediately threatened. The gravity of sudden cardiac arrest, congenital heart abnormalities, and atherosclerotic heart disease is not surprising considering that the cardiorespiratory system supports every other physiological system in the body.

The major cardiorespiratory functions can be grouped into six categories:

- Delivery of oxygen and nutrients such as carbohydrate, protein, fat, vitamins, and minerals to every cell in the body
- Removal of carbon dioxide and other metabolic waste products
- Transport of hormones and other bioactive molecules from endocrine glands to target receptors on cells
- Support of thermoregulation and control of body fluid balance
- Maintenance of acid–base balance and control of the body's pH
- Support of immune function via physical and humoral barriers to infectious organisms

The cardiovascular system (the heart, the blood vessels, and the blood) and the respiratory system (the airways, the lungs, and the lung/blood interface) are often studied and written about as two separate organ systems. This is an understandable distinction considering the vast complexity of each system; however, in this chapter the two systems will be considered together to conserve space and simplify the understanding of how the respiratory and cardiovascular systems function seamlessly to meet the demands of exercise.

Breathing (*pulmonary ventilation*) is the first of four steps required to transport oxygen from the air to all the cells in the body. When air is drawn into the lungs through the nose and mouth, it is quickly moistened and warmed (unless the ambient air is already warmer than the body temperature) in its passage through the airways (nasal cavity, pharynx, larynx, trachea, and bronchial tree). The second step is *pulmonary diffusion*—the exchange of oxygen and carbon dioxide between the lungs and the blood. That gas exchange occurs in the respiratory bronchioles and the alveoli, the respiratory zone of the lung that is anatomically designed to allow oxygen to pass from lungs to blood and carbon dioxide to pass from blood to lungs. The *transport* of oxygen and carbon dioxide in the blood is the third step, a process involving both gases being dissolved in the plasma or bound to hemoglobin in red blood cells (RBCs) and being transported to and from the peripheral tissues via the heart. The fourth and final step in the process is that of *capillary diffusion*, the exchange of oxygen and carbon dioxide between the blood in the capillaries and the cells.

Anyone who has exercised or been exposed to high altitude recognizes the central importance of pulmonary ventilation in maintaining homeostasis, let alone in meeting the demands of vigorous physical activity. The active process of inspiration requires contraction of the diaphragm and the external intercostal muscles to increase the volume of the thorax and thereby expand the lungs, while expiration is typically a passive process that relies on relaxation of the diaphragm and intercostal muscles to decrease the volume of the thorax. When lung volume changes, the pressure inside the lung either increases (expiration) or decreases (inspiration) and air is either drawn into or forced out of the lungs. This relationship between lung volume and lung

pressure is described by *Boyle's gas law* which states that the product of gas pressure and volume is a constant value whenever temperature is also constant. In other words, increasing the lung volume on inspiration causes the pressure inside the lungs to fall and that causes air to rush in, moving from an area of higher pressure (outside the body) to an area of lower pressure (inside the lungs). On expiration, the elastic properties of the lungs and the intercostal muscles cause the thorax to recoil, decreasing lung volume and increasing pressure inside the lungs, forcing air out. The forced breathing that occurs during exercise also benefits the cardiovascular system because the regular changes in intrathoracic pressure function as a *respiratory pump* that helps blood fill and empty from the pulmonary veins and vena cava, assisting the return of blood to the heart. Contracting skeletal muscles also contribute to blood return during exercise by functioning as a *muscle pump* that helps squeeze venous blood back toward the heart.

Blood returning to the heart through the venous system has a reduced oxygen content and elevated carbon dioxide content compared to arterial blood. The right side of the heart pumps blood through the pulmonary artery to the lungs where it flows through the pulmonary capillaries that completely envelop the alveolar sacs. The pulmonary capillaries are so small that RBCs must pass through in single file, providing maximum exposure for gas exchange with the alveoli. The alveolar-capillary membrane can be thought of as the *respiratory membrane* because it is well designed to facilitate gas exchange. Oxygen molecules have to pass only through the alveolar wall and across the capillary wall into the blood, with carbon dioxide molecules making the opposite journey. The respiratory membrane is very thin, so the 300 million alveoli are constantly bathed in blood.

Whether on a sunny beach or standing atop Mount Everest, the air we breathe contains 20.93% oxygen, 0.03% carbon dioxide, and 79.04% nitrogen. The reason that the air is "thin" at altitude is because the atmospheric (barometric) pressure is less than at sea level. For example, on the beach, where the atmospheric pressure is 760 mmHg, the partial pressure of oxygen is 760 × 0.2093 or 159 mmHg. On Mount Everest, the atmospheric pressure is estimated to be 253 mmHg, so the partial pressure of oxygen is only 53 mmHg (253 × 0.2093). When the partial pressure of oxygen in the air drops, less oxygen can be dissolved in blood, as described by Henry's law (gasses dissolve in liquids in proportion to their partial pressures).

At sea level, when the air we breathe enters the lungs, it is humidified with water vapor from the respiratory tract and mixed with the residual air in the alveoli, so the partial pressure of oxygen drops from 159 to 105 mmHg. That pressure is still well above the partial pressure of oxygen in the venous blood (about 40 mmHg), so oxygen moves down this pressure gradient from the alveoli into the blood. During intense exercise, with muscle cells taking up more oxygen than they do at rest, the partial pressure of oxygen in venous blood is even lower, so the diffusing capacity for oxygen may be three times that at rest, ensuring that the blood remains fully oxygenated, even though the blood passing through the lungs is in contact with the alveoli for less than 1 second. Even during all-out exercise, almost all of the hemoglobin molecules (more than 98%) bind immediately to oxygen. An exception may be the highly trained middle distance athlete, in whom the blood leaving the lungs may not be fully saturated with oxygen (Williams et al., 1986). Each RBC contains about 250 million hemoglobin molecules and each hemoglobin molecule binds four oxygen molecules, so each RBC can transport one billion molecules of oxygen.

The carbon dioxide produced by muscle and other cells during rest and exercise reaches the lungs in venous blood with a partial pressure of 46 mmHg. The partial pressure of carbon dioxide in the alveoli is 40 mmHg, a small difference, but one that is more than enough to allow carbon dioxide to move from blood to lungs, in large part because the carbon dioxide has a *diffusion coefficient* (an index of how rapidly a gas crosses a membrane) that is 20 times greater than that of oxygen.

The high partial pressure of oxygen in the blood ensures that hemoglobin is almost fully saturated with oxygen. At the muscle cells, where it is hotter and more acidic than in the lungs, oxygen molecules readily detach from hemoglobin and move out of

the capillaries into the cells for use in energy metabolism. Carbon dioxide is produced during cellular metabolism and diffuses out of the muscle cells into the blood and the RBCs where it can bind to hemoglobin for its trip to the lungs in the venous part of the circulatory system. Muscle cells also produce hydrogen ions during hard exercise as a byproduct of energy metabolism (every lactate molecule is accompanied by one hydrogen ion). An increase in hydrogen ions lowers the pH (increased acidity) of muscle and blood, a change that impairs muscle function and disrupts homeostasis throughout the body. To keep the pH of blood from dropping too far, the hydrogen ions combine with bicarbonate ions in the blood to form additional carbon dioxide molecules for transport to the lungs. In that way, a potentially harmful fall in pH is prevented, allowing intense exercise to continue without risk to health. Nutritional interventions such as "loading" with sodium bicarbonate and sodium citrate solutions or the use of β-alanine have been used by athletes as additional buffers for the hydrogen ions produced during intense exercise in the hope of improved performance (Chapter 26).

An increased capacity of the blood to carry oxygen is obviously an important characteristic for athletes and is one of the central cardiorespiratory adaptations that occur with endurance training. Not surprisingly, the oxygen-carrying capacity of the blood depends mostly on the hemoglobin content; more RBCs mean more hemoglobin and that means more oxygen can be carried to the tissues. In healthy men, blood normally contains about 14–18 g of hemoglobin per 100 ml of blood; in women, the range is 12–16 g/100 ml. Each gram of hemoglobin can bind 1.34 ml of oxygen, so blood can carry 16–24 ml of oxygen/100 ml when hemoglobin is fully saturated. Athletes and others with low hemoglobin levels or iron-deficiency anemia have compromised aerobic capacities and impaired performance because the oxygen-carrying capacity of their blood is reduced.

When arterial blood leaves the lungs, it carries an average of 20 ml of oxygen/100 ml, a value that drops to 15 ml O_2/100 ml once the blood has passed through the capillary system where some of the oxygen is taken up by cells for use in energy metabolism. The 5 ml O_2/100 ml is referred to as the (a-v)O_2 difference, that is, the difference in oxygen content between arterial and venous blood. During all-out exercise, the (a-v)O_2 difference can increase to more than 15 ml O_2/100 ml blood. The ability of the cardiorespiratory system to deliver oxygenated blood to the muscles and the muscles' ability to extract oxygen from the blood defines a person's aerobic capacity, as measured by maximal oxygen consumption (VO_2 max).

While breathing (i.e., pulmonary ventilation) is an involuntary response, we also have voluntary control of pulmonary ventilation. We can temporarily stop breathing or we can temporarily increase our breathing rate to high levels. In both cases, the respiratory centers in the brain override our voluntary actions and restore the normal rate and depth of breathing. The respiratory centers (one for inspiration, one for expiration) do not act alone in controlling breathing to the lungs. Other brain neurons are sensitive to hydrogen ions and carbon dioxide, and when stimulated, these neurons activate the inspiratory center to increase pulmonary ventilation which results in lowering the levels of hydrogen ions and carbon dioxide. In addition, chemoreceptors in the aorta and the carotid artery respond to changes in the partial pressure of oxygen in arterial blood and to changes in hydrogen ion and carbon dioxide content. In fact, the strongest stimulus to breathing is the partial pressure of carbon dioxide in the blood. As carbon dioxide increases, so does the level of hydrogen ions, causing blood pH to fall. An increase in breathing removes carbon dioxide, lowers hydrogen ion concentration, and ensures adequate oxygenation of arterial blood.

During exercise, the body has to match the rate of pulmonary ventilation with cardiac output, the volume of blood pumped by the heart each minute, to ensure that cells throughout the body are supplied with sufficient oxygen. Cardiac output (expressed in liters/minute) is a product of heart rate and stroke volume (the volume of blood pumped by the left ventricle with each heartbeat). During exercise, cardiac output increases in proportion to the workload because heart rate and stroke volume both increase. Of course, there is an upper limit to both heart rate and stroke volume and those maximum values dictate maximal cardiac output. Training results in an

increase in stroke volume and therefore maximum cardiac output also increases. In fact, the heart's ability to deliver oxygenated blood to the muscles is the primary determinant of VO_2 max.

As with skeletal muscle, cardiac muscle hypertrophies with training, especially aerobic training. The hypertrophy of the left ventricle allows for a stronger contraction, which helps increase stroke volume and cardiac output. Improved fitness also results in a slower heart rate at rest and during exercise and an increase in blood volume, the combined effect of which is greater filling of the left ventricle after each heartbeat (increased end-diastolic volume). Greater filling of the left ventricle is accompanied by a greater stretch of the left ventricular wall and an increased force of contraction, a response known as the *Frank–Starling mechanism*. One reason why dehydration during exercise may be detrimental to both health and performance is that dehydration reduces blood volume, stroke volume, and cardiac output, compromising the heart's capacity to deliver blood to muscles and skin (to enable heat loss).

The total volume of blood typically ranges between 3 and 6 liters, depending in large part on body size and fitness. Larger, more aerobically fit individuals have a greater blood volume than smaller, sedentary individuals. A challenge for the body during vigorous exercise is to manage blood flow so that organs such as contracting muscles, skin, and brain are well supplied with blood by diverting blood from other organs that have a lesser need such as less-active muscles, stomach, intestines, liver, and kidneys. The body accomplishes this balancing act by dilating blood arterioles (small vessels preceding the tissue capillaries) in tissues that require increased blood flow and constricting arterioles in tissues that get by with less blood flow. Vasoconstriction and vasodilation of arterioles are under intrinsic and extrinsic control, a control system that allows for rapid and large changes in blood flow. For example, vasoconstriction and vasodilation are so effective at redistributing blood flow that blood flow to muscles during vigorous exercise can increase up to 25 times resting values. *Intrinsic control* is local control. In other words, when metabolites produced by muscles during exercise flood out of the muscle cells into the surrounding extracellular space, they quickly come in contact with the smooth muscle cells that line the arterioles. Metabolites such as carbon dioxide, lactic acid, hydrogen ions, acetylcholine (ACh), adenosine, and potassium ions cause arterioles to dilate so that blood flow to that area rises, increasing oxygen delivery and augmenting the removal of metabolic "waste" products. Other dilator substances are produced directly by the arteriole smooth muscle cells, including nitric oxide and prostaglandins. Finally, changes in blood pressure within the arteriolar bed can cause vasodilation or vasoconstriction. Whenever arterioles dilate, the pressure within those vessels falls and that further relaxes the smooth muscle cells, promoting even more blood flow.

Local control of blood flow is a powerful way to ensure that the most metabolically active cells receive the blood they need. But local control of blood flow is just that: local. Another control system is needed to shift (shunt) blood among organs during exercise. This system is referred to as *extrinsic control* of blood flow and is primarily regulated by the sympathetic nervous system. Arteries and arterioles are richly supplied with nerves. When those nerves fire, the smooth muscle cells in the vessel wall contract, dramatically reducing blood flow. Under normal circumstances at rest, there is constant sympathetic input to arterioles, so a small amount of vasoconstriction is always present. This "*tonic vasoconstriction*" is beneficial because it helps maintain normal blood pressure. If that normal sympathetic "tone" is reduced, blood flow increases. Of course, when sympathetic tone increases, blood flow decreases—exactly what happens during exercise to shunt blood from less metabolically active organs to more metabolically active organs. For example, blood flow to the stomach and intestines is markedly reduced during vigorous exercise as a result of increased sympathetic tone to those organs (an example of extrinsic control of blood flow). At the same time, metabolites produced by active muscles cause an increase in local blood flow (intrinsic control) as sympathetic tone to those muscles decreases (extrinsic control), further augmenting blood flow. In this example, blood is effectively shunted from the stomach and intestines so that more blood is

available to active muscles. However, competition for blood can occur if an athlete were to eat a meal too soon before training or competition. When that happens, the stomach and intestines are denied the increased blood flow they need for digestion and absorption, and gastrointestinal discomfort may result.

It is impossible to discuss blood flow and blood distribution without at least a brief mention of how blood pressure is controlled. Pressure is required to push blood around the body and return it to the heart. The contraction of the left ventricle is the source of most of that pressure (along with help from the respiratory and muscle pumps mentioned earlier). Blood pressure increases during exercise as the heartbeats harder and faster to ensure that ample blood is delivered to meet the needs of the most metabolically active cells. Our bodies rely on internal reflexes to maintain blood pressure to meet the blood flow needs of our organs. Specialized receptors provide the constant feedback required to manage blood pressure. *Baroreceptors* (pressure receptors) in the aorta and carotid arteries respond to stretching of the vessel walls. When blood pressure is elevated, the vessel walls stretch and the baroreceptors send signals that result in decreased heart rate and increased vasodilation of arterioles throughout the body. Just the opposite series of events occurs when blood pressure drops and the baroreceptors sense less stretch than normal. *Chemoreceptors* and *mechanoreceptors* also participate in blood pressure control by providing feedback about the chemical environment in muscles and about the changes in length and tension of muscles.

Blood pressure can also be influenced by the viscosity of the blood. Not surprisingly, the heart has to work harder if the blood becomes thicker (higher viscosity) so blood pressure increases accordingly. For example, the viscosity of blood increases slightly with dehydration as plasma water is lost from the vascular space. But the largest influence on blood viscosity is blood hematocrit, a simple measure of the percentage of blood comprised by the RBCs. In healthy, sedentary people, hematocrit values range from 41% to 50% in men and from 36% to 44% in women. Anemia lowers hematocrit because RBC content is lowered—either fewer cells or smaller cells. Blood doping techniques, including erythropoietin injections and the infusion of RBCs, have been used by athletes to increase RBC content and hematocrit in an attempt to increase the oxygen-carrying capacity of blood and improve performance. Both techniques are effective in that regard, but at the risk of raising hematocrit and blood viscosity to dangerous levels that have claimed the lives of some athletes. The normal response to endurance training is for hematocrit levels to fall, not rise, as plasma volume increases more than the increase in RBC content. The result is a greater oxygen-carrying capacity of blood and a greater blood volume to better serve muscles and skin during exercise.

Neuromuscular Function

The study of exercise physiology centers around the contraction of skeletal muscle, the fundamental requirement for all human movement. The contraction of muscle cells (often referred to as *muscle fibers*) in smooth muscle, cardiac muscle, and skeletal muscle represents the transformation of chemical energy into mechanical energy, a process initiated by way of nervous impulses from the brain, from spinal reflexes, or from local cellular pacemakers. In all types of muscle, nutrients provided by the diet play critical roles in neuromuscular function, from the propagation of nervous impulses, to initiating contraction of the myofibrils, to fueling continued contraction and relaxation, and to ensuring that muscles recover, repair, adapt, and grow in response to training.

Smooth muscle, sometimes termed *involuntary muscle*, is not under conscious control. Smooth muscle is found in the walls of most blood vessels, allowing local blood flow to be altered by the constriction or dilation of the vessel. Smooth muscle is also found in the walls of internal organs, allowing for peristaltic contraction and relaxation in the gut to aid digestion and absorption, for contraction of the urethra during urination, and in the contraction and relaxation cycles of the uterus and vagina during childbirth. Most of the heart's structure is composed of cardiac muscle cells. *Cardiac muscle* essentially controls its own activity, but is also affected by neural and hormonal input, especially during exercise.

Skeletal muscles are appropriately named because most attach to and move the skeleton. The human body contains more than 600 skeletal muscles, all of which operate similarly in terms of physiology and metabolism. Large groups of skeletal muscles in the arms, chest, back, abdomen, thighs, and lower legs are of particular interest in sports nutrition because their function during exercise in large part dictates energy expenditure and fuel utilization. After exercise, those same muscles must recover adequately to sustain subsequent training or competition, a time when proper nutrition is of great importance.

All three muscle types are of interest to exercise physiologists, but this chapter will focus on the physiology of skeletal muscle (see Figure 2.1) because of the central role it plays in the study of sports nutrition.

Skeletal muscle cells are multinucleated, a fact that sets them apart from most cells in the body. Muscle cells do not extend from one end of the muscle to the other but often divide into compartments within each muscle. As a result of this compartmentalization, the longest human muscle cells are about 12 cm (4.7 inches), corresponding to about 500,000 *sarcomeres*, the basic functional unit of the muscle cell. The number of cells in different muscles ranges from several hundred (e.g., the tensor tympani, attached to the eardrum) to more than a million (e.g., the medial gastrocnemius muscle).

The muscle cell membrane is referred to as the *sarcolemma*, a combination of the *plasmalemma* and the *basement membrane.* The plasmalemma fuses with the tendon and is critical to various aspects of skeletal muscle function. For example, when the muscle cell is contracted, the plasmalemma takes on a folded appearance; when the muscle is stretched, these folds disappear, ensuring that the plasmalemma remains functional regardless of muscle length. The plasmalemma is also important in nerve transmission across the muscle cell and is involved in acid–base balance and metabolite/nutrient transport. Of particular interest to exercise physiologists,

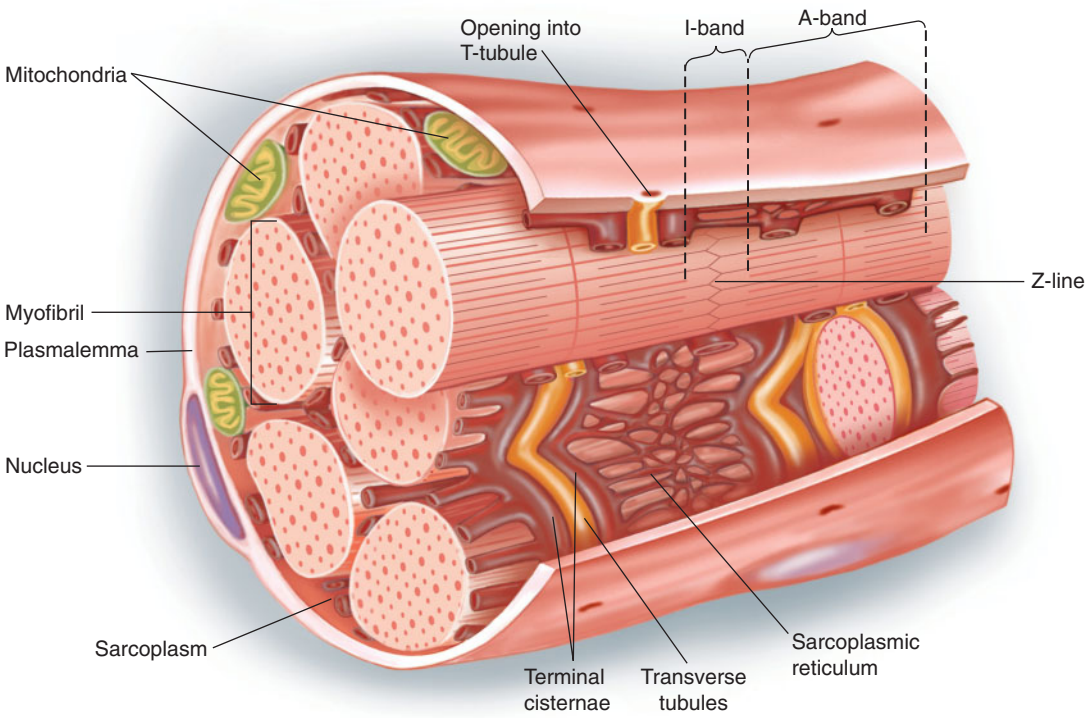

Figure 2.1 Important contractile elements of a single muscle cell.
Source: Adapted with permission from Wilmore et al. (2008a).

satellite cells are located between the plasmalemma and the basement membrane. Satellite cells are important during periods of muscle growth (e.g., during growth spurts and hypertrophy from strength training) as well as in the muscle's response to injury, immobilization, and training.

The *sarcoplasm* of muscle cells is a gel-like substance that fills the space surrounding the *myofibrils* (the contractile filaments). Suspended in the sarcoplasm adjacent to the myofibrils are enzymes, minerals, fats, proteins, glycogen, myoglobin, and various cellular organelles (e.g., mitochondria, endoplasmic reticulum, lysosomes).

Although about 75% of muscle weight is water (an indication of the importance of muscle cell hydration to its function), muscle cell proteins constitute a large proportion of the remaining weight. Roughly two-thirds of all skeletal muscle protein is *myosin*, which comprises two protein strands twisted together. Each myosin filament contains many protruding myosin heads that form cross-bridges that interact during muscle contraction with specialized active sites on the thin *actin filaments*. Titin and other structural proteins stabilize the myofibrils. The *muscle hypertrophy* that results from normal growth and sport training occurs from increased production of contractile proteins (i.e., actin and myosin), structural proteins (i.e., titin, dystrophin, integrins, etc.), and regulatory proteins (i.e., enzymes, signaling molecules, etc.).

Skeletal muscle cells are organized into *motor units* of varying size consisting of one a motor neuron and all the muscle cells it innervates. In the case of the muscle cells controlling the movements of the eyes, each motor unit may contain only five muscle cells, allowing for small, precise, low-force movements. In a large skeletal muscle such as the gastrocnemius, the motor units are large, with perhaps 1700 cells or more per motor unit. When a motor unit is activated, all of its muscle cells contract.

Skeletal muscle motor units differ in the speeds at which their muscle cells shorten and in their ability to generate maximal force. In other words, motor units differ in muscle cell type (i.e., *muscle fiber type*). Type I cells (often called slow or slow-twitch) and type II cells (often called fast or fast-twitch) are distinguished by the size of their motor neurons (smaller in type I), the number of cells within each motor unit (typically <300 in type I; >300 in type II), their different speeds of contraction (faster in type II), and maximal force production (greater in type II). Although only one form of type I cell has been identified, in humans, there are at least three forms of type II cells: type IIa, type IIc, and type IIx. On average, most muscles are composed of roughly 50% type I cells and 25% type IIa cells. The remaining 25% are mostly type IIx, with type IIc cells making up only 1–3% of the muscle. These numbers are only averages because the exact percentage of each of these cell types varies greatly among athletes and even within muscles of the same athlete.

Regardless of the cell type, whenever the motor neuron depolarizes, the muscle cell membrane (plasmalemma) also depolarizes. As the action potential sweeps across the plasmalemma, transverse tubules (extensions of the plasmalemma) that invaginate muscle cells carry the action potential inward, allowing for almost instant release of calcium ions stored in the *sarcoplasmic reticulum*, a longitudinal network of channels that parallel the myofibrils. Calcium ions bind with a protein called *troponin*, causing an adjacent protein, *tropomyosin*, to move off the myosin binding sites on the actin molecule, allowing the nearby myosin heads to bind with the active sites. Once bound, the myosin heads tilt, pulling the thin actin filament past the thicker myosin filament. Tilting of the myosin head requires energy in the form of ATP. Once neural input to the motor unit stops, calcium is actively pumped out of the sarcoplasm back into the sarcoplasmic reticulum. Tropomyosin then returns to cover the myosin binding sites on actin and relaxation of the muscle cell ensues. This series of events is referred to as the *sliding-filament theory* of muscle contraction.

To meet their energy needs during rest and exercise, muscle cells require ATP to maintain membrane-bound electrolyte pumps, such as the Na^+–K^+ pump (an enzyme called ATPase releases energy from ATP for this purpose), to fuel the tilting of myosin heads once contraction begins (via myosin ATPase), and to pump calcium ions back into the sarcoplasmic reticulum once contraction ends (via SERCA ATPase). At rest, it is estimated that each muscle cell contains roughly one billion

ATP molecules and those molecules will be used (and then regenerated) within 2 minutes to meet the resting energy needs of the cell. To put that number in perspective, we produce and use the equivalent of half of our body weight in ATP each day (Meyer & Wiseman, 2012). During intense exercise, the ATP needs of some muscle cells might be more than 100 times greater than at rest, requiring the muscle cell to rapidly produce the needed ATP. To keep up with the demand for ATP, the continued presence of calcium ions in the sarcoplasm stimulates feed-forward control of ATP production, while the by-products of ATP—ADP, AMP, and Pi—provide feedback control of ATP production. In this manner, the large ATP requirements of muscle cells during exercise are met, providing that the demand for ATP does not surpass the muscle's ability to produce it (Chin, 2010).

Thermoregulatory Function

Athletes who begin training programs in the heat of summer are challenged not only by the physical demands of rigorous training but also by the additional stress imposed by environmental heat. Heat stress affects the function of skeletal muscle, the brain and central nervous system (CNS), and the cardiorespiratory system. Because heat stress has such widespread physiological ramifications, it is not surprising that even mild heat stress can impair performance, as discussed later in this section. Cold stress also affects physiological responses to exercise and can impair performance and jeopardize health if core body temperature drops too low.

During exercise in both hot and cold environments, the heat produced by metabolism and muscle contractions must be lost to the surroundings to keep the body temperature from rising to dangerous levels. An increase in body temperature is a normal response to exercise and the body has mechanisms that allow that rise in temperature to occur and prevent it from rising too high. Tragically, those mechanisms are not fail-safe and deaths from *hyperthermia* (heat stroke; dangerously high body temperature) are still too common among athletes. Thermoregulation can also fail in cold environments, increasing the risk of frostbite and death. Fortunately, the human body can *acclimate* over days and weeks to heat or cold, reducing health-related risks and improving the capacity for physical work. (*Acclimatization* is the preferable term when referring to the thermoregulatory adaptations that occur over months or years of exposure to heat or cold.)

Body temperature at rest is regulated within ±1°C (1.8°F), even when the body is exposed to large changes in environmental temperature. Conscious human behavior assists in thermoregulation because whenever it becomes too hot, we seek the shade or a cool room and remove clothing. When we become too cold, we put on additional clothing, avoid the wind, and look for sources of heat (a warm room, a fireplace, a hot cup of coffee). As a result, humans can remain comfortable and safe in temperatures ranging from well below 0°C to above 40°C. In the simplest terms, maintaining a safe internal body temperature requires a balance between heat production by the body and heat loss from the body. When this balance is disturbed, body temperature either rises or falls.

Each time an ATP molecule is used for energy, about 75–80% of that energy is "lost" as heat. In that regard, every cell is a mini-furnace, constantly releasing heat to surrounding cells and blood. In turn, the body has to release heat to its surroundings in order to maintain a normal resting body temperature of 36.1–37.8°C (97–100°F). During exercise, heat production increases markedly and so must heat loss to prevent the body from overheating. Simply put, during both rest and exercise, the body has to move heat from the muscles to the skin where it can be transferred to the environment. The movement of heat from muscle to skin is one important reason why an increased skin blood flow is so vital during exercise. Once at the skin, heat can be transferred to the environment by radiation, evaporation, convection, and conduction.

Most athletes do not lose much heat by *conduction* during exercise because very rarely are athletes in direct prolonged contact with a colder surface, such as the ice during an ice hockey game. *Convection* can be a significant source of heat loss

(think of the wind-chill factor) because athletes are often exposed to cold air (or water, in the case of swimmers) that swirls around the skin and speeds heat loss. Convection can also be a source of heat gain, as on hot days when the environmental temperature is higher than the skin temperature. At rest, over two-thirds of total heat loss from the body results from convection and radiation. *Radiation* of heat occurs as a result of the infrared rays given off by all heat-producing objects, whether that is the hot body of an athlete or the hot-water radiator in a home. As with convection and conduction, radiation is also a two-way street. We quickly gain heat from the sun by radiation and most of the heat we lose at rest is radiated from our bodies to the cooler surroundings. Radiation, convection, and conduction are collectively considered avenues of *dry heat exchange*, and *insulation* is defined as resistance to dry heat exchange. Insulation is an important consideration during exercise in cold environments but should be minimized in warm environments by wearing clothing that aids heat loss.

The most important avenue for heat loss during exercise in warm environments—and the primary avenue during vigorous exercise—is *evaporation*. When sweat evaporates from the surface of the skin and is converted to vapor, heat is lost from the body. About 80% of the heat loss during vigorous, sweaty exercise occurs as a result of the evaporation of sweat. Of course, the heat-loss benefit of evaporating sweat comes at a price: dehydration. Consuming adequate volumes of fluid during exercise helps ensure that sweating continues and, even more importantly, helps maintain blood volume and thereby adequate blood flow to muscles and skin.

Sweating begins once core body temperature reaches a preset threshold temperature and will continue until core temperature drops below that threshold. If the environment is very humid the amount of sweat that can be evaporated is reduced and heat loss is impeded. If sweat droplets fall to the ground, there is no heat loss from evaporation; heat loss occurs only when sweat evaporates from the skin. For example, plastic sweat suits minimize evaporation and heat loss, causing core temperature to rise quickly, potentially to dangerous levels. Fortunately, when sweating is not impeded, the evaporation of sweat can effectively transfer heat from the body to the environment. For example, the evaporation of 1 liter of sweat from the skin results in a loss of 580 kcal of heat. During vigorous exercise, average hourly sweat loss in fit athletes is typically about 1 liter, sometimes more, sometimes less, depending upon the athlete's genetic predisposition to sweating, fitness level, exercise intensity, and environmental conditions. In other words, athletes are well equipped to stay safely cool during exercise in hot environments provided that ample sweat can evaporate from their skin.

The control of body temperature resides in the *preoptic anterior hypothalamus*, the body's thermostat. *Thermoreceptors* in the brain, skin, spine, and muscles provide constant feedback to the hypothalamus and to the *cerebral cortex*, making us consciously aware if we become too hot or too cold. As discussed previously, arterioles are under control of the sympathetic nervous system and can dilate or constrict in response to changes in body temperature. Millions of sweat glands respond to increases in core temperature by secreting salty water on the skin's surface for evaporative heat loss. In cold environments, skeletal muscles can shiver involuntarily to increase heat production.

Exercise in the heat and cold can be limited by both physiological and psychological factors. Both environments can become quickly uncomfortable, sapping the psychological drive or motivation to continue exercise. The cardiovascular system is also limited in its capacity to maintain sufficient cardiac output to meet the many competing demands for blood flow, particularly in the face of a progressively falling blood volume, as occurs with dehydration. When core temperature rises too high, brain function can be adversely affected, as can muscle function. Under most circumstances, athletes respond to the combination of these signals by slowing down or stopping exercise, allowing core temperature to decrease. But when athletes disregard these signals, or when coaches push athletes too hard, heat exhaustion and heat stroke can occur.

Fortunately, the human body can acclimate to heat stress and, to a lesser extent, to cold stress. Heat acclimation begins to occur within days of training in warm environments and, within 2 weeks of proper conditioning, most of the important physiological adaptations have occurred. Acclimated athletes drink more, have a larger blood volume, sweat sooner, sweat more, sweat over more of their bodies, and their sweat glands conserve more sodium to help maintain a larger blood volume. Cardiac output and skin blood flow both increase as a result of heat acclimation, heart rate during exercise is lower, core temperature is lower during both rest and exercise, and performance in the heat is improved.

Acclimation to the cold is less clear-cut than is heat acclimation. Enhanced balance between skin vasoconstriction (to conserve heat) and vasodilation (to prevent frostbite), an increase in metabolic heat production (both shivering and non-shivering *thermogenesis*), and the ability to tolerate a decrease in core temperature have been reported. Such adaptations would help reduce the risk of hypothermia and frostbite. The muscles controlling the fingers and toes are most susceptible to becoming cold and losing force production and dexterity, an experience than anyone living in or visiting cold climates has likely experienced. Cold muscles contract with less force than warm muscles, but proper clothing insulation, a constant supply of warm blood, and muscle heat production with exercise usually are sufficient to keep muscles from getting cold. In that regard, fatigue and the fall in heat production that accompanies it can reduce core temperature and increase the risk of hypothermia. From a nutrition perspective, maintaining blood glucose during prolonged exposure to the cold is important for prolonging the onset of fatigue and maintaining the muscles' capacity to shiver.

Fatigue

We all know fatigue when we feel it, but for purposes of science, fatigue is defined as *a decrement in muscular performance with continued effort*, often accompanied by general sensations of "tiredness." An alternative definition is *the inability to maintain the required power output to continue muscular work at a given intensity*. Unlike muscle weakness or damage, fatigue is reversible after rest.

Lactic acid is commonly cited by laypersons as the cause of muscle fatigue. Not only is this an oversimplification of the numerous factors that cause fatigue, but mounting evidence suggests that lactic acid plays a positive role in sustaining exercise. Lactate molecules produced by glycolysis in the cytoplasm of a muscle cell (Figure 2.2) can be taken up by the mitochondria within that same cell and directly oxidized to form ATP, particularly in cells with a high density of mitochondria (type I muscle cells). Second, lactate produced primarily by type II muscle cells can be transported to adjacent type I cells by diffusion or active transport. Third, lactate can be transported out of muscle cells and through circulation to other cells where it can be directly oxidized, a process referred to as the *lactate shuttle*. Finally, some of the lactate produced in the muscle is transported to the liver, where it is reconverted to pyruvic acid and back to glucose, then transported back to the working muscle (*the Cori cycle*). Without such lactate recycling, prolonged exercise would be limited.

Fatigue is a complex phenomenon and the specific mechanism of fatigue depends on the type and intensity of the exercise, the cell type of the involved muscles, the subject's training status, and even diet. Many questions about fatigue remain unanswered, especially about cellular sites of fatigue within the muscle cells. In reality, fatigue is rarely caused by any one single factor but by multiple factors acting at multiple sites. Plausible sites of fatigue include the active muscle (limited energy delivery, accumulation of metabolites, failure of the contractile machinery) and alterations in neural control of muscle contraction (at the motor-unit level or at the brain or CNS).

Energy Depletion

During repeated maximal contractions, fatigue coincides with local *phosphocreatine* (PCr) depletion. As PCr is depleted, the ability to quickly replace ATP is hindered and muscle ATP concentration also decreases. Additionally, inorganic phosphate (P_i), which increases during intense short-term exercise

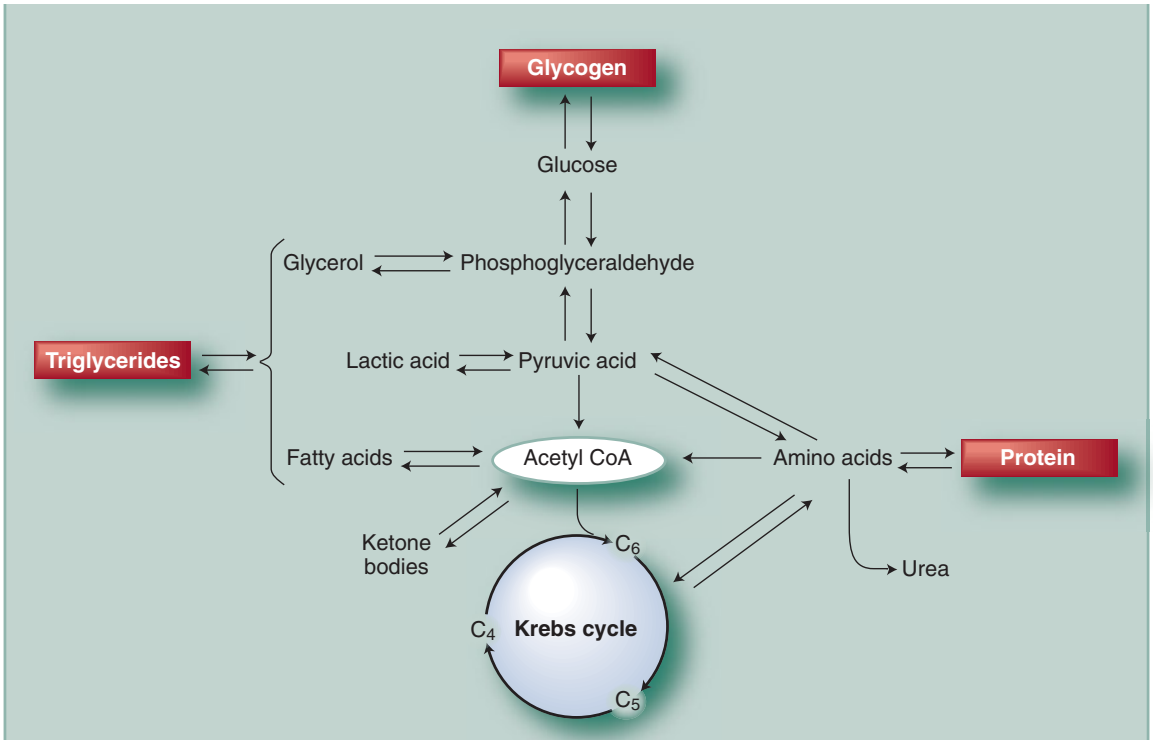

Figure 2.2 Lactic acid is formed during glycolysis, an anaerobic form of energy production from carbohydrates.
Source: Adapted with permission from Wilmore et al. (2008b).

because of the breakdown of PCr, has emerged as a potential cause of fatigue in short-duration, high-intensity exercise. In events lasting longer than a few seconds, muscle glycogen becomes the primary source of ATP synthesis. As with PCr, studies have shown a correlation between muscle glycogen depletion and fatigue during some types of exercise. The sensation of fatigue in long-duration exercise coincides with a decreased concentration of muscle glycogen, but not with its rate of depletion. Marathon runners, especially but not exclusively inexperienced runners, commonly experience a sudden onset of fatigue ("hitting the wall") around 30–35 km (19–22 miles) and at least part of this sensation can be attributed to muscle glycogen depletion.

In exercise lasting several hours, the liver breaks down stored glycogen to provide a constant supply of blood glucose. In the early stages of exercise, energy production requires relatively little blood glucose, but in the later stages of an endurance event, blood glucose makes a large contribution. Liver glycogen stores are limited and the liver cannot produce glucose rapidly from other substrates. Consequently, blood glucose concentration can decrease when muscle uptake exceeds the liver's glucose production. Unable to obtain sufficient glucose from the blood, the muscles must rely more heavily on their glycogen reserves, accelerating muscle glycogen depletion and leading to earlier exhaustion.

The depletion of muscle glycogen may be the first step in a series of events that leads to fatigue during prolonged exercise, in part because a minimal rate of glycogen breakdown is needed for optimal oxidative ATP production from carbohydrates and fats. Further, as glycogen is depleted, exercising muscle relies more heavily on the metabolism of free fatty acids (FFAs), which must be transported into the mitochondria. The rate of transfer may limit FFA oxidation to the point where FFA transfer can no longer keep up with the need for fat oxidation. In addition, FFAs cannot be oxidized as rapidly as carbohydrate to form the ATP at the rate required to sustain vigorous exercise. It is important to note

too that, as pointed out in Chapter 1, the use of fat requires more oxygen per unit of energy produced. Where oxygen supply is limited, there is a major advantage to using carbohydrate as a fuel.

Metabolic By-products

Various by-products of metabolism have been implicated in fatigue, in addition to the aforementioned inorganic phosphate. Additional metabolic by-products that have received the most attention in discussions of fatigue are heat, lactate, and hydrogen ions.

Muscle contraction generates heat, some of which is retained in the body, causing core temperature to rise. It has been hypothesized that high muscle temperatures can impair both skeletal muscle function and metabolism. As noted in the section "Thermoregulatory Function," the ability to continue vigorous exercise is affected by ambient temperature. Galloway and Maughan (1997) studied time to exhaustion in male cyclists at four different air temperatures: 4°C (39°F), 11°C (52°F), 21°C (70°F), and 31°C (88°F), with results shown in Figure 2.3.

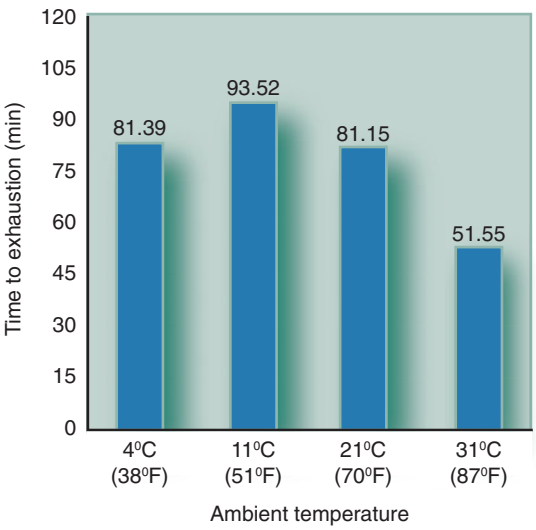

Figure 2.3 Time to exhaustion for a group of men performing cycle exercise at about 70% VO_2 max. The subjects were able to perform longer (delay fatigue longer) in a cool environment of 11°C. Exercising in colder or warmer conditions hastened fatigue. Redrawn from Galloway and Maughan (1997).

Exercise time was longest at an air temperature of 11°C and was shorter at colder and warmer temperatures, and fatigue occurred earliest at 31°C. Precooling of muscles similarly prolongs exercise, while preheating causes earlier fatigue.

Short sprints may lead to large accumulations of lactic acid. But as previously described, the presence of lactic acid does not solely or directly account for fatigue. Lactic acid dissociates into a lactate molecule and a hydrogen ion, with the accumulation of hydrogen ions resulting in acidosis, a drop in intracellular pH. Because cells and body fluids contain buffers, including bicarbonate, hydrogen ion concentration remains relatively low even during the most severe exercise, limiting the fall in muscle pH to approximately 6.4–6.6 at exhaustion. However, pH changes of this magnitude adversely affect energy production and muscle contraction by slowing the rate of glycolysis, displacing calcium ions within the muscle cell, interfering with actin–myosin cross-bridge formation, and decreasing contractile force. Low muscle pH may be the primary cause of fatigue during all-out exercise lasting from about 30 seconds up to 10–15 minutes.

Neuromuscular Fatigue

Under some circumstances, fatigue may result from an inability of motor nerves to activate muscle cells. Any of several neural mechanisms, both peripheral and central, can contribute to fatigue. Fatigue may be related to events at the neuromuscular junction, including a reduction in the release or synthesis of ACh, alterations in acetylcholinesterase activity, an increased cell membrane stimulation threshold, and accumulation of ACh competitors and/or potassium that may decrease the cell's membrane potential. Some evidence also suggests that fatigue may also be attributable to retention of calcium ions within the sarcoplasmic reticulum, which would decrease the calcium available for muscle contraction.

Without doubt, there is CNS involvement in most types of fatigue. When muscles appear to be fatigued, verbal encouragement, shouting, playing music, and direct electrical stimulation of the muscle can all increase the strength of muscle contraction.

The precise mechanisms underlying the CNS role in causing, sensing, and even overriding fatigue are not fully understood.

Principles of Training

Delaying the onset of physical and mental fatigue is a primary goal of most training programs and of related nutritional interventions such as fluid replacement and carbohydrate feeding. A common denominator among the various types of physical training is the increased production inside muscle cells of the proteins required for muscle contraction, the structural proteins that form the intracellular framework that supports the contractile proteins, and the regulatory proteins, enzymes, signaling molecules, and organelles that allow for heightened protein synthesis and energy metabolism. In simple terms, training is the stimulus that provokes thousands of intracellular reactions that culminate in the production of new proteins to enhance the capacity of muscle and other tissues for exercise. Of equal importance are the changes that take place in the brain and CNS, as well as in the endocrine, cardiorespiratory, and immune systems, that support the improvements in motor skills and physical capacity. An additional consideration is that all of these adaptations can be acutely and chronically influenced by the athlete's nutrition intake.

There are a few key principles that govern the physiological and metabolic responses to exercise training. For example, the responses to a given training program are known to vary widely among athletes. Some people are "high responders" who adapt quickly to the demands of training and show quick improvement in performance. Other athletes are "low responders" and show little-to-no adaptation or performance improvement in response to the same training regimen. The difference among individuals in the response to training is referred to as the *principle of individuality*.

Responses to training are also specific to the type of training performed. This knowledge forms the *principle of specificity*. To achieve the optimal responses, training programs must be designed to stress the physiological and metabolic systems that are critical for performance. However, as discussed below, research demonstrates that endurance athletes can benefit from training that might not be considered specific to the physiological and metabolic systems critical to endurance performance, a finding that suggests the need for a less-strict definition of the principle of specificity. At the other end of the training spectrum, the *principle of reversibility* holds that the adaptations to training will eventually be lost if training is substantially decreased or stopped.

Perhaps the most important training principle is the *principle of progressive overload*, the understanding that gradual, systematic increases in training frequency, intensity, and duration lead to improvements in exercise capacity and performance. Closely related to the principle of progressive overload is the *principle of variation*, also referred to as the *principle of periodization*. Systematic variations in training mode, volume, and intensity can help achieve peak performance by introducing new stressors to promote additional adaptations.

Repeated days and weeks of training can be considered a positive "stress" because the adaptations caused by that cumulative stress typically improve the capacity for energy production, oxygen delivery, muscle contractions, and many other adaptations that enhance exercise performance. Research indicates that the major physiological and metabolic changes associated with training typically occur within the first 2–3 months. The rate and extent of training adaptations appear to be genetically limited, the scientific basis for the *principle of individuality*. However, regardless of genetic limitations, it is clear that exercise training represents a stress to which the body can effectively respond if given adequate rest and nutrition, the basic requirements for optimizing the signaling mechanisms that provoke the desired physiological and metabolic responses.

It is also clear that the stress of training can have a negative effect on training responses, resulting in failure to adapt and poorer performance. Coaches and athletes alike are often fearful that they have undertrained and that performance will plateau or decline over time. More often, it is arguably too much training that causes performance plateaus and decrements. Whenever the training load exceeds the athlete's ability to adapt, performance will suffer until the desired adaptations eventually occur, possibly

requiring a temporary reduction in training load. In that regard, the athlete might be considered to have *overreached* in their training relative to their ability to adapt. Athletes who persist with high-volume training in the face of continued performance deficits risk suffering from *overtraining*, a state of prolonged physical and mental fatigue that may require weeks or months of rest to remedy. Symptoms of overtraining include loss of appetite, loss of body weight, disturbed sleep, irritability, lack of motivation, difficulty concentrating, and feelings of depression. The physiological and metabolic responses to the overtraining syndrome are not well understood, but it is thought that failure of skeletal muscles to continue to adapt to the stresses of training along with changes in the brain, alterations in neurotransmitter chemistry, impaired immune response, and altered neuroendocrine response are also likely to be involved.

Although the *principle of specificity* holds that training responses are specific to the type of training performed, research shows that short-duration, high-intensity interval training can produce physiological and metabolic adaptations that are typically associated with prolonged endurance training (Gibala, 2007). Similar improvements in muscle oxidative capacity and exercise capacity have been reported in subjects who completed 2.5 hours of high-intensity interval training (with a total exercise time of only 15 minutes) compared to subjects who undertook 10.5 hours of conventional endurance training. These findings suggest that recruiting both type I and type II muscle cells provokes intracellular adaptations that improve aerobic capacity and endurance performance. More research is needed to create a broader scientific understanding of how high-intensity training improves endurance performance and to uncover how high-intensity training is best integrated into an endurance athlete's training regimen.

Summary

Exercise physiology and sports nutrition are closely connected. There is little doubt that proper nutrition plays an indispensable role in helping athletes maintain, recover from, and adapt to rigorous training and competition. For example, it has long been recognized that minimizing dehydration during exercise helps protect health and performance by minimizing negative perturbations in cardiovascular and thermoregulatory function (Sawka et al., 2007). Ingesting simple carbohydrates improves performance capacity during intense and prolonged exercise by maintaining blood glucose concentration and muscle glucose uptake and enhancing overall carbohydrate oxidation (Rodriguez et al., 2009). Consuming ample amounts of carbohydrate following exercise and throughout each day is required to restore and maintain the high muscle and liver glycogen stores necessary for training and competition (Rodriguez et al., 2009). The consumption of high-quality proteins after exercise and with meals and snacks supplies the essential amino acids required for the recovery, repair, and growth of muscle tissue, a critical element of any training program (Rodriguez et al., 2009). Supplementation of the athlete's diet with creatine has been shown to improve muscle PcCr stores and improve performance in short-duration exercise tasks that require repeated explosive movements (Rodriguez et al., 2009) and ingestion of foods high in nitrate content (e.g., spinach and beets) is associated with improved exercise capacity and lower oxygen cost of physical activity (Lansley et al., 2011a, 2011b). Consumption of β-alanine, conjugated linoleic acid, sodium bicarbonate, ω3, 6, 9 fatty acids, and juice flavonoids and polyphenols are yet other examples of how nutrition may influence physiological and metabolic responses (Burke et al., 2009).

References

Burke, L.M., Castell, L.M., & Stear, S.J. (2009) BJSM reviews: A–Z of supplements: dietary supplements, sports nutrition foods and ergogenic aids for health and performance. Part 1. *British Journal of Sports Medicine* 43, 728–729.

Chin, E.R. (2010) Intracellular Ca2+ signaling in skeletal muscle: decoding a complex message. *Exercise and Sports Sciences Reviews* 38, 76–85.

Galloway, S.D.R. & Maughan, R.J. (1997) Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Medicine & Science in Sports & Exercise* 29, 1240–1249.

Gibala, M.J. (2007) High-intensity interval training: a time-efficient strategy for health promotion? *Current Sports Medicine Reports* 6, 211–213.

Hurd, S. & Lenfant, C. (1987a) Pulmonary research: what topics? What costs? *The American Review of Respiratory Disease* 135, 521–522.

Hurd, S. & Lenfant, C. (1987b) Pulmonary research: where have we come and what does the future hold? *The American Review of Respiratory Disease* 136, 542–544.

Joyner, M.J. (2011) Physiology: alone at the bottom, alone at the top. *Journal of Physiology* 589, 1005.

Kenney, W.L., Wilmore, J. H., & Costill, D.L. (2012) *Physiology of Sports and Exercise*, 5th edn. Human Kinetics, Champaign, IL.

Lansley, K.E., Winyard, P.G., Bailey, S.J., et al. (2011a) Acute dietary nitrate supplementation improves cycling time trial performance. *Medicine & Science in Sports & Exercise* 43, 1125–1131.

Lansley, K.E., Winyard, P.G., Fulford, J., et al. (2011b) Dietary nitrate supplementation reduces the O_2 cost of walking and running: a placebo-controlled study. *Journal of Applied Physiology* 110, 591–600.

Meyer, R.A. & Wiseman, R.W. (2012) The metabolic systems: control of ATP synthesis in skeletal muscle. In *ACSM's Advanced Exercise Physiology* (eds. P.A. Farrell, M.J. Joyner, & V.J. Caiozzo), 2nd edn., pp. 363-–378,. Lippincott Williams & Wilkins, Philadelphia, PA, .

Rodriguez, N.R., Di Marco, N.M., & Langley, S. (2009) American College of Sports Medicine, American Dietetic Association, Dietitians of Canada position stand. Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41, 709–731.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.

Wagner, P.D. & Paterson, D. (2011) Physiology: found in translation. *Journal of Applied Physiology* 589, 3899–3900.

Williams, J.H., Powers, S.K., & Stuart, M.K.(1986) Hemoglobin desaturation in highly trained athletes during heavy exercise. *Medicine & Science in Sports & Exercise* 18, 168–173.

Wilmore, J.H., Costill, D.L., & Kenney, W.L.(2008a) *Physiology of Sport and Exercise*, 4th edn. p. 28, Human Kinetics, Champaign, IL.

Wilmore, J.H., Costill, D.L., & Kenney, W.L.(2008b) *Physiology of Sport and Exercise*, 4th edn. p. 56, Human Kinetics, Champaign, IL.

Chapter 3

Biochemistry of Exercise

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Introduction

Biochemistry is the study of events such as reactions, energy transfer, signaling, and transport processes at the subcellular and molecular levels, and an in-depth understanding of sport nutrition requires some knowledge of these processes. This chapter describes the sources of energy available for muscle force generation and explains how acute exercise modifies energy metabolism through intracellular effects and the action of hormones (for further details, see the books by Maughan & Gleeson (2010) and Hargreaves & Spriet (2006)). Both the diet before exercise and feeding during exercise influence the hormonal and metabolic responses to exercise. Training also modifies the metabolic response to exercise, and training-induced adaptations encompass both biochemical responses (e.g., changes in gene expression, protein content, and enzyme activities in trained muscles) and physiological responses (e.g., changes in the local capillary network, heart, maximal oxygen uptake). These adaptations are determined largely by the mode of exercise and the intensity, frequency, and volume of the exercise stimulus. Training-induced adaptations of skeletal muscle are also influenced to some degree by the composition and timing of nutrient intake. Some dietary components when ingested in sufficient amounts acutely before and/or during exercise or chronically during training can have performance-enhancing (ergogenic) effects. Thus, influences of diet and training on biochemical aspects of the acute response to exercise and the chronic adaptation to training are also briefly described in this chapter.

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Subcellular Skeletal Muscle Structure

Overview

Anatomical and physiological aspects of muscle structure are covered in Chapter 2, so will be summarized only briefly here. Muscles are composed of long, cylindrical cells called fibers. These fibers contain the internal organelles and structures that allow muscle to contract and relax. Individual muscles are made up of many parallel fibers that may extend the entire length of the muscle. Inside the muscle fiber is the sarcoplasm (muscle cell cytoplasm), a red viscous fluid containing nuclei, mitochondria, myoglobin, and about 500 thread-like 1–3 mm thick myofibrils that are continuous from end to end in the muscle fiber. Surrounding the myofibrils is an elaborate bag-like membranous structure called the sarcoplasmic reticulum (SR). Its interconnecting membranous tubules lie in the narrow spaces between the myofibrils, surrounding and running parallel to them. Energy is stored in the sarcoplasm as fat (triacylglycerol droplets), glycogen, a small pool of free amino acids (most of which are not used for energy metabolism), phosphocreatine (PCr), and adenosine triphosphate (ATP).

Muscle Contraction

The myofibrils are composed of overlapping thin and thick filaments made of protein, and the arrangement

of these filaments gives skeletal muscle its striated appearance when viewed through a microscope. The thick filaments are composed of myosin molecules, each of which consists of a rod-like tail and a globular head containing ATPase activity sites and actin-binding sites. ATP is the energy currency of the cell. The breakdown of ATP to adenosine diphosphate (ADP) and inorganic phosphate (P_i) by the myosin ATPase provides the energy for muscle contraction. The thin filaments are composed of actin molecules and the regulatory proteins (e.g., tropomyosin and troponin). When calcium ions (Ca^{2+}) and ATP are present in sufficient quantities, the filaments form actomyosin and shorten by sliding over each other. Hydrolysis of ATP to ADP and P_i by the myosin ATPase provides the energy required for crossbridge formation and the return of myosin to its activated state, giving it the potential energy needed for the next crossbridge cycle.

Fiber Types

Human muscles contain a mixture of the three different fiber types—I, IIa, and IIX—although the proportions in which these types are found differ substantially among muscles and also among individuals (Komi & Karlsson, 1978). The myosin of the different fiber types exists in different molecular forms (isoforms), and the myofibrillar ATPase activity of the different isoforms displays differential pH sensitivity that provides the basis for the chemical staining and identification of fiber types. The biochemical characteristics of the three fiber types are summarized in Table 3.1.

Type I fibers are small-diameter red cells that contain relatively slow-acting myosin ATPases and hence contract slowly. The red color is caused by myoglobin, which is capable of binding oxygen and releasing it only at very low partial pressures. Type I fibers have numerous energy-producing

Table 3.1 Biochemical characteristics of human muscle fiber types. Values of metabolic characteristics of Type II fibers are shown relative to those found in Type I fibers.

Characteristic	Type I	Type IIa	Type IIX
Nomenclature	Slow, Red Fatigue resistant	Fast, Red Fatigue resistant	Fast, White Fatiguable
Metabolic character	Oxidative	Oxidative/Glycolytic	Glycolytic
Myosin ATPase activity	1.0	>2	>2
ATPase activity at pH 10.3	0	High	High
ATPase activity at pH 4.6	0	0	High
Capillary density	1.0	0.8	0.6
Mitochondrial density	1.0	0.7	0.4
Myoglobin content	1.0	0.6	0.3
Phosphorylase activity	1.0	2.1	3.1
PFK activity	1.0	1.8	2.3
Citrate synthase activity	1.0	0.8	0.6
SDH activity	1.0	0.7	0.4
Phosphocreatine content	1.0	1.2	1.2
Glycogen content	1.0	1.3	1.5
Triacylglycerol content	1.0	0.4	0.2
Carnosine content	1.0	1.7	2.3

ATPase = Adenosine triphosphatase
PFK = Phosphofructokinase
SDH = Succinate dehydrogenase

mitochondria, mostly located close to the periphery of the fiber, near the blood capillaries that provide a rich supply of oxygen and nutrients. Type I fibers possess a high capacity for oxidative metabolism, are extremely fatigue resistant, and are specialized for repeated contractions over prolonged periods. Not surprisingly, the leg muscles of endurance athletes possess a high proportion of type I fibers.

Type IIX fibers contain little myoglobin and so are relatively pale in color. They possess rapidly acting myosin ATPases; therefore, their contraction (and relaxation) time is relatively fast. These fibers have fewer mitochondria and a poorer capillary supply, but they have greater glycogen and PCr stores compared with the type I fibers (Table 3.1). High glycogenolytic and glycolytic enzyme activity gives type IIX fibers a high capacity for rapid (but relatively short-lived) ATP production in the absence of oxygen (anaerobic capacity). Lactate and hydrogen ions accumulate quickly in these fibers and they fatigue rapidly. Thus, these fibers are best suited for delivering rapid, powerful contractions for brief periods. The metabolic characteristics of type IIa fibers lie between the extreme properties of type I and type IIX fibers. Type IIa fibers contain fast-acting myosin ATPases like the type IIX fibers but have an oxidative capacity more like that of type I fibers. Not surprisingly, the leg muscles of sprint and power athletes possess a high proportion of type II fibers.

The differences in activation threshold of the motor neurons supplying the different fiber types determine the order in which fibers are recruited during exercise, and in turn influence the metabolic response to exercise. During most forms of movement, an orderly hierarchy of motor unit recruitment occurs that roughly corresponds with a progression from type I to type IIa to type IIX. Light exercise uses mostly type I fibers, moderate exercise uses both type I and type IIa fibers, and severe exercise uses all fiber types.

Energy for Muscle Force Generation

ATP Hydrolysis

Energy availability is a potential prerequisite for performing work or producing force. ATP is the only source of energy that can be used directly not only for muscle contraction but also for other energy-requiring processes in the cell such as protein synthesis and the reuptake of Ca^{2+} into the SR and the sarcolemmal Na^+–K^+–ATPase (commonly known as the sodium pump).

The hydrolysis of ATP yields approximately 31 kJ of free energy per mole of ATP degraded to ADP and P_i:

$$ATP + H_2O \rightarrow ADP + H^+ + P_i - 31 \text{ kJ per mole of ATP}$$

The resting concentration of ATP in skeletal muscle is about 4–5 mmol/kg wet weight (w.w.) of muscle, which alone can only provide enough energy to sustain a few seconds of intense exercise. Since depletion of ATP would be fatal to the cell, the ATP concentration must be maintained by resynthesis from ADP at essentially the same rate at which ATP is being broken down.

ATP Resynthesis

Three mechanisms are involved in the resynthesis of ATP for muscle force generation: (1) PCr hydrolysis; (2) glycolysis, which involves the metabolism of glucose-6-phosphate (G6P), derived from muscle glycogen or blood-borne glucose, and produces ATP by substrate-level phosphorylation reactions; and (3) the products of carbohydrate, fat, protein, and alcohol metabolism enter the tricarboxylic acid (TCA) cycle in the mitochondria and are oxidized to carbon dioxide and water; this process is known as oxidative phosphorylation and yields energy for the synthesis of ATP.

These mechanisms regenerate ATP at sufficient rates to prevent a significant fall in the intramuscular ATP concentration. PCr breakdown and glycolysis are anaerobic mechanisms (i.e., do not require oxygen) that occur in the sarcoplasm. Each uses only one specific substrate for energy production: PCr and G6P, respectively. The aerobic processes in the mitochondria utilize a variety of different substrates, and the sarcoplasm contains a variety of enzymes that can convert carbohydrates, fats, and proteins into usable substrate, primarily a two-carbon acetyl group linked to coenzyme A (acetyl-CoA), which can be completely oxidized in the mitochondria with the resultant production of ATP. A general summary of the main energy sources and pathways of energy metabolism is presented in Figure 3.1.

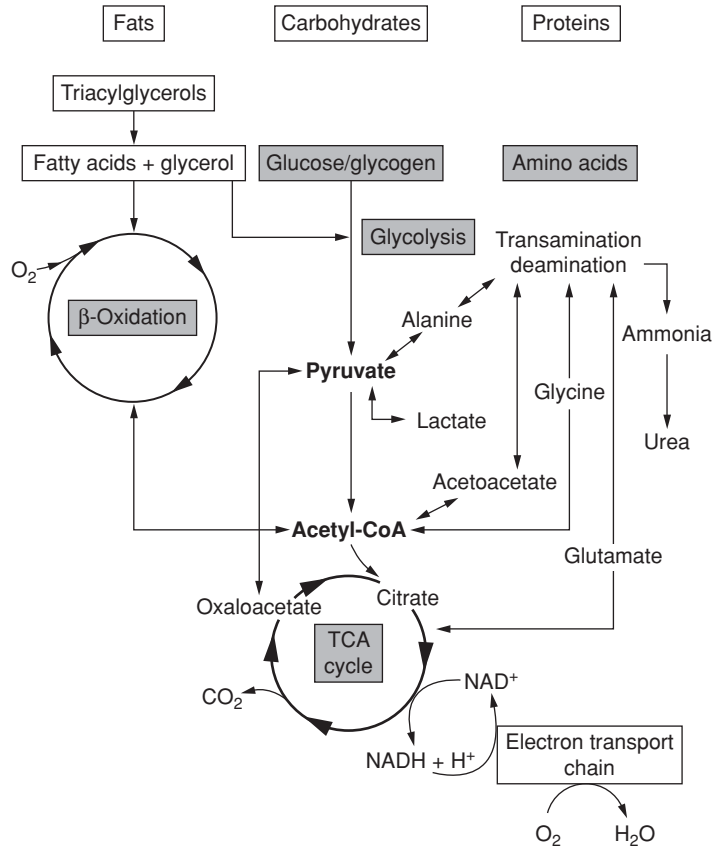

Figure 3.1 Summary of the main pathways of energy metabolism using carbohydrate, fats, and proteins as energy sources. Carbohydrate may participate in both anaerobic and aerobic pathways. In glycolysis, glucose or glycogen is broken down to lactate under anaerobic conditions and pyruvate under aerobic conditions. The pyruvate is converted to acetyl-coenzyme A (CoA) and is completely oxidized in the tricarboxylic (TCA) cycle. Fats in the form of triacylglycerols are hydrolyzed to fatty acids and glycerol, the latter entering the glycolytic pathway (in the liver but not in muscle) and the fatty acids being converted via the *β*-oxidation pathway to acetyl-CoA and subsequently oxidized in the TCA cycle. Protein catabolism can provide amino acids that can be converted by the removal of the amino group into either TCA cycle intermediates or into pyruvate or acetoacetate and subsequent transformation to acetyl-CoA.

Phosphocreatine Breakdown

Some of the energy for ATP resynthesis is supplied rapidly and without the need for oxygen via the breakdown of PCr which is found within the muscle fiber at a concentration that is three to four times greater than that of ATP. When PCr is broken down to creatine and P_i by the action of the enzyme creatine kinase (CK), a large amount of free energy is released (43 kJ per mole of PCr) that is used to restore ATP as soon as it begins to fall during exercise:

$$ADP + PCr + H^+ \rightarrow ATP + Cr - 43 \text{ kJ per mole of PCr}$$

Note that the resynthesis of ATP through the breakdown of PCr buffers some of the hydrogen ions (H^+) formed as a result of ATP hydrolysis. This action helps to prevent a rapid acidification of the muscle sarcoplasm, which could induce premature failure of the contractile mechanism.

During extremely intense exercise, PCr can be almost completely depleted. The reactions of ATP and PCr hydrolysis are reversible, however, and when energy is readily available from other sources (oxidative phosphorylation), creatine and phosphate can be rejoined to form PCr.

Table 3.2 Capacity and power of anaerobic systems for the production of ATP

	Capacity mmol ATP/kg d.m.	Power mmol ATP/kg d.m./s
Phosphagen system	55–95	9
Glycolytic system	190–300	4.5
Combined	250–370	11

Values are expressed per kg dry mass (d.m.) of muscle and are based on estimates of ATP provision during high intensity exercise of human vastus lateralis muscle.

The PCr in muscle is immediately available at the onset of exercise and can be used to resynthesize ATP very quickly. This rapid energy transfer corresponds to the ability to produce high-power output (see Tables 3.2 and 3.3). The major disadvantage of this process compared with other means of regenerating ATP is its limited capacity; the total amount of energy available is small. Studies on dietary creatine supplementation (~20 g/day for 1 week) indicate that the muscle PCr store can be increased by ~10% and muscle-free creatine by ~40% and that this can improve the performance of repeated sprints (Greenhaff, 2000; Hespel et al., 2006).

An additional pathway to regenerate ATP when ATP and PCr stores are depleted is through a kinase reaction that utilizes two molecules of ADP to generate one molecule of ATP (and one molecule of adenosine monophosphate (AMP)). This reaction is catalyzed by the enzyme myokinase:

$$ADP + ADP \rightarrow ATP + AMP - 31 \text{ kJ per mole of ADP}$$

This reaction becomes important only during high-intensity exercise. Even then, the amount of energy available in the form of ATP is extremely limited, and the real importance of the reaction may be the formation of AMP, which is a potent activator of a number of enzymes involved in energy metabolism.

Table 3.3 Maximal rates of ATP resynthesis from anaerobic and aerobic metabolism and approximate delay time before maximal rates are attained following onset of exercise

	Max rate of ATP resynthesis mmol ATP/kg d.m./s	Delay time
PCr breakdown	9.0	0–2 seconds
Glycolysis	4.5	5–10 seconds
Glycogen oxidation	2.8	5–10 minutes
Glucose (from blood) oxidation	1.2	~90 minutes
Fat oxidation	1.0	>2 hours
Amino acid oxidation	0.1	>2 hours

ATP, adenosine triphosphate.

Glycolysis

Glycolysis involves the breakdown of glucose (or glycogen) in a series of chemical reactions that yield pyruvate. This process does not require oxygen but does result in a rapid rate of ATP synthesis from reactions involving substrate-level phosphorylation. The pyruvate must be removed (allowing regeneration of the oxidized form of the essential cofactor nicotinamide adenine dinucleotide (NAD^+)) for the reactions to proceed and the rate of ATP resynthesis via this means is somewhat slower than for PCr breakdown (Tables 3.2 and 3.3). In low-intensity exercise, when adequate oxygen is available to the muscle, pyruvate is converted to carbon dioxide and water by oxidative metabolism in the mitochondria. In some situations when oxygen availability is limited (e.g., isometric exercise that occludes muscle blood flow) or when the rate of formation of pyruvate is extremely high (e.g., sprinting), the pyruvate can also be removed by conversion to lactate, a reaction that does not involve oxygen.

Muscle Uptake of Glucose from the Blood

A specific transporter protein, GLUT4, carries glucose molecules across the sarcolemmal membrane. After the glucose molecule is inside the muscle cell, an irreversible phosphorylation (addition of a phosphate group) reaction, catalyzed by the enzyme hexokinase, occurs to prevent the loss of glucose from the cell. The glucose is converted to G6P using ATP as the phosphate donor. Skeletal muscles lack the enzyme glucose-6-phosphatase and so are not able to re-form free glucose following the formation

of G6P. Thus, the addition of a phosphate group to glucose ensures that the glucose is effectively trapped inside the cell. Note that this is an important difference between skeletal muscle and the liver. In the liver, which contains glucose-6-phosphatase, G6P can be broken down to form free glucose that can be released into the blood. In this way the liver plays an important role in the maintenance of the blood glucose concentration, whereas muscle tissue cannot do so directly. Hexokinase is inhibited by an accumulation of G6P, and the increasing concentration of G6P during high-intensity exercise limits the contribution that the blood glucose can make to carbohydrate metabolism in the active muscles.

Muscle Glycogen Breakdown

If glycogen, rather than blood glucose, is the substrate for glycolysis, a single glucose molecule is split off by the enzyme glycogen phosphorylase, and the products are glucose-1-phosphate and a glycogen molecule that is one glucose residue shorter than the original. The substrates are glycogen and P_i, so, unlike the hexokinase reaction, no breakdown of ATP occurs. Phosphorylase acts on the α-1,4 carbon bonds at the free ends of the glycogen molecule but cannot break the α-1,6 bonds forming the branch points. These bonds are hydrolyzed by the combined actions of a debranching enzyme and amylo-1,6-glucosidase, releasing free glucose, which is quickly phosphorylated to G6P by hexokinase.

Glycolytic Pathway from Glucose-6-Phosphate to Pyruvate

The enzyme phosphoglucomutase rapidly converts the glucose-1-phosphate formed by the action of phosphorylase on glycogen to G6P, which then proceeds down the glycolytic pathway (see Figure 3.2). After a further phosphorylation, the glucose molecule is cleaved to form two molecules of the three-carbon sugar glyceraldehyde-3-phosphate. The second stage of glycolysis is the conversion of glyceraldehyde-3-phosphate into pyruvate, accompanied by the formation of ATP and reduction of NAD^+ to NADH.

The net effect of glycolysis is the conversion of one molecule of glucose to two molecules of pyruvate, with the formation of two molecules of ATP, two molecules of water, two free hydrogen ions, and the conversion of two molecules of NAD^+ to NADH. The net equation is

$$\text{Glucose} + 2\text{ADP} + 2\text{P}_\text{i} + 2\text{NAD}^+ \rightarrow$$
$$2\text{Pyruvate} + 2\text{ATP} + 2\text{H}_2\text{O} + 2\text{NADH} + 2\text{H}^+$$

If glycogen rather than glucose is the starting substrate, three molecules of ATP are produced because no initial investment of ATP is made when the first phosphorylation step occurs. Although this net energy yield appears small, the relatively large carbohydrate store available and the rapid rate at which glycolysis proceeds make energy supplied in this way crucial for the performance of high-intensity exercise. The 800 m runner, for example, obtains about 60% of the total energy requirement from anaerobic metabolism and may convert about 100 g of carbohydrate (mostly glycogen and equivalent to about 550 mmol of glucose) to lactate in less than 2 minutes. The amount of ATP released in this way (three ATP molecules per glucose molecule degraded, about 1667 mmol of ATP in total) far exceeds the ATP available from PCr hydrolysis. This high rate of anaerobic metabolism not only allows a faster "steady state" speed than is possible with aerobic metabolism alone but also allows a faster pace in the early stages, before the cardiovascular system has adjusted to the demands and before the delivery and utilization of oxygen have increased in response to the exercise stimulus.

The reactions of glycolysis occur in the cytoplasm of the muscle cell, and some pyruvate will escape from active muscle tissues when the rate of glycolysis is high, but most is further metabolized. The fate of the pyruvate produced depends not only on factors such as exercise intensity but also on the metabolic capacity of the tissue. When glycolysis proceeds rapidly, the availability of NAD^+, which is necessary as a cofactor in the glyceraldehyde 3-phosphate dehydrogenase reaction, becomes limiting. Reduction of pyruvate to lactate will regenerate NAD^+ in muscle, and this reaction can proceed in the absence of oxygen. That is not to say, however, that lactate formation occurs only in the absence of

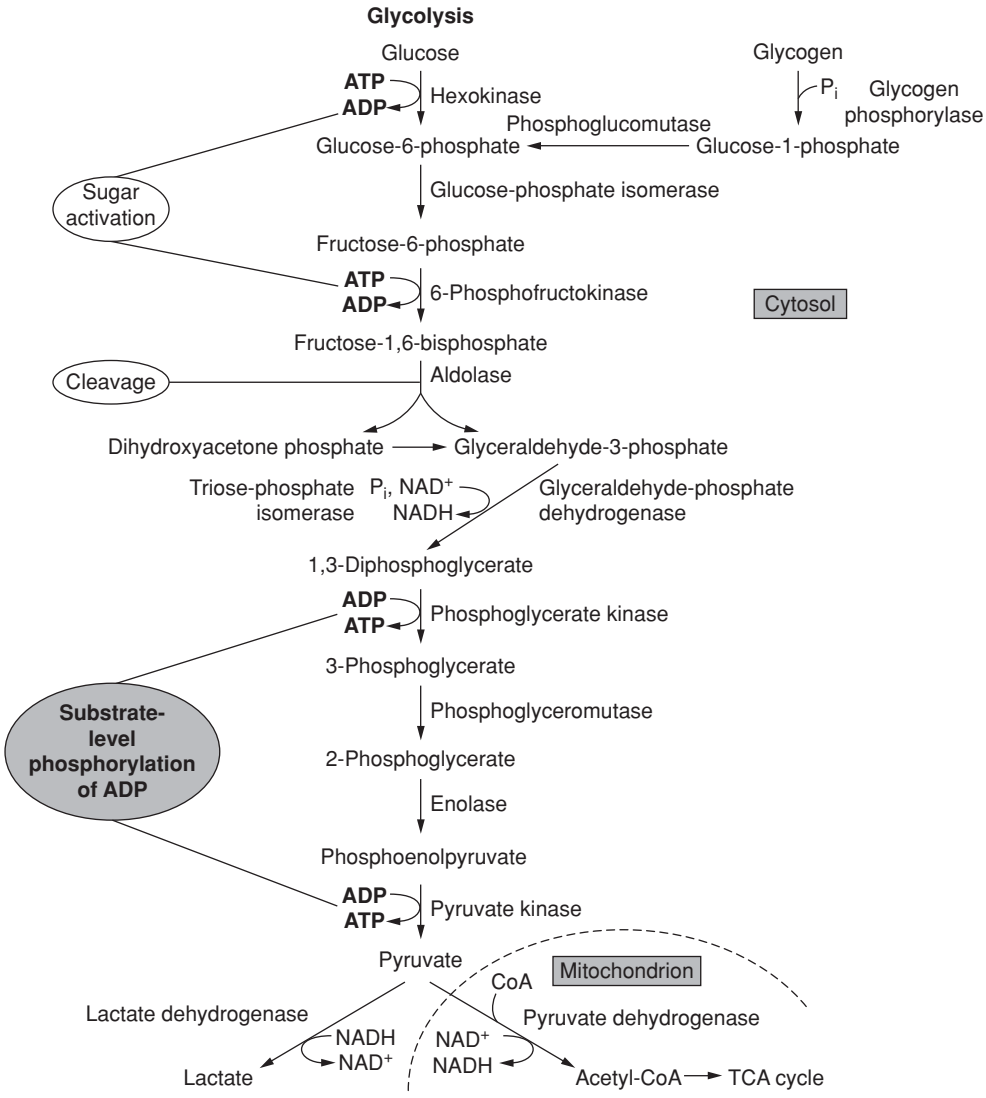

Figure 3.2 The reactions of glycolysis. Glucose, a six-carbon sugar, is first phosphorylated and then cleaved to form two molecules of the three-carbon sugar glyceraldehyde-3-phosphate, which is subsequently converted into pyruvate, accompanied by the formation of ATP and reduction of NAD^+ to NADH. Glycolysis makes two molecules of ATP available for each molecule of glucose that passes through the pathway. If muscle glycogen is the starting substrate, three ATP molecules are generated for each glucose unit passing down the pathway. Pyruvate may enter the mitochondria and be converted into acetyl-CoA or be reduced to form lactate in the cytosol. P_i, inorganic phosphate; TCA, tricarboxylic acid.

oxygen. Even at low exercise intensities, such as when walking, some lactate formation occurs. Lactate can accumulate within the muscle fibers, reaching much higher concentrations than those reached by any of the glycolytic intermediates including pyruvate.

When glycolysis is a major means of resynthesizing ATP, H^+ will accumulate causing intracellular

pH to fall, inhibiting some enzymes such as phosphorylase and phosphofructokinase (PFK), and the contractile mechanism begins to fail. A low pH also stimulates nociceptors in the muscle, causing the perception of pain. Although the negative effects of the acidosis are often stressed, the energy made available by anaerobic glycolysis allows the performance of high-intensity exercise that would otherwise not be possible. The main contributors to H^+ accumulation during high-intensity exercise are ATP breakdown and the H^+ associated with lactate formation during glycolysis. There is some debate as to the relative importance of these sources of H^+ generation and this issue is considered in more detail in Chapter 26.

Buffers Prevent Excessive Falls in pH

Processes that buffer (remove or mop up) H^+ as they start to accumulate are present in muscle. Proteins and phosphate ions act as chemical buffers as they are able to accept H^+. The monocarboxylate transporters located in the sarcolemmal membrane remove lactate and H^+ simultaneously from the muscle by cotransport into the interstitial fluid and the expression of these transporters is increased by high-intensity interval training (Pilegaard et al., 1999). The movement of H^+ out of the muscle can be enhanced by an increase in the extracellular fluid bicarbonate concentration. This can be achieved by acute oral sodium bicarbonate loading and ingesting a dose of ~0.3 g/kg b.m. 1–2 hours before exercise has been shown to improve performance in exercise lasting 2–6 minutes (Hespel et al., 2006; Wilkes et al., 1983). The dipeptide carnosine also plays an important role in the buffering of intracellular acidosis during high-intensity exercise. The normal carnosine concentration in untrained human mixed muscle is about 4 mmol/kg w.w. but can be as high as 10 mmol/kg w.w. in 800 m runners and rowers. High carnosine concentrations are found in individuals with a high proportion of type II fibers, because these fibers are enriched with the dipeptide. Muscle carnosine content is lower in women, declines with age, and is lower in vegetarians, whose diets do not contain β-alanine. Sprint-trained athletes display markedly high muscular carnosine, but the effect of several weeks of high-intensity training on muscle carnosine is rather small. High carnosine levels in elite sprinters are therefore either genetically determined or a result of slow adaptation to years of training. Recent studies have shown that the chronic oral ingestion of β-alanine can elevate the carnosine content of human skeletal muscle by up to 80% and that such muscle carnosine loading leads to improved performance in high-intensity exercise in both untrained and trained individuals (Artioli et al., 2010; Derave et al., 2010; Sale et al., 2010). β-alanine supplementation is now common among athletes in high-intensity sports, and research studies have proliferated in recent years.

Carbohydrate Oxidation in Aerobic Metabolism

Pyruvate may also undergo oxidative metabolism to carbon dioxide (CO_2) and water. This process occurs within the mitochondrion, and pyruvate that is produced in the sarcoplasm is transported across the mitochondrial membrane by a specific monocarboxylic acid transporter. The three-carbon pyruvate is converted by oxidative decarboxylation into a two-carbon acetate group, which is linked by a thio-ester bond to coenzyme A (CoA) to form acetyl-CoA. This reaction, in which NAD^+ is converted to NADH, is catalyzed by the pyruvate dehydrogenase enzyme complex. Acetyl-CoA is also formed from the metabolism of fatty acids (FAs) within the mitochondria, in a metabolic process called β-oxidation and so the final subsequent steps of oxidative degradation are common to both fat and carbohydrate. Acetyl-CoA is oxidized to CO_2 in the TCA cycle (also known as the Krebs cycle and the citric acid cycle). The reactions involve combination of acetyl-CoA with oxaloacetate to form citrate, a six-carbon TCA (Figure 3.3). A series of reactions lead to the sequential loss of hydrogen atoms and CO_2, resulting in the regeneration of oxaloacetate.

Molecular oxygen (O_2) does not participate directly in these reactions. The hydrogen atoms are carried by the reduced coenzymes NADH and flavin adenine dinucleotide ($FADH_2$). These coenzymes act as carriers and donate pairs of electrons to the electron transport chain which sits in the inner membrane of the mitochondrion, allowing

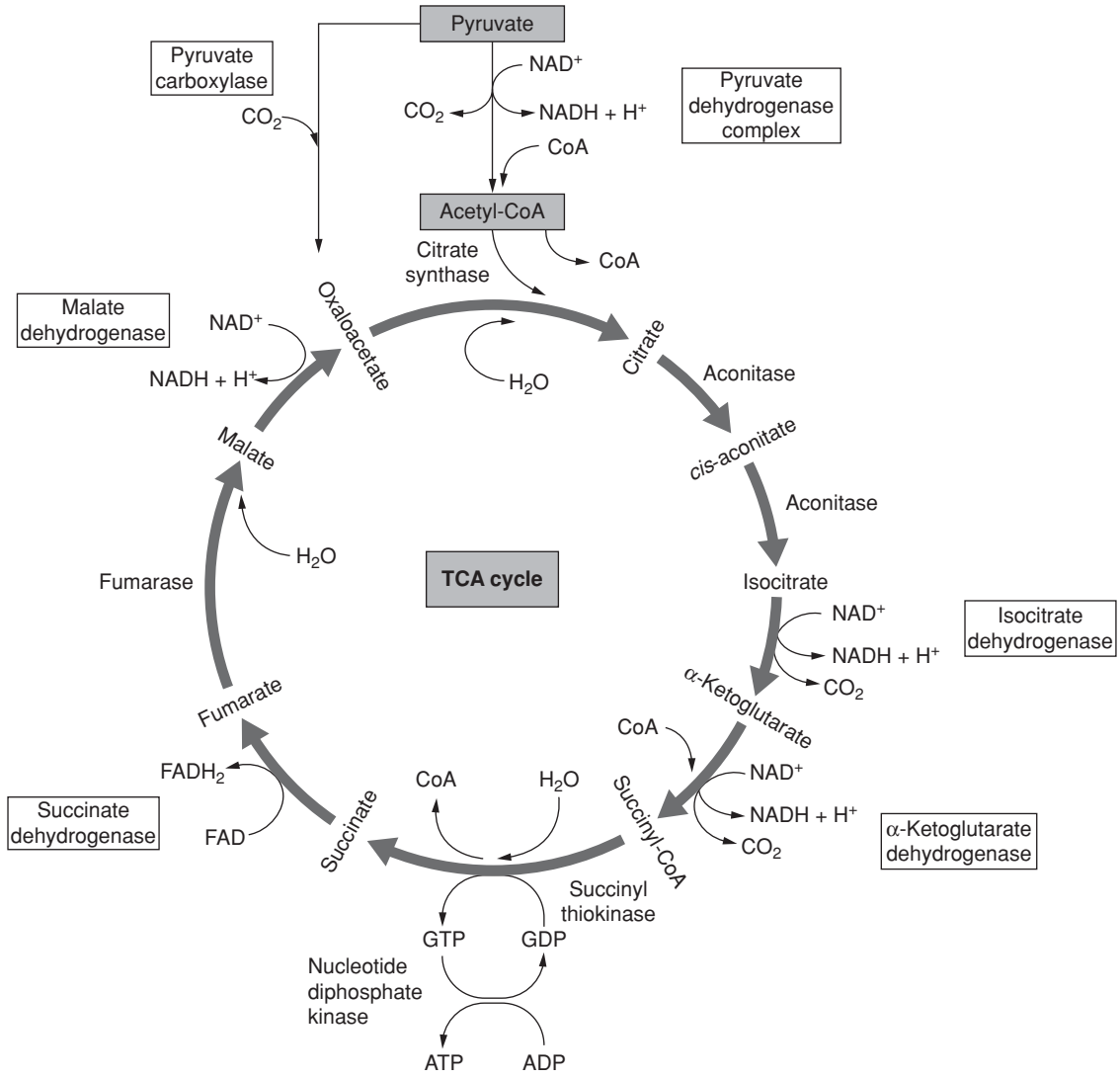

Figure. 3.3 Summary of reactions of the tricarboxylic acid (TCA) cycle showing sites of substrate-level phosphorylation, CO_2 production, and NAD^+ and flavin adenine dinucleotide (FAD) reduction. The two-carbon (2C) acetate units of acetyl-CoA are combined with 4C oxaloacetate to form 6C citrate. The latter undergoes two successive decarboxylation reactions to yield 4C succinate, which in subsequent reactions is converted into 4C oxaloacetate, completing the TCA cycle. GDP, guanosine diphosphate; GTP, guanosine triphosphate.

oxidative phosphorylation with the subsequent regeneration of ATP from ADP (Figure 3.4). The aerobic (O_2 requiring) process of electron transport and oxidative phosphorylation regenerates ATP from ADP and P_i, thus conserving some of the chemical energy contained within the original substrates in the form of high-energy phosphates. As long as the O_2 supply is adequate and substrate is available, NAD^+ and FAD are continuously regenerated and TCA metabolism proceeds.

An interesting concept has developed recently following the finding that dietary nitrate (NO_3^-) supplementation can improve exercise performance via a reduction in the oxygen requirement at a given

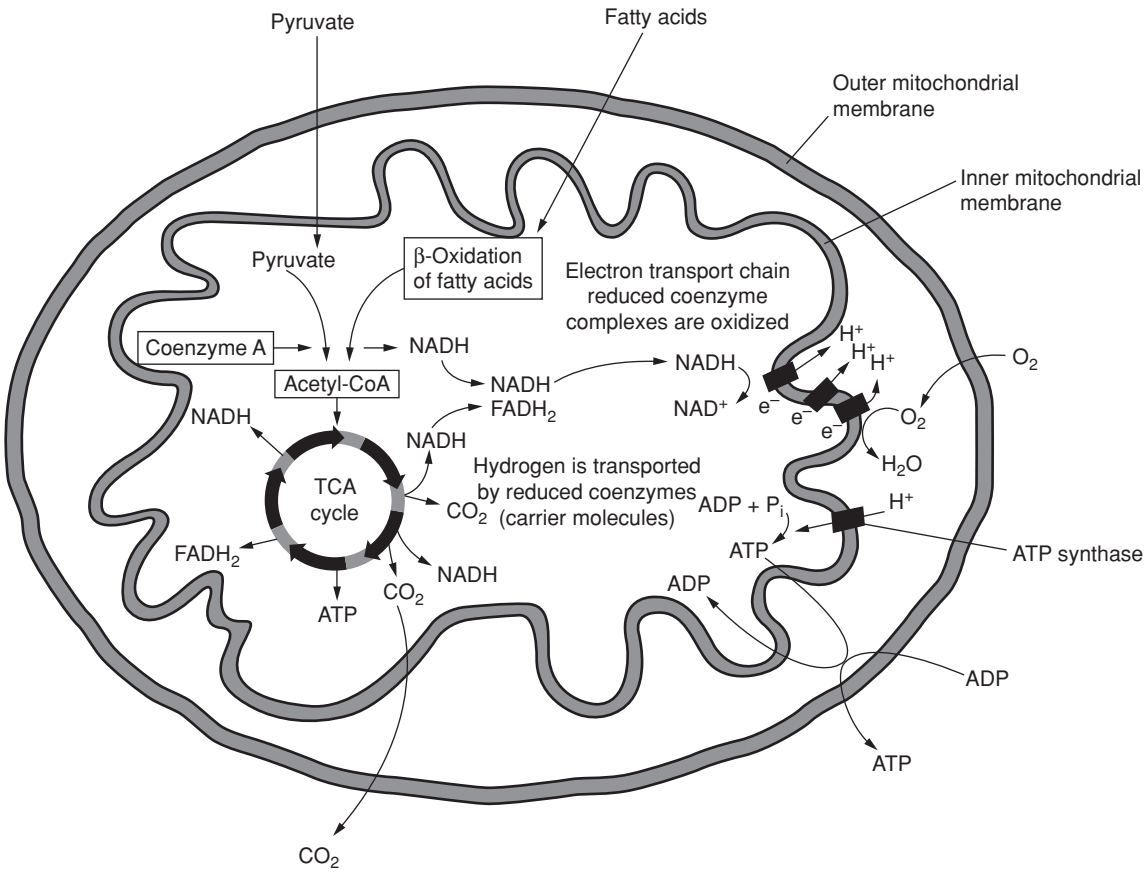

Figure 3.4 Schematic diagram showing the relationship of the tricarboxylic (TCA) cycle to the electron transport chain. The main function of the TCA cycle is to reduce the coenzymes NAD^+ and flavin adenine dinucleotide (FAD) to NADH and $FADH_2$ that act as carriers of H^+ ions and electrons, which are donated to the electron transport chain. Molecular oxygen acts as the terminal electron acceptor and the hydrogen ion gradient generated across the inner mitochondrial membrane is used to drive the synthesis of ATP from ADP and P_i.

submaximal exercise intensity (Lansley et al., 2011a, 2011b; Larsen et al., 2007). The effects of dietary nitrate are thought to be mediated by increased formation of nitrite (NO_2^-) and nitric oxide (NO). The latter may increase mitochondrial oxidative phosphorylation efficiency by increasing the P/O ratio (i.e., more ATP is formed per amount of oxygen consumed). Skeletal muscle mitochondria harvested from muscle biopsies after nitrate supplementation display an increased P/O ratio and the improved mitochondrial P/O ratio correlates with the reduction in oxygen cost during exercise (Larsen et al., 2011). Mechanistically, nitrate reduces the expression of ATP/ADP translocase, a protein involved in proton conductance. Thus, it appears that dietary nitrate has profound effects on basal mitochondrial function and is able to alter the efficiency of aerobic ATP production. Nitrate is found in several plants including beetroot, celery, cress, lettuce, and spinach, and a popular means of ingesting nitrate is drinking beetroot juice.

Fat Oxidation in Aerobic Metabolism

Lipolysis The first step in the breakdown of stored fat is the splitting of triacylglycerol into its

FA and glycerol components in a process called lipolysis. This is catalyzed by a hormone-sensitive lipase. Insulin promotes triacylglycerol synthesis and inhibits lipolysis, whereas catecholamines (adrenaline and noradrenaline), glucagon, growth hormone, and cortisol stimulate lipolysis by activating the hormone-sensitive lipase. Glycerol may diffuse, and FAs are transported out of the adipose cells and into the circulation. Both the rates of lipolysis and adipose tissue blood flow influence the rate of entry of FA and glycerol into the circulation. During prolonged exercise at about 50% of VO_2 max, adipose tissue blood flow increases. During intense exercise, however, sympathetic vasoconstriction causes a fall in adipose tissue blood flow, resulting in the accumulation of FA within the adipose tissue and effectively limiting the entry of FA (and glycerol) into the circulation. Another factor that limits fat mobilization during high-intensity exercise is the accumulation of lactate in the blood. Lactate promotes the reesterification of FA back into triacylglycerol and therefore limits the entry of FA into the bloodstream.

Glycerol in plasma can be taken up by the liver and phosphorylated to glycerol-3-phosphate, which can be used to form triacylglycerol as described earlier, or, alternatively, can be oxidized to dihydroxyacetone phosphate, which can enter the glycolytic pathway or be converted to glucose. FAs are poorly soluble in water, and most of the FAs in plasma are transported loosely bound to albumin, the most abundant protein in plasma. The usual resting plasma concentration of FA is 0.2–0.4 mM. However, during (or shortly after) prolonged exercise, the plasma FA concentration may increase to about 2 mM.

FA Uptake by Muscle Uptake of FA by muscle is directly related to the plasma FA concentration, and hence the mobilization of fat stores is an important step in ensuring an adequate nutrient supply for prolonged muscular work. FA transport across the sarcolemmal membrane into the muscle fiber occurs by a carrier-mediated transport mechanism that becomes saturated at high-plasma unbound FA concentration (approximately equivalent to 1.5 mM total FA concentration). After the FAs enter the muscle cell, they are converted into a CoA derivative by the action of ATP-linked fatty acyl-CoA synthetase (also known as thiokinase), in preparation for β-oxidation, the major pathway for FA breakdown. The latter process occurs in the mitochondria and is the sequential removal of two-carbon units from the FA chain in the form of acetyl-CoA, which can then enter the TCA cycle. Fatty acyl-CoA molecules in the muscle sarcoplasm are transported into the mitochondria through formation of an ester of the FA with carnitine. The enzyme regulating the transport of FA by carnitine is carnitine acyltransferase (CAT) and this transport process may be the main rate-limiting step in the utilization of FAs for energy production in muscle. Carnitine has been promoted as a supplement that can aid weight loss by increasing fat oxidation and that can also enhance endurance exercise performance by promoting fat use and thus sparing the limited glycogen stores. However, until recently it was thought that dietary carnitine supplementation in humans does not affect muscle carnitine content and well-controlled studies showed no effect on exercise performance (Jeukendrup & Gleeson, 2010). Now all this has changed following the recognition that the carnitine content of human muscle can be increased if carnitine is ingested during insulin infusion or when carbohydrate is coingested (Wall et al., 2011). Very recent placebo-controlled studies are now emerging that confirm substantial elevations (~20%) of muscle carnitine content in humans following prolonged daily supplementation with carnitine tartrate and carbohydrate ingested at the same time. It has been shown that this results in muscle glycogen sparing during low-intensity exercise (consistent with an increase in fat utilization) and lower lactate accumulation during high-intensity exercise (Wall et al., 2011). Furthermore, these changes were associated with an improvement in work output in 30-minute exercise performance trials.

At high-exercise intensities (above about 60% of VO_2 max), the rate of fat oxidation cannot provide sufficient ATP for muscle contraction, and, increasingly, ATP is derived from carbohydrate oxidation and anaerobic glycolysis. Energy cannot be derived from fat through anaerobic pathways. After its release into the mitochondrial matrix, the fatty

acyl-CoA is able to enter the β-oxidation pathway. CAT is inhibited by malonyl-CoA, a precursor for FA synthesis. Hence, when the ATP supply is sufficient, surplus acetyl-CoA will be diverted away from the TCA cycle to malonyl-CoA, reducing catabolism of FAs and promoting their formation and subsequent triacylglycerol synthesis.

Amino Acid Oxidation in Aerobic Metabolism In most situations, carbohydrates and fats supply most of the energy required to regenerate ATP to fuel muscular work. Protein catabolism can provide up to 20 different amino acids, up to 8 of which may eventually be oxidized (though only the branched-chain amino acids are quantitatively important in this regard), and this normally contributes less than 5% of the energy provision for muscle contraction during physical activity. During starvation and when glycogen stores become depleted, protein catabolism may become an increasingly important source of energy for muscular work but even then rarely exceeds 10% of the total energy provision. Before amino acids can be oxidized, the amino ($-NH_2$) group must be removed by transamination (transferring it to a keto acid, which results in the formation of a different amino acid) or by oxidative deamination to form free ammonia (NH_3). After the removal of the amino group from an amino acid, the remaining carbon skeleton (the keto acid) is eventually oxidized to CO_2 and H_2O in the TCA cycle.

Fuel Stores in the Body

Carbohydrates are stored in the body as the glucose polymer glycogen. Skeletal muscle contains a significant store of glycogen in the sarcoplasm. The glycogen content of skeletal muscle at rest is approximately 13–18 g/kg w.w. (75–100 mmol glucosyl units/kg w.w.). For cycling and running, a total of about 300 g of glycogen is available in the leg muscles. About 100 g of glycogen are stored in the liver of an adult human in the postabsorptive state, which can be released into the circulation to maintain the blood glucose concentration at about 5 mM (0.9 g/l). Fats are stored as triacylglycerol, mainly in white adipose tissue. Triacylglycerol molecules must be broken down by a lipase enzyme to release FA into the circulation for uptake by working muscle. Skeletal muscle also contains some triacylglycerol that can be used as an energy source during exercise after lipolysis. Some of this intramuscular triacylglycerol may be contained in adipose cells dispersed among the muscle fibers in the tissue, but evidence from light and electron microscopy suggests the existence of triacylglycerol droplets located close to the mitochondria within the fibers themselves.

Early studies of FA turnover during exercise using ^{14}C-labeled FAs showed that during prolonged exercise, plasma-derived FA could account for only about 50% of the total amount of fat oxidized, suggesting that intramuscular triacylglycerol could be providing a significant amount of the FA during prolonged exercise. Measurements of changes in intramuscular triacylglycerol content before and after exercise strongly support this view. Several studies have reported reductions of about 25–35% in triacylglycerol content after 1–2 hours of exercise at 55–70% of VO_2 max (van Loon, 2004).

Human skeletal muscle contains approximately 12 g/kg w.w. of triacylglycerol, and type I fibers contain more triacylglycerol than type II fibers do. Between 12 and 20 MJ of chemical potential energy is estimated to be available for oxidation after intramuscular lipolysis. The lipolysis is probably mediated by an intracellular lipase similar to the hormone-sensitive lipase of adipose tissue; evidence suggests that catecholamines regulate the mobilization of intramuscular triacylglycerol stores.

Fat stores in the body are far larger than carbohydrate stores, and fat is a more efficient form of energy storage, releasing 37 kJ/g (9 kcal/g) compared with 16 kJ/g (4 kcal/g) from carbohydrate. Each gram of carbohydrate stored also retains about 3 g of water, further decreasing the efficiency of carbohydrate as an energy source. But the energy yield per liter of O_2 consumed during fat oxidation is about 8–10% less than for carbohydrate (about 19.5 kJ/l O_2 for fat compared with 20.9 kJ/l O_2 for carbohydrate). The energy cost of running a marathon is about 12,000 kJ (2900 kcal); if this energy could be derived from the oxidation of fat alone, the total amount of fat required would be about 320 g, compared with 750 g of carbohydrate and an additional 2.3 kg of associated water. Aside from the

weight that would have to be carried, this amount of carbohydrate exceeds the total amount normally stored in the liver and muscles combined. The total storage capacity for fat is extremely large, and for most practical purposes, the amount of energy stored in the form of fat far exceeds that required for any exercise task (see Table 3.4).

The main problem associated with the utilization of fat as a fuel for exercise is the rate at which it can be taken up by muscle and oxidized to provide energy. Fat oxidation can only supply ATP at a rate sufficient to maintain exercise at an intensity of about 60% of VO_2 max. To generate ATP to sustain higher exercise intensities, there is an increasing reliance on carbohydrate. Both the oxidative pathway of carbohydrate utilization and the anaerobic pathway of glycolysis can supply ATP at a much faster rate than fat oxidation can (Table 3.3). During most forms of submaximal exercise, a mixture of fat and carbohydrate is oxidized to provide energy for muscular contraction. Obviously, utilizing more fat allows greater sparing of the limited carbohydrate reserves, permitting exercise to be prolonged.

Unlike carbohydrate (as glycogen) and fat (as triacylglycerol), protein is stored only as functionally important molecules (e.g., structural proteins, enzymes, ion channels, receptors, and contractile proteins), and the concentration of free amino acids in most extracellular and intracellular body fluids is quite low (e.g., the total free amino acid concentration in muscle sarcoplasm is about 20 mM). Hence, carbohydrate and fat are the preferred fuels for exercise and the contribution of protein to energy expenditure during even prolonged exercise does not usually exceed a maximum of about 5–10% of total energy expenditure.

Table 3.4 Energy stores in the average 70 kg male athlete who has 10% body fat and a VO_2 max of 70 ml/kg/min

	Mass (kg)	Energy (kJ)	Exercise time (min)
Liver glycogen	0.08	1280	16
Muscle glycogen	0.40	6400	80
Blood glucose	0.01	160	2
Fat	7.0	260,000	3250
Protein	13.0	220,000	2750

The value for blood glucose includes the glucose content of all extracellular fluid. Not all of this, and not more than a very small part (<1%) of the total protein, is available for use during exercise. Also shown are the approximate times these stores would theoretically last for if they were the only source of energy available during exercise at marathon running pace (equivalent to an energy expenditure of about 80 kJ/min, and a VO_2 of 4000 ml/min which for this individual would be a relative exercise intensity of about 80% VO_2 max).

Regulation of Energy Metabolism

Because the intramuscular ATP concentration remains fairly constant during most forms of exercise ATP must be continuously regenerated at the same rate that it is being used. This situation provides a sensitive mechanism for the control of energy metabolism within the cell. This control is exerted through changes in a number of intracellular factors, and further, control is effected through the actions of the sympathetic nervous system and hormones that can also bring about changes in the activities of some enzymes involved in fuel mobilization and utilization.

Intracellular Factors

Within the muscle fiber, several intracellular factors control the activity of key rate-limiting or flux-generating enzymes involved in energy metabolism, which allows rapid (virtually instantaneous) alteration in the rate of ATP resynthesis to occur when such an alteration is needed, such as at the onset of exercise.

The decline in cellular concentration of ATP at the onset of muscle force generation and parallel increases in ADP and AMP concentrations (i.e., a decline in the energy charge) directly stimulate anaerobic and oxidative ATP resynthesis. The relatively low concentration of ATP (and ADP) inside the cell means that any increase in the rate of hydrolysis of ATP (e.g., at the onset of exercise) produces a rapid change in the ratio of ATP to ADP (and also increases the intracellular concentration of AMP). These changes, in turn, activate enzymes that immediately stimulate the breakdown of intramuscular fuel stores to provide energy for ATP resynthesis. In this way, energy metabolism increases rapidly after the start of exercise.

ATP, ADP, and AMP are activators or inhibitors of the enzymatic reactions involved in PCr, carbohydrate, and fat degradation and utilization (see Figure 3.5). For example, CK, the enzyme responsible for the rapid rephosphorylation of ATP at the initiation of muscle force generation, is rapidly activated by an increase in cytoplasmic ADP concentration and is inhibited by an increase in cellular ATP concentration. Similarly, glycogen phosphorylase, the enzyme that catalyzes the conversion of glycogen to glucose-1-phosphate, is activated by increases in AMP and P_i (and Ca^{2+}) concentration and is inhibited by an increase in ATP concentration.

The rate-limiting step in the glycolytic pathway is the conversion of fructose-6-phosphate to fructose-1,6-diphosphate, which is catalyzed

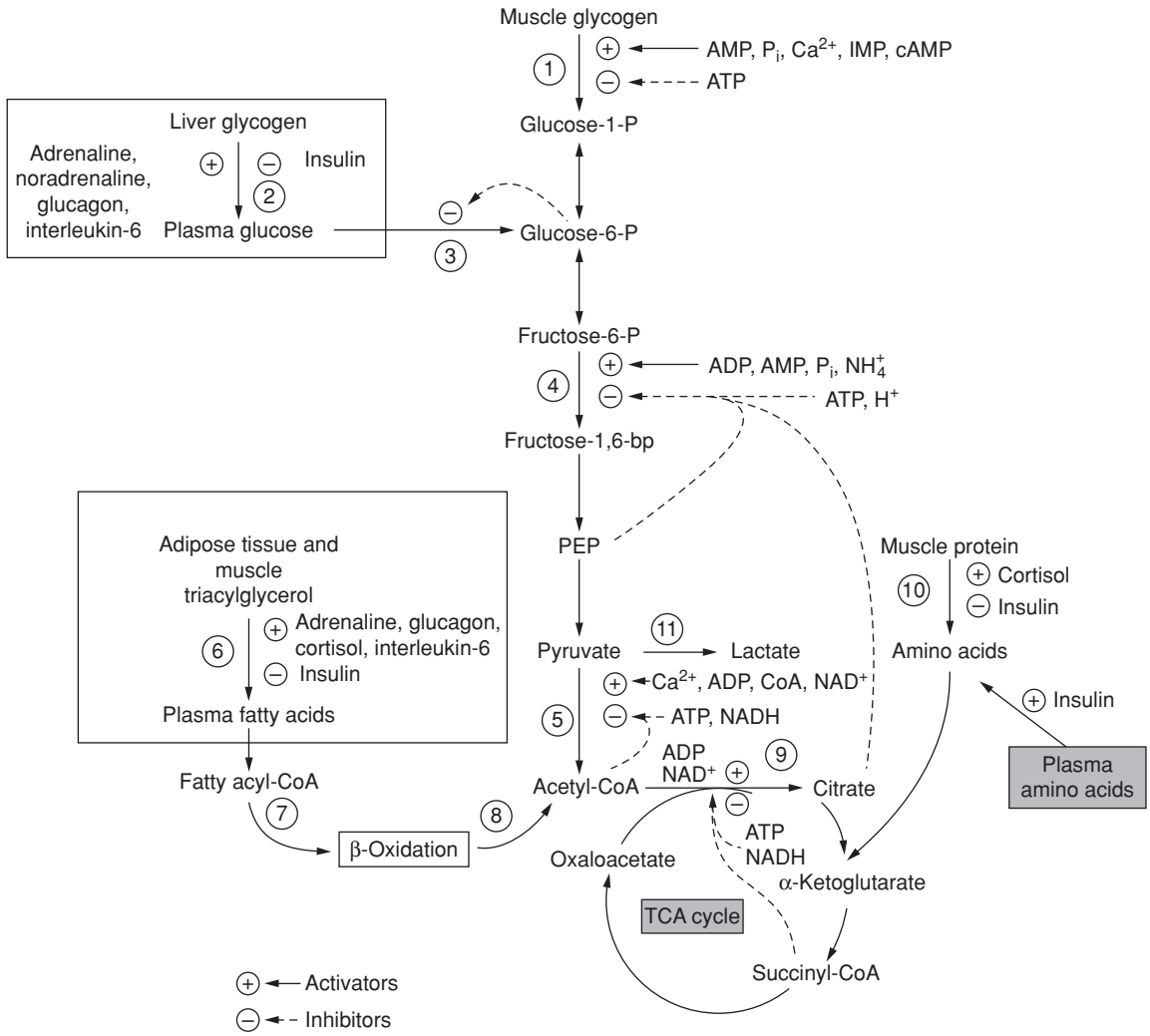

Figure 3.5 Metabolic pathways of importance to energy provision during exercise showing the main sites of regulation and the principal hormonal and allosteric activators and inhibitors. Enzymes: 1, glycogen phosphorylase (muscle); 2, glycogen phosphorylase (liver); 3, hexokinase; 4, phosphofructokinase; 5, pyruvate dehydrogenase; 6, hormone-sensitive lipase; 7, carnitine acyltransferase; 8, 3-hydroxyacyl dehydrogenase; 9, citrate synthase; 10, proteases; 11, lactate dehydrogenase. AMP, adenosine monophosphate; cAMP, cyclic AMP; PEP, phosphoenolpyruvate; P_i, inorganic phosphate; TCA, tricarboxylic acid.

by PFK. The activity of this complex enzyme is affected by many intracellular factors and it plays an important role in controlling flux through the pathway. The PFK reaction regulates the metabolism of both glucose and glycogen. The activity of PFK is stimulated by increased concentrations of ADP, AMP, P_i, ammonia, and fructose-6-phosphate and is inhibited by ATP, H^+, citrate, phosphoglycerate, and phosphoenolpyruvate. Thus, the rate of glycolysis is stimulated when ATP and glycogen breakdown are increased at the onset of exercise. Accumulation of citrate and, thus, inhibition of PFK may occur when the rate of the TCA cycle is high and provides a means whereby the limited stores of carbohydrate are spared when the availability of FAs is high. Inhibition of PFK also causes accumulation of G6P, which inhibits the activity of hexokinase and reduces the entry into the muscle of glucose, which is not needed.

Conversion of pyruvate to acetyl-CoA by the pyruvate dehydrogenase complex is the rate-limiting step in carbohydrate oxidation. It is stimulated by an increased intracellular concentration of Ca^{2+} and decreased ratios of ATP/ADP, acetyl-CoA/free CoA, and NADH/NAD^+ and thus offers another site of regulation of the relative rates of fat and carbohydrate catabolism. If the rate of formation of acetyl-CoA from the β-oxidation of FAs is high, as after 1–2 hours of submaximal exercise, then this activity can reduce the amount of acetyl-CoA derived from pyruvate and cause accumulation of phosphoenolpyruvate and inhibition of PFK, thus slowing the rate of glycolysis and glycogenolysis. A key regulatory point in the TCA cycle is the reaction catalyzed by citrate synthase. The activity of this enzyme is inhibited by ATP, NADH, succinyl-CoA, and fatty acyl-CoA; the activity of the enzyme is also affected by citrate availability. Hence, when cellular energy levels are high, flux through the TCA cycle is relatively low but can be greatly increased when ATP and NADH utilization is increased, such as during exercise.

Hormones and Cytokines

Many hormones influence energy metabolism in the body but during exercise the interaction among insulin, glucagon, and the catecholamines (adrenaline and noradrenaline) is mostly responsible for fuel substrate availability and utilization; cortisol and growth hormone also have some significant effects. Recently, it has been recognized that a cytokine called interleukin-6 (IL-6), which is released from contracting skeletal muscle fibers during exercise, also plays a role in the regulation of fuel mobilization and metabolism (Pedersen & Febbraio, 2008).

Insulin is secreted by the β-cells of the islets of Langerhans in the pancreas. Its basic biological effects are to inhibit lipolysis and increase the uptake of glucose from the blood by the tissues, especially skeletal muscle, liver, and adipose tissue; the cellular uptake of amino acids is also stimulated by insulin. These effects reduce the plasma glucose concentration, inhibit the release of glucose from the liver, stimulate the synthesis of glycogen (in the liver and muscle), promote the synthesis of lipid and inhibit FFA release (in adipose tissue), increase muscle amino acid uptake, and inhibit protein breakdown. The primary stimulus for increased insulin secretion is a rise in the blood glucose concentration (e.g., following a meal). Exercise usually results in a fall in insulin secretion.

Glucagon is secreted by the α-cells of the pancreatic islets and basically exerts effects that are opposite to those of insulin. It raises the blood glucose level by increasing the rate of glycogen breakdown (glycogenolysis) in the liver. It also promotes the formation of glucose from noncarbohydrate precursors (gluconeogenesis) in the liver. The primary stimulus for increased secretion of glucagon is a fall in the concentration of glucose in blood. During most types of exercise, the blood glucose concentration does not fall, but during prolonged exercise, when liver glycogen stores become depleted, a drop in the blood glucose concentration (hypoglycemia) may occur.

The catecholamines, adrenaline and noradrenaline, are released from the adrenal medulla. Noradrenaline is also released from sympathetic nerve endings and leakage from such synapses appears to be the main source of the noradrenaline found in blood plasma. The catecholamines have many systemic effects throughout the body

including stimulation of the heart rate and contractility and alteration of blood vessel diameters. They also influence substrate availability, with the effects of adrenaline being the more important of the two. Adrenaline, like glucagon, promotes glycogenolysis in both the liver and muscle. Adrenaline also promotes lipolysis in adipose tissue, increasing the availability of plasma FFA, and inhibits insulin secretion. The primary stimulus for catecholamine secretion is the activation of the sympathetic nervous system by stressors such as exercise, hypotension, and hypoglycemia. Substantial increases in the plasma catecholamine concentration can occur within seconds of the onset of high-intensity exercise. However, the relative exercise intensity has to be above about 50% VO_2 max in order to significantly elevate the plasma catecholamine concentration.

Growth hormone, secreted from the anterior pituitary gland, also stimulates mobilization of FFA from adipose tissue and increases in plasma growth hormone concentration are related to the intensity of exercise performed. During prolonged strenuous exercise, cortisol secretion from the adrenal cortex is increased. Cortisol is a steroid hormone that increases the effectiveness of the actions of catecholamines in some tissues (e.g., its actions further promote lipolysis in adipose tissue). However, its main metabolic effects are to promote protein degradation and amino acid release from muscle and to stimulate gluconeogenesis in the liver. The primary stimulus to cortisol secretion is stress-induced release of adrenocorticotropic hormone from the anterior pituitary gland, though cortisol secretion is also increased by increased circulating IL-6, a cytokine (a peptide chemical messenger) produced by several different cells and tissues. IL-6 is most well known for its actions in the regulation of immune function. It is secreted from activated macrophages but contracting skeletal muscle fibers also produce and release IL-6 into the circulation and, indeed, muscle secretion of IL-6 has been shown to be almost entirely responsible for the up to 100-fold rise in plasma IL-6 concentration during prolonged exercise such as marathon running (Fischer, 2006). The release of IL-6 is primarily regulated by an altered intramuscular milieu in response to exercise: changes in Ca^{2+} homeostasis, impaired glucose availability (glycogen depletion), and increased formation of reactive oxygen species are all associated with exercise and capable of activating transcription factors known to regulate IL-6 synthesis. Acute IL-6 administration to humans increases lipolysis, fat oxidation, liver glycogenolysis, and insulin-mediated glucose disposal (Pedersen & Febbraio, 2008). IL-6 also has anti-inflammatory effects (Gleeson et al., 2011) and may exert some of its biological effects through stimulation of cortisol and IL-10 secretion (Steensberg et al., 2003) and inhibition of the proinflammatory cytokine tumor necrosis factor alpha (Starkie et al., 2003).

Metabolic Responses to Exercise

The most important factor influencing the metabolic response to exercise is exercise intensity. The physical fitness of the subject also modifies the metabolic response to exercise, and other factors—including exercise duration, substrate availability, nutritional status, diet, feeding during exercise, mode of exercise, previous exercise, drugs, environmental temperature, and altitude—are also important.

High-Intensity Exercise

During high-intensity exercise, the inability of aerobic means of ATP resynthesis to supply ATP at a sufficiently high rate stimulates rapid anaerobic energy production from both PCr and glycogen breakdown. PCr breakdown begins at the onset of contraction to buffer the rapid accumulation of ADP from ATP hydrolysis but the rate of PCr hydrolysis declines only after a few seconds of maximal force generation. If high-intensity exercise is to continue beyond the first few seconds, a marked increase in the contribution from glycolysis to ATP resynthesis is necessary. Anaerobic glycolysis involves several more steps than PCr hydrolysis, although compared with oxidative phosphorylation, it is still extremely rapid. Anaerobic glycolysis starts at the onset of contraction, but unlike PCr hydrolysis, it does not reach a maximal rate until after 5–10 seconds of exercise, and it can be maintained at this level for about 10–20 seconds during maximal muscle force generation. Exercise at an intensity equivalent to 95–100% of VO_2 max can be sustained for about

5 minutes before fatigue ensues. Under these conditions, carbohydrate oxidation can make a significant contribution to ATP production, but its relative importance is often underestimated.

Fatigue is often defined as the inability to maintain a given or expected force or power output and is an inevitable feature of maximal exercise. Typically, the loss of power output or force production is likely to be in the region of 40–60% of the maximum observed during 30 seconds of all-out exercise. Many factors contribute to fatigue but during maximal short-duration exercise, it will be caused primarily by a gradual decline in anaerobic ATP production or an increase in ADP accumulation caused by a depletion of PCr and a fall in the rate of glycolysis.

In high-intensity exercise lasting 1–5 minutes, H^+ accumulation may contribute to the fatigue process (Green, 1991). Reduced muscle pH may cause some inhibition of PFK and phosphorylase, reducing the rate of ATP resynthesis from glycolysis. This development is unlikely to be important in exercising muscle, however, because the *in vitro* inhibition of PFK by a reduced pH is reversed in the presence of other activators such as AMP. Furthermore, it appears that the rate of glycogenolysis/glycolysis is not affected by muscle acidity in exercising humans (Bangsbo et al., 1996). However, although likely to be related to the fatigue process, it is unlikely that H^+ accumulation is wholly responsible for muscle fatigue (Westerblad et al., 2002). For example, studies involving human volunteers have demonstrated that muscle force generation after fatiguing exercise can recover rapidly, despite also having a very low muscle pH value. The consensus appears to be that the maintenance of force production during high-intensity exercise is pH dependent, but the initial reduction in force generation is more related to reduced PCr availability.

One of the consequences of rapid PCr hydrolysis during high-intensity exercise is the accumulation of P_i, which has been identified as an important potential cause of muscle fatigue (Westerblad et al., 2002). P_i may act directly on the myofibrils and decrease crossbridge force production and myofibrillar Ca^{2+} sensitivity. By acting on SR Ca^{2+} handling, increased P_i may also increase tetanic sarcoplasmic-free Ca^{2+} concentration in early fatigue by stimulating the SR Ca^{2+} release channels, inhibiting the ATP-dependent SR Ca^{2+} uptake, and reducing tetanic sarcoplasmic-free Ca^{2+} concentration in late fatigue by entering the SR, precipitating with Ca^{2+}, and thereby decreasing the Ca^{2+} available for release. Ca^{2+} release by the SR as a consequence of muscle depolarization is essential for the activation of muscle contraction coupling. During fatiguing contractions, Ca^{2+} transport slows and Ca^{2+} transients become progressively smaller due to a reduction in Ca^{2+} reuptake by the SR or increased Ca^{2+} binding. Strong evidence that a disruption of Ca^{2+} handling is responsible for fatigue comes from studies showing that the stimulation of SR Ca^{2+} release caused by the administration of caffeine to isolated muscle can improve muscle force production, even in the presence of a low muscle pH (Green, 1991). Alternatively, fatigue during high-intensity exercise may be associated with an excitation-coupling failure and possibly a reduced nervous drive caused by reflex inhibition at the spinal level. In the latter hypothesis, accumulation of interstitial potassium in muscle may play a major role (Bangsbo, 1997; Sjogaard, 1991).

When repeated bouts of maximal exercise are performed, the rates of muscle PCr hydrolysis and lactate accumulation decline. In the case of PCr, this response is thought to occur because of incomplete PCr resynthesis during recovery between successive exercise bouts. The mechanisms responsible for the fall in the rate of lactate accumulation are unclear but seem likely to be related to a decreased rate of glycogenolysis and greater pyruvate dehydrogenase activity with successive sprints.

Prolonged Exercise

The term *prolonged exercise* is usually used to describe exercise intensities that can be sustained for about 30–180 minutes. Because the rate of ATP demand is relatively low compared with high-intensity exercise, PCr, carbohydrate, and fat can all contribute to energy production. The rates of PCr degradation and lactate production during the first minutes of prolonged exercise are closely related to the intensity of exercise performed, and energy production during this period would likely be compromised without this contribution from anaerobic metabolism. After a steady state has been reached, however,

carbohydrate and fat oxidation become the principal means of resynthesizing ATP. Muscle glycogen is the principal fuel during the first 30 minutes of exercise at 60–80% of VO_2 max and the rate of muscle glycogen utilization depends on exercise intensity.

During the early stages of exercise, fat oxidation is limited by the delay in the mobilization of FAs from adipose tissue. At rest after an overnight fast, the plasma FA concentration is about 0.3–0.4 mM. This concentration is commonly observed to fall during the first hour of moderate-intensity exercise, followed by a progressive increase as lipolysis is stimulated by the actions of catecholamines, glucagon, and cortisol (Figure 3.6). During very prolonged exercise, the plasma FA concentration can reach 1.5–2.0 mM, and muscle uptake of blood-borne FA is proportional to the plasma FA concentration.

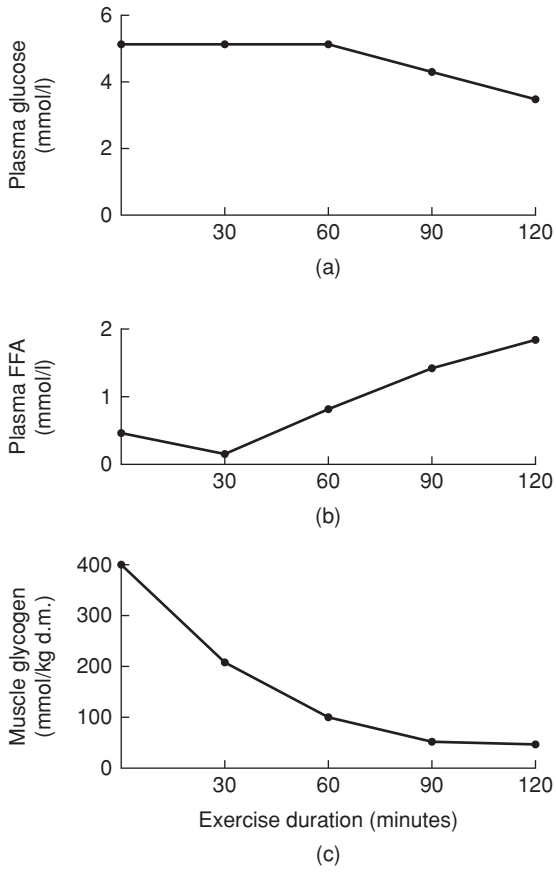

Figure 3.6 Changes in the concentrations of (a) plasma glucose, (b) plasma free fatty acids (FFA), and (c) muscle glycogen during continuous exercise at an intensity equivalent to about 70% VO_2 max.

The glycerol released from adipose tissue cannot be used directly by muscle, which lacks the enzyme glycerol kinase. But glycerol (together with alanine and lactate) is taken up by the liver and used as a gluconeogenic precursor to help maintain liver glucose output as liver glycogen levels decline. The utilization of blood glucose is greater at higher work rates and increases during prolonged submaximal exercise. At an exercise intensity of 70% of VO_2 max, the contribution of blood glucose to ATP resynthesis peaks after about 90 minutes (Figure 3.7). The decline in blood glucose utilization after 90 minutes is attributable to the increasing availability of plasma FA as fuel (which appears to inhibit muscle glucose uptake directly), the depletion of liver glycogen stores, and a falling blood glucose concentration.

At marathon running pace for the faster runners, muscle carbohydrate stores alone can fuel about 80 minutes of exercise before depletion. The simultaneous utilization of body fat and hepatic carbohydrate stores, however, enables ATP production to be maintained and exercise to continue. At marathon running pace, the muscle and hepatic carbohydrate stores will ultimately become depleted. At

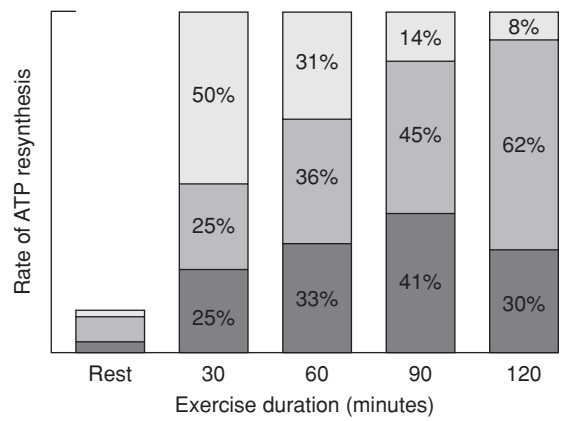

Figure 3.7 Changes in the relative contributions of the major fuel sources to ATP resynthesis during prolonged submaximal exercise at an intensity equivalent to about 70% VO_2 max (approximately 10 times the resting metabolic rate): blood glucose; plasma free fatty acids; muscle glycogen; and triacylglycerol. Light shade of grey: Muscle glycogen; medium shade of grey: Fat; dark shade of grey: Plasma glucose.

this point, ATP production becomes compromised because of the inability of fat oxidation to increase sufficiently to offset this deficit. The rate of ATP resynthesis from fat oxidation alone cannot meet the ATP requirement for exercise intensities higher than about 50–60% of VO_2 max. The factor that limits the maximum rate of fat oxidation during exercise (i.e., why it cannot increase to compensate for carbohydrate depletion) may be the rate of uptake of FA into muscle from blood or the transport of FA into the mitochondria, rather than in the rate of β-oxidation of FA in the mitochondria. Recent studies that have elevated muscle carnitine concentration via combined carnitine and carbohydrate ingestion have shown that this results in muscle glycogen sparing during low-intensity exercise (consistent with an increase in fat utilization) and lower lactate accumulation during high-intensity exercise along with improved exercise performance (Wall et al., 2011).

In sedentary people, the glycogen store in muscle is fairly resistant to change. The combination of exercise and dietary manipulation, however, can have a dramatic effect on muscle glycogen storage. A clear positive relationship exists between preexercise muscle glycogen content and subsequent endurance performance (Bergstrom et al., 1967). Furthermore, the ingestion of carbohydrate during prolonged exercise decreases muscle glycogen utilization and fat mobilization and oxidation, and it increases the rate of carbohydrate oxidation and endurance capacity (Coyle, 2004). Therefore, the contribution of orally ingested carbohydrate to total ATP production under these conditions must be greater than that normally derived from fat oxidation. In part, this is because when exercise is performed with high initial muscle and liver glycogen levels, or when carbohydrate is ingested during exercise, the hormonal (adrenaline, noradrenaline, glucagon, cortisol) and cytokine (IL-6) response to exercise is attenuated compared with when exercise is performed in a carbohydrate-depleted state (Gleeson, 2005). These hormones and cytokines are involved in the stimulation of lipolysis, so fat mobilization is delayed and the rate of fat oxidation is less when carbohydrate is consumed during exercise.

The precise biochemical mechanism by which muscle glycogen depletion causes fatigue is currently unresolved. However, the inability of muscle to maintain the rate of ATP synthesis in the glycogen-depleted state possibly results in ADP and P_i accumulation and consequently fatigue. Of course, a substantial fall in intramuscular ATP concentration is unlikely in this form of exercise, because very low ATP concentrations cause rigor and irreversible damage to muscle fibers. Hence, some factor other than muscle glycogen depletion, probably linked to low muscle glycogen concentrations, must act to constrain the activity of glycogen-depleted muscle before rigor can develop.

Starvation rapidly depletes the liver of carbohydrate, which falls by about 50% during overnight fasting. The rate of hepatic glucose release in resting, postabsorptive individuals is sufficient to match the carbohydrate demands of only the central nervous system. Approximately 70% of this release is derived from liver carbohydrate stores, and the remainder is derived from liver gluconeogenesis. During exercise, the rate of hepatic glucose release is related to exercise intensity. Liver carbohydrate stores contribute 90% of this release, ultimately resulting in liver glycogen depletion.

Prolonged exercise, particularly after a period of fasting or a diet low in carbohydrate, can result in hypoglycemia, which may be a direct cause of fatigue. Alterations in the liver rather than muscle glycogen concentrations could possibly be the more important determinant of the marked difference in exercise capacity induced by high-carbohydrate and low-carbohydrate diets. Hypoglycemia is detected by the brain and causes symptoms of tiredness and dizziness. This central fatigue could then reduce the degree of skeletal muscle recruitment by the motor cortex, causing a fall in muscle force generation. Furthermore, afferent chemoreceptor information from both the hepatic portal system (monitoring hepatic portal blood glucose concentration) and the skeletal muscle (monitoring muscle glycogenolysis) possibly feeds back to the motor cortex. These signals increase as hypoglycemia develops, and the availability of muscle glycogen declines, inducing more central fatigue and causing athletes to reduce their exercise intensity.

Thus, carbohydrate ingestion during exercise can also delay fatigue development by slowing the rate of liver glycogen depletion and helping to maintain the blood glucose concentration. Central fatigue is certainly a possibility during prolonged exercise, and undoubtedly the development of hypoglycemia contributes to this. Central fatigue also develops as core temperature increases toward 40 °C when prolonged exercise is performed in hot ambient conditions. Fatigue is a protective mechanism designed to prevent irreversible muscle damage and—even more important—to prevent neural damage by hypoglycemia and/or hyperthermia. Several drugs including amphetamines and caffeine have been shown to be ergogenic via actions on the brain (Jones, 2008) that reduce sensations of fatigue, increase alertness, and improve cognitive function—important for sports in which motor skills, concentration, and decision making play a role in success. However, these drugs may override the protective fatigue mechanisms and so in some exercise situations can be a health hazard.

Metabolic Adaptations to Exercise Training

Endurance Training Adaptation and the Influence of Nutrition

Muscle adaptations to aerobic endurance training include increases in capillary density and mitochondrial size and number. The activity of the TCA cycle and of other oxidative enzymes increases, and a concomitant increase occurs in the capacity to oxidize both fat and carbohydrate (Greenhaff & Hultman, 1999). Training adaptations in muscle affect substrate utilization. Endurance training also increases the relative cross-sectional area of type I fibers, increases intramuscular content of triacylglycerol, and increases the capacity to use fat as an energy source during submaximal exercise. Trained subjects also demonstrate increased reliance on intramuscular triacylglycerol as an energy source. These effects and other physiological effects of training, including increased maximum cardiac output and VO_2 max, improved oxygen delivery to working muscle, and attenuated hormonal responses to exercise, decrease the rate of muscle glycogen and blood glucose utilization and decrease the rate of lactate accumulation during submaximal exercise (Saltin, 1985). These adaptations contribute to marked improvement in endurance capacity after training.

Alterations in substrate use with endurance training could be caused, at least in part, by less disturbance to ATP homeostasis during exercise. With increased mitochondrial oxidative capacity after training, smaller decreases in ATP and PCr and smaller increases in ADP and P_i are needed to balance the rate of ATP synthesis with the rate of ATP utilization. In other words, with more mitochondria, the amount of oxygen as well as the ADP and P_i required per mitochondrion will be less after training than before training. The smaller increase in ADP concentration would result in the formation of less AMP by the myokinase reaction, and also less IMP and ammonia would be formed by AMP deamination. Smaller increases in the concentrations of ADP, AMP, P_i, and ammonia could account for the slower rate of glycolysis and glycogenolysis in trained muscle compared with untrained muscle.

In recent years, interest has developed in the influence of nutrition on endurance training adaptations (Hawley et al., 2011). It is now recognized that low levels of muscle glycogen may enhance the mRNA content of some genes involved in exercise metabolism. Several studies have shown that the genes for selected metabolic enzymes including citrate synthase, β-hydroxyacyl-CoA-dehydrogenase, and pyruvate dehydrogenase kinase are upregulated to a greater extent in response to exercise when exercise is performed with low preexercise glycogen content (Burke, 2010; Hawley et al., 2011). The role of muscle glycogen could be explained by the fact that some signaling proteins such as AMP-activated protein kinase (AMPK) possess glycogen-binding domains, and when glycogen is low, these proteins are more active toward their specific targets. Indeed, the activity of both AMPK and p38 mitogen-activated protein kinase (p38 MAPK) is elevated when exercise is performed with low muscle glycogen (Burke, 2010; Matsakas & Patel, 2009). This may be beneficial to individuals undertaking endurance exercise training as AMPK and

p38 MAPK are believed to play a critical role in regulating mitochondrial biogenesis and endurance training adaptations. However, it is not yet known whether these exaggerated responses in a glycogen-depleted state translate into greater protein contents or higher functional activities. It is likely that signals responsive to both increased fat availability and decreased CHO availability would work in concert to determine the exact responses in gene expression in skeletal muscle. The mechanisms by which the adaptations to a high-fat/low-CHO diet are mediated appear to be related to FFA activation of the family of peroxisome proliferator-activated receptors (PPARs) and/or an insulin effect consequent to the decreased CHO availability.

The level of antioxidants in the diet may also have some influence on endurance training adaptations. Reactive oxygen and nitrogen species (RONS) are involved in the modulation of cell signaling pathways and the control of several (redox-sensitive) transcription factors. Although high levels of RONS may interfere with muscle function, moderate levels of RONS are actually essential in the development of optimal force production in muscle (Powers & Jackson, 2008; Reid, 2008). These findings raise a number of important questions concerning the possible role of free radical species as signals for wider adaptive responses in these and other tissues and question the approach to protection of tissues which involves advocating the use of widespread supplementation with antioxidant nutrients. It is entirely feasible that those adaptations to stress mediated by free radicals play an important role in maintaining cell viability in tissues routinely subjected to repeated stresses (e.g., muscle following exercise) and that increased consumption of some antioxidant nutrients might interfere with these necessary adaptive processes. In one recent study, 14 men were trained for 8 weeks and 5 of the men were supplemented daily with an oral dose of 1 g vitamin C (Gomez-Cabrera et al., 2008). The administration of vitamin C significantly impaired endurance capacity. The adverse effects of vitamin C may result from its capacity to reduce the exercise-induced expression of key transcription factors involved in mitochondrial biogenesis: PPAR coactivator 1, nuclear respiratory factor 1, and mitochondrial transcription factor A. Vitamin C also prevented the exercise-induced expression of cytochrome *c* (a marker of mitochondrial content) and of the antioxidant enzymes, superoxide dismutase and glutathione peroxidase. Thus, it seems that supplementation with large doses of antioxidants may interfere with the function of RONS and may reduce cellular adaptations to exercise training, though some studies have not supported this concept (Yfanti et al., 2010). A lot more research needs to be done to determine the role of RONS in training, but it seems that they may not always be damaging and the role of RONS in modulating signaling pathways may be important in adaptive processes. In recent *in vitro* and animal studies, several polyphenolic phytochemicals (e.g., epicatechins, quercetin, and resveratrol) have been found to exert an exercise mimetic effect, promoting an enhancement of mitochondrial biogenesis (Hawley et al., 2011), and further research may determine if these nonnutritive plant compounds can influence exercise-induced adaptations in humans.

Strength Training Adaptation and the Influence of Nutrition

Training for strength, power, or speed has little if any effect on aerobic capacity. Heavy resistance training or sprinting brings about specific changes in the immediate (ATP and PCr) and short-term (glycolytic) energy delivery systems, increases in muscle-buffering capacity (mediated mostly by an increase in sarcolemmal MCT expression), and improvements in strength or sprint performance. Several months of heavy resistance training causes hypertrophy of the muscle fibers, thus increasing total muscle mass and the possible maximum power output.

The timing and composition of postexercise nutrition influences protein turnover and hence the response to a hypertrophic stimulus. If an individual is in a fasted state before a resistance training bout and remains fasted afterward, both protein synthesis and degradation are elevated in the posttraining period, but breakdown exceeds synthesis resulting in a net loss of muscle tissue protein (Phillips, 2011; Rennie, 2005). The ingestion of protein or amino acids immediately after exercise can prevent this

loss by both promoting muscle protein synthesis and reducing breakdown such that a net gain of tissue protein occurs (see Chapter 11). The supply of essential amino acids may be the limiting factor and recent studies indicate that ingestion of at least 25 g of protein (containing about 10 g of essential amino acids) is needed to achieve optimal muscle tissue protein gain after a resistance exercise session (Phillips, 2011). The stimulation of protein synthesis after hard exercise in combination with amino acid ingestion may last for up to 24 hours or even longer and offers athletes an opportunity to promote more effective adaptations to their training programs. Recent studies have established that regular ingestion of protein in close proximity to resistance training sessions results in greater muscle hypertrophy and strength gains than consumption of similar amounts of protein at other times (Burd et al., 2009; Phillips, 2011). Coingestion of carbohydrate to maximize the insulin response may not be needed if the intake of protein postexercise is adequate but is a sensible option for the athlete who wants to maximize training adaptation and restore muscle glycogen.

Research has shown that the essential amino acid leucine plays a key role in the stimulation of muscle protein synthesis and appears to be a key activator in switching on muscle protein synthesis following resistance exercise (Phillips, 2011). Thus, high-quality, rapidly digested, leucine-rich proteins such as whey protein would appear to be ideal products for stimulating muscle protein gain and promotion of hypertrophy.

References

Artioli, G.G., Gualano, B., Smith, A., et al. (2010) Role of beta-alanine supplementation on muscle carnosine and exercise performance. *Medicine & Science in Sports & Exercise* 42(6), 1162–1173.

Bangsbo, J. (1997) Physiology of muscle fatigue during intense exercise. In *The Clinical Pharmacology of Sport and Exercise* (eds. T. Reilly & M. Orme), pp. 123–133. Elsevier, Amsterdam.

Bangsbo, J. Madsen, K., Kiens, B., et al. (1996) Effect of muscle acidity on muscle metabolism and fatigue during intense exercise in man. *Journal of Physiology* 495, 587–596.

Bergstrom, J., Hermansen, L., Hultman, E., et al. (1967) Diet, muscle glycogen and physical performance. *Acta Physiologica Scandinavica* 71, 140–150.

Burd, N.A., Tang, J.E., Moore, D.R., et al. (2009) Exercise training and protein metabolism: influences of contraction, protein intake, and sex-based differences. *Journal of Applied Physiology* 106, 1692–1701.

Burke, L.M. (2010) Fueling strategies to optimize performance: training high or training low? *Scandinavian Journal of Medicine and Science in Sports* 20 (Suppl. 2), 48–58.

Coyle, E.F. (2004) Fluid and fuel intake during exercise. *Journal of Sports Sciences* 22(1), 39–55.

Derave, W., Everaert, I., Beeckman, S., et al. (2010) Muscle carnosine metabolism and beta-alanine supplementation in relation to exercise and training. *Sports Medicine* 40(3), 247–263.

Fischer, C.P. (2006) Interleukin-6 in acute exercise and training: what is the biological relevance? *Exercise Immunology Review* 12, 6–33.

Gleeson, M. (ed.) (2005) *Immune Function in Sport and Exercise*. Elsevier, Edinburgh.

Gleeson, M., Bishop, N.C., Stensel. D.J., et al. (2011) The anti-inflammatory effects of exercise: mechanisms and implications for the prevention and treatment of disease. *Nature Reviews Immunology* 11, 607–615.

Gomez-Cabrera, M.C., Domenech, E., Romagnoli, M., et al. (2008) Oral administration of vitamin C decreases muscle mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. *American Journal of Clinical Nutrition* 87, 142–149.

Green, H.J. (1991) How important is endogenous muscle glycogen to fatigue in prolonged exercise? *Canadian Journal of Physiology and Pharmacology* 69, 290–297.

Greenhaff, P.L. (2000) Creatine. In *Nutrition in Sport* (ed. R.J. Maughan), pp. 379–392. Blackwell, Oxford.

Greenhaff, P.L. & Hultman, E. (1999) The biochemical basis of exercise. In *Basic and Applied Sciences for Sports Medicine* (ed. R.J. Maughan), pp. 69–89. Butterworth-Heinemann, Oxford.

Hargreaves, M. & Spriet, L. (2006) *Exercise Metabolism*, 2nd edn. Human Kinetics, Champaign, IL.

Hawley, J.A., Burke, L.M., Phillips, S.M., et al. (2011) Nutritional modulation of training-induced skeletal muscle adaptations. *Journal of Applied Physiology* 110, 834–845.

Hespel, P., Maughan, R.J., & Greenhaff, P.L. (2006) Dietary supplements for football. *Journal of Sports Sciences* 24, 749–761.

Jeukendrup, A.E. & Gleeson, M. (2010) *Sports Nutrition: An Introduction to Energy Production and Performance*, 2nd edn. Human Kinetics, Champaign, IL.

Jones, G. (2008) Caffeine and other sympathomimetic stimulants: modes of action and effects on sports performance. *Essays in Biochemistry* 44, 109–123.

Komi, P.V. & Karlsson, J. (1978) Skeletal muscle fibre types, enzyme activities and physical performance in young males and females. *Acta Physiologica Scandinavica* 103, 210–218.

Lansley, K.E., Winyard, P.G., Bailey, S.J., et al. (2011a) Acute dietary nitrate supplementation improves cycling time trial performance. *Medicine & Science in Sports & Exercise* 43(6), 1125–1131.

Lansley, K.E., Winyard, P.G., Fulford, J., et al. (2011b) Dietary nitrate supplementation reduces the O_2 cost of walking and running: a placebo-controlled study. *Journal of Applied Physiology* 110(3), 591–600.

Larsen, F.J., Weitzberg, E., Lundberg, J.O., et al. (2007) Effects of dietary nitrate on oxygen cost during exercise. *Acta Physiologica (Oxford, England)* 191(1), 59–66.

Larsen, F.J., Schiffer, T.A., Borniquel, S., et al. (2011) Dietary inorganic nitrate improves mitochondrial efficiency in humans. *Cell Metabolism* 13(2),149–159.

Matsakas, A. & Patel, K. (2009) Intracellular signalling pathways regulating the adaptation of skeletal muscle to exercise and nutritional changes. *Histology and Histopathology* 24, 209–222.

Maughan, R.J. & Gleeson, M. (2010) *The Biochemical Basis of Sports Performance,* 2nd edn. Oxford University Press, Oxford.

Pedersen, B.K. & Febbraio, M.A. (2008) Muscle as an endocrine organ: focus on muscle-derived interleukin-6. *Physiological Reviews* 88, 1379–1406.

Phillips, S.M. (2011) The science of muscle hypertrophy: making dietary protein count *Proceedings of the Nutrition Society* 70, 100–103.

Pilegaard, H., Domino, K., Noland, T., et al. (1999) Effect of high-intensity exercise training on lactate/H+ transport capacity in human skeletal muscle. *American Journal of Physiology* 276(2 Pt 1), E255–E261.

Powers, S.K. & Jackson, M.J. (2008) Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. *Physiological Reviews* 88, 1243–1276.

Reid, M.B. (2008) Free radicals and muscle fatigue: of ROS, canaries, and the IOC. *Free Radical Biology and Medicine* 44, 169–179.

Rennie, M.J. (2005) Body maintenance and repair: how food and exercise keep the musculoskeletal system in good shape. *Experimental Physiology* 90, 427–436.

Sale, C., Saunders, B., & Harris, R.C. (2010) Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids* 39(2), 321–333.

Saltin, B. (1985) Physiological adaptation to physical conditioning. *Acta Medica Scandinavica* 711(Suppl.), 11–24.

Sjogaard, G. (1991) Role of exercise-induced potassium fluxes underlying muscle fatigue: a brief review. *Canadian Journal of Physiology and Pharmacology* 69, 238–245.

Starkie, R., Ostrowski, S.R., Jauffred, S., et al. (2003) Exercise and IL-6 infusion inhibit endotoxin-induced TNF-alpha production in humans. *FASEB Journal* 17, 884–886.

Steensberg, A., Fischer, C.P., Keller, C., et al. (2003) IL-6 enhances plasma IL-1ra, IL-10, and cortisol in humans. *American Journal of Physiology* 285, E433–E437.

van Loon, L.J. (2004) Intramyocellular triacylglycerol as a substrate source during exercise. *Proceedings of the Nutrition Society* 63(2), 301–307.

Wall, B.T., Stephens, F.B., Constantin-Teodosiu, D., et al. (2011) Chronic oral ingestion of L-carnitine and carbohydrate increases muscle carnitine content and alters muscle fuel metabolism during exercise in humans. *Journal of Physiology* 589(Pt 4), 963–973.

Westerblad, H., Allen, D.G., & Lannergren, J. (2002) Muscle fatigue: lactic acid or inorganic phosphate the major cause? *News in Physiological Sciences* 17(1), 17–21.

Wilkes, D., Gledhill, N., & Smyth, R. (1983) Effect of acute induced metabolic alkalosis on 800-m racing time. *Medicine & Science in Sports & Exercise* 15, 277–280.

Yfanti, C., Akerström, T., Nielsen, S., et al. (2010) Antioxidant supplementation does not alter endurance training adaptation. *Medicine & Science in Sports & Exercise* 42(7), 1388–1395.

PART 2

ENERGY AND MACRONUTRIENTS

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Chapter 4

How to Assess the Energy Costs of Exercise and Sport

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Introduction

Regular participation in exercise and sports activities confers numerous health- and performance-related benefits that contribute to a reduction in chronic diseases and disabling conditions, increased longevity, and an enhanced quality of life (Garber et al., 2011). The specific physiologic adaptations that occur with exercise activities depend on the frequency and duration of participation and the intensity of the sporting event. In general, the greater the product of the frequency, duration, and intensity of an exercise program, the greater the health- and performance-related benefits.

The energy expended in exercise and sports is the most variable portion of one's daily energy expenditure (EE). Total daily EE (TEE) is defined as the sum of the energy expended at rest (resting energy expenditure (REE)), the energy expended while eating and digesting food (thermic effect of food (TEF)), and the energy expended through daily physical activity and intentional exercise and sport (activity-related EE). The largest portion of one's TEE is from the REE (~55–70%) which is largely dependent on age, sex, height, and weight. Activity-related EE has the most influence on variations in daily TEE and is largely a function of the duration and intensity of acute bouts of activity.

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The energy cost of exercise can be estimated in a variety of ways. The greatest precision is obtained using direct measurements of heat production or of oxygen (O_2) utilization or carbon dioxide (CO_2) production in a laboratory chamber. Less precision is obtained from estimating activity-related EE from physiological data and movement monitors performed in laboratory and field settings. This chapter provides an overview of direct and indirect methods used in laboratory and field settings to assess the energy cost of exercise and sports activities performed by adults.

Definitions Related to Energy Expenditure

The construct of EE includes various components identified in Table 4.1. It is important to note that the terms physical activity, exercise, and sport are not synonymous. Physical activity refers to the construct of all movement, which may include movement for occupation, home maintenance, family care, transportation, and leisure-time activities, including exercise and sport. Exercise is a structured form of physical activity that has an outcome of one or more attributes of physical fitness. Sport is an organized form of exercise with rules of conduct, time, score, or distance boundaries, and an outcome of performance. Likewise, physical activity and EE differ. Physical activity involves moving the body or parts of the body through space and is measured as the product of frequency,

Table 4.1 Terms related to the construct of energy expenditure in exercise and sports

Energy	The capacity to do work
Energy expenditure	The exchange of energy required to perform biological work
Kilocalorie (kcal)	1000 calories, 4.184 kJ
Kilojoule (kJ)	The unit of energy in the International System of Units. 1000 joules or 0.238 kcal
Metabolic rate	The amount of energy liberated or expended in a given unit of time
Metabolic equivalent (MET)	A unit used to estimate the metabolic cost (oxygen uptake) of physical activity. One MET equals the resting metabolic rate of approximately 3.5 ml O_2/kg/min, 1 kcal/kg/h, or 4.184 kJ/kg/h
Direct calorimetry	Estimation of the energy expenditure through measurement of the amount of heat produced by a subject enclosed within a small chamber
Indirect calorimetry	Estimation of the energy expenditure through measurement of the amount of oxygen consumed and the amount of carbon dioxide produced
Body mass	A person's body weight
Physical activity	Bodily movement that is produced by the contraction of skeletal muscle and that substantially increases energy expenditure
Exercise	Planned, structured, and repetitive bodily movement done to improve or maintain one or more components of physical fitness. Exercise is a subcategory of physical activity
Sport	Organized form of exercise with rules of conduct, time, score, or distance boundaries, and an outcome of performance excellence
Frequency	The dimension of physical activity referring to how often an activity is performed
Duration	The dimension of physical activity referring to the amount of time an activity is performed
Intensity	The dimension of physical activity referring to the rate of energy expenditure while the activity is performed

Source: From Pettee et al. (2008), with permission.

duration, and intensity of movement and expressed as hours and/or minutes of an activity. EE is the outcome of physical activity and is measured in kilocalories (kcal) or kilojoules (kJ). The amount or volume of physical activity performed is referred to as a dose, described by the frequency of participation and duration of an acute bout of activity. The intensity can be measured by increases in the heart rate (HR), volume of air expired (VE), and oxygen uptake (VO_2). Oxygen uptake can be expressed in absolute terms as milliliters or liters per minute (VO_2 ml/min or l/min), relative to body mass as milliliters per kilogram body mass per minute (VO_2 ml/kg/min), or converted to EE as kilojoules using an energy equivalent of 21.0 kJ per liter of oxygen consumed. The intensity of physical activities is often expressed as a metabolic equivalent (MET) defined as the EE of a physical activity divided by the energy cost of rest or REE. The most precise measure of REE is by computing oxygen uptake under resting conditions. The standard value of one MET is 3.5 ml/kg/min. There are two points of view regarding the unit used to denote REE as the denominator when computing MET levels. Using one's measured REE is advised when computing an individual's MET level for an exercise or sport activity, as this accounts for individual differences in the REE which can result in MET levels that differ between individuals (Kozey et al., 2010). For population use, application of the standard REE value provides consistency between multiple assessments. The Compendium of Physical Activities, which provides MET values for a comprehensive list of physical activities, uses the standard REE value to denote the oxygen cost of rest. Table 4.2 provides an example of MET values for selected exercise and sports activities from the 2011 Compendium of Physical Activities (Ainsworth et al., 2011).

Methodologies to Estimate the Energy Cost of Exercise and Sport

Various measurement methods can be used to estimate the energy cost of exercise and sports. These include direct methods that have the highest precision yet are the most costly in terms of money and

Table 4.2 List of the metabolic costs (METs) of selected exercise and sports activities (Ainsworth et al., 2011)

Category	Activity	METs
Bicycling	Bicycling, leisure, 9.4 mph	5.8
	Bicycling, 12–13.9 mph, leisure, moderate effort	8
	Bicycling, 12 mph, seated, hands on brake hoods or bar drops, 80 rpm	8.5
	Bicycling, 14–15.9 mph, racing or leisure, fast, vigorous effort	10
Conditioning	Health club exercise classes, general, gym/weight training combined in one visit	5
	Water aerobics, water calisthenics, water exercise	5.3
	Circuit training, including kettlebells, some aerobic movement with minimal rest, general, vigorous intensity	8
	Bicycling, stationary, RPM/spin bike class	8.5
	Rope skipping, general	11
Dancing	Ballet, modern, or jazz, general, rehearsal, or class	5
	Aerobic dance, general	7.3
	General dancing (e.g., disco, folk, Irish step dancing, line dancing, polka, contra, country)	7.8
Fishing and hunting	Fishing, jog or line, standing, general	1.8
	Fishing, general, includes bending, walking	3.5
	Hunting, birds	3.3
	Hunting, hiking with hunting gear	9.5
Running	Jog/walk combination (jogging component of less than 10 min)	6
	Running, 5 mph (12 min/mile)	8.3
	Running, 7.5 mph (8 min/mile)	11.8
	Running, 10 mph (6 min/mile)	14.5
Sports	Billiards	2.5
	Volleyball, noncompetitive, 6–9 member team, general	3
	Bowling, indoor, bowling alley	3.8
	Golf, general	4.8
	Rock climbing, ascending or traversing rock, low-to-moderate difficulty	5.8
	Basketball, general	6.5
	Tennis, general	7.3
	Football (US soccer), competitive	10
Walking	Walking, 2.8–3.2 mph, level, moderate pace, firm surface	3.5
	Walking, 3.5 mph, level, brisk, firm surface, walking for exercise	4.3
	Walking, for exercise, 3.5–4 mph, with ski poles, Nordic walking, level, moderate pace	4.8
	Backpacking, hiking, or organized walking with a daypack	7.8
Water activities	Sailing, Sunfish/Laser/Hobby Cat, keel boats, ocean sailing, yachting, leisure	3.3
	Canoeing, rowing, 4–5.9 mph, moderate effort	5.8
	Swimming, crawl, medium speed, ~50 yards/min, vigorous effort	8.3
	Water jogging	9.8
	Windsurfing or kitesurfing, crossing trial	11
Winter activities	Skiing, downhill, alpine or snowboarding, light effort, active time only	4.3
	Snow shoeing, moderate effort	5.3
	Skating, ice, general	7
	Skiing, cross country, 4–4.9 mph, moderate speed and effort, general	9
	Skiing, cross-country, skating	13.3

participant burden and indirect methods that are less expensive and time consuming for participants but are less precise. The energy cost of movement can be measured by direct and indirect calorimetry (Schutz, 1995), doubly labeled water (DLW), HR, body temperature, and ventilation. Each method has strengths and limitations that will be highlighted.

Direct Methods

Direct methods used to estimate the energy cost of exercise and sport include laboratory-based room calorimetry, field-based DLW, and labeled bicarbonate.

Direct calorimetry measures the heat production during rest and movement. It requires a specialized chamber (room calorimeter) with sensors capable of measuring the heat generated as a by-product of metabolic processes. Oxygen is supplied to the chamber and carbon dioxide is collected to determine the rate of O_2 consumption and the rate of carbon dioxide production (VCO_2): these values are used to compute the respiratory exchange ratio (*R*) as VCO_2/VO_2. The *R*-value is an indirect indicator of the respiratory quotient (RQ) which is an index of substrate metabolism: the RQ as measured at the tissue is a true index of substrate use, but the *R*-value as measured in expired air is influenced by the exchange of CO_2 with the plasma bicarbonate pool. Combustion of only fat sources of fuel would result in an *R*-value of 0.7 and combustion of only carbohydrate fuel sources (as seen in high-intensity exercise) results in an *R* of 1. Fats produce the most energy per gram (39.3 kJ/g), protein second highest (~23.7 kJ/g), and carbohydrates the least (18 kJ/g).

Room calorimeters are used frequently in research settings to identify the effects of exercise and diet on EE. They are also used in clinical settings to understand metabolic anomalies and the effects of treatments on these anomalies. Calorimetry chambers are rarely used to assess the energy costs of specific activities or exercise routines because of the expense in operating the chamber, the time it takes to calibrate the system prior to assessments, and the limited space to perform the activity. Consequently, measures other than calorimeters often are used to estimate the energy costs of exercise and sports.

DLW is useful to estimate TEE over a short period of time (7–21 days) to assess the average daily EE of individuals. EE is estimated from analyzing labeled isotopes in the release of H_2O and CO_2 produced by varying frequency, duration, and intensity of movement. Body water is labeled by ingestion of a solution containing stable water isotopes ^2H_2O and $H_2{}^{18}O$ in volumes proportional to body weight. The isotopes are collected in urine and tracked using an isotope-ratio mass spectrometer prior to ingesting the labeled isotopes (referent value), shortly after ingestion (baseline), and during the subsequent days of free-living physical activity representing the rate of EE. The labeled hydrogen ($^2H_2$) is excreted in water in the urine, breath, and sweat, while the labeled oxygen (^{18}O) is lost as water and also in carbon dioxide. The difference in the isotope turnover rate provides a measure of VCO_2, which can be used to estimate VO_2 and to compute the RQ and TEE (Brockway, 1987).

The DLW method is considered a "gold standard" for field assessments of EE and has an accuracy of ±6% of TEE values obtained in a room calorimeter (Seale & Rumpler, 1997). DLW has several advantages over other methods due to its precision in estimating TEE. Also, it does not require individuals to wear monitors or sensors or stay confined in a small chamber. However, DLW cannot estimate the energy cost of individual activities and the method can be inconvenient and expensive. Individuals undergoing DLW monitoring must collect their urine output in a plastic container at the beginning and end of the observation period for laboratory analysis. Also, the expense of the labeled isotopes is high, with a single dose costing approximately US$300.

Labeled bicarbonate is a similar to the DLW method to assess the energy costs of total body movement. It differs from DLW in the solution, delivery method used to administer the isotopes, and the time needed to estimate TEE (days vs. weeks). Individuals are infused with a bicarbonate solution with the carbon atom isotopically labeled ($NaH^{14}CO_3$) that is absorbed into the body's carbon pool. The labeled carbons are recovered from expired air, blood, urine, or saliva. As with DLW, VCO_2 is determined from the dilution of the labeled carbons with EE calculated from VCO_2 and estimated RQ. The estimation of TEE is accurate to within 6% of values obtained in calorimetry chambers. While the precision of estimate is good, a major limitation is the complexity of the method and the need to infuse the solution intravenously.

Indirect Methods

Indirect methods used to estimate the energy cost of exercise and sports include indirect calorimetry (VO_2),

physiological measures (HR, body temperature, ventilation), and movement monitors (accelerometers, pedometers, and multi-modal sensors).

Indirect calorimetry can be used to estimate REE and activity-related EE through the assumed relation between VO_2 and the energy costs of substrate oxidation (McArdle et al., 1991). Based on the underlying principle that the aerobic production of energy requires oxygen, a rough estimate of the energy produced during exercise is 21 kJ per liter of oxygen consumed. To measure EE using indirect calorimetry, VO_2 and VCO_2 must be measured. This requires measuring the fraction of O_2 (F_IO_2) and CO_2 (F_ICO_2) in inspired air, the fraction of O_2 (F_EO_2) and CO_2 (F_ECO_2) in expired air, and the volume of inspired air (V_I) and expired air (V_E). The inspired or expired air volume is measured with a ventilation meter and the fraction of O_2 and CO_2 in the expired air is measured with O_2 and CO_2 analyzers. EE is computed through a multiple-step process of first computing the VO_2, VCO_2, and *R*-value and then using the Weir equation (Weir, 1949) to estimate EE:

$$VO_2 = (V_I \times F_IO_2) = (V_E \times F_EO_2)$$
$$VCO_2 = (V_E \times F_ECO_2) - (V_I \times F_ICO_2)$$
$$R = VCO_2/VO_2$$
$$EE(kJ) = [VO_2(3.9 + 1.1R)] \times 4.186$$

The VO_2 values obtained from the above formulae (McMurray & Ondrak, 2008) are in liters per minute. To compare the VO_2 between people, a value relative to an individual's total body mass (ml/kg/min) or fat-free mass (ml/kg fat-free mass/min) must be used.

Indirect calorimetry has been used to determine the MET levels commonly used to express the energy cost of exercise and sports activities and to assess the metabolic cost of movement. Indirect calorimetry can be performed in a laboratory using a metabolic cart (e.g., ParvoMedics, Sandy, UT, USA) or in a field setting using a portable oxygen uptake analyzer (e.g., Oxycon Mobile, CareFusion, San Diego, CA, USA). While many exercises can be modified for performance in a laboratory setting, as in pedaling a cycle ergometer or running on a treadmill, a major limitation of measuring oxygen uptake in a laboratory is the need to adapt body movements so expired air can be transferred from the mouthpiece through a plastic hose to the metabolic cart. Portable oxygen uptake analyzers provide the mobility needed to assess the oxygen cost of exercise and sports as they are performed in field settings, without the limitations noted in a laboratory setting. This allows for more natural movement patterns that may reflect a truer oxygen cost of movement. Nevertheless, portable oxygen uptake systems still require wearing cumbersome equipment to assess VO_2 in field settings, which has the potential to alter the gross activity-related EE during free-living settings.

HR responses to exercise can be used to identify the intensity of exercise performed and to estimate activity-related EE. Three ways to express the HR response to exercise are the percentages of maximal HR ($\%HR_{max}$) and HR reserve (%HRR) and the FLEX HR (ACSM, 2009). The $\%HR_{max}$ is computed as a percentage of one's measured or predicted maximal HR (220 − age) and is used most often to establish exercise intensity thresholds to optimize aerobic endurance. The %HRR is the percentage of difference between one's measured or predicted maximum HR and resting HR and reflects the range of heart beats that can increase during exercise. Predicting the energy costs of exercise from the HR is based on the assumption of a strong, linear relation between HR and VO_2 during steady-state exercise involving large muscle groups (Wilmore & Haskell, 1971) (Figure 4.1). This assumption is true only for HR values in the middle of the HR–VO_2 curve. During low-intensity exercise, there is a nonlinear relation between the HR and EE due to a lag in the HR response to exercise. During high-intensity exercise, the HR levels off while the energy cost of an activity may continue to increase. The FLEX HR was developed to account for the nonlinearity in the HR–VO_2 curve and to account for the individual differences in estimating the activity-related EE from the HR–VO_2 relationship (Spurr et al., 1988). Determined using laboratory-based indirect calorimetry, the FLEX HR identifies the HR value threshold between linear and nonlinear parts of the HR–VO_2 curve. For HR values below the FLEX HR, the activity-related energy cost is assigned REE values. Activities eliciting an HR above the FLEX HR are assigned an activity-related energy cost based on regression

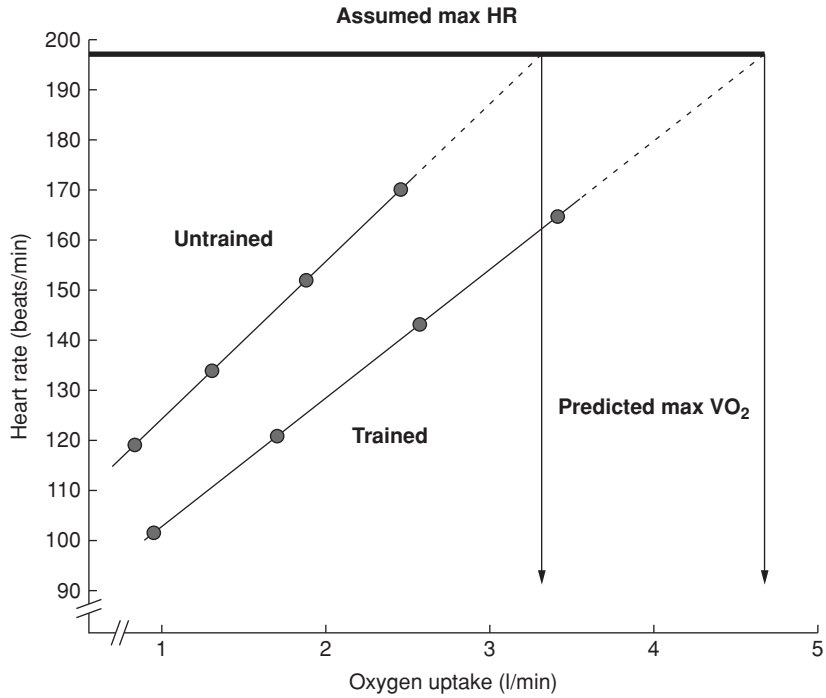

Figure 4.1 Application of the linear relationship between submaximal heart rate and oxygen uptake. Note that trained individuals are able to work at a higher absolute workload (%VO_2 max) with a lower heart rate than in untrained individuals.

equations developed for an individual's HR–VO_2 calibration curve (Livingstone et al., 1990). Algorithms used to estimate the EE of activity from the FLEX HR are provided as follows:

REE = REE (HR < FLEX HR)
Activity-related EE = EE from regression equation (HR ≥ FLEX HR)

The FLEX HR method has acceptable correlations when used to estimate TEE from the HR values (r = 0.73) and from DLW (r = 0.53) (Heymsfield et al., 2005). However, between-person measurement variability is high. Livingstone et al. (1990) reported differences of –22% to +52% between TEE estimated from DLW and from the FLEX HR. Readers interested in a detailed description of the FLEX HR method are directed to the web site describing physical activity and EE assessment methods maintained by the Medical Research Council (MRC, 2011).

In 1997, Swain and Leutholtz noted that the %HRR had a stronger relationship to the %VO_2 max reserve (%VO_2R computed as VO_2 max – VO_2 rest) than the %VO_2 max during cycling exercise. Comparing activity-related EE obtained from indirect calorimetry with predicted values from the %VO_2R–%HRR curve showed a near-perfect association, suggesting these models have greater precision to estimate activity-related EE than the %VO_2 max–%HHR curve models. A strong relationship between the %VO_2R and %HRR curve has also been observed during treadmill running (Swain et al., 1998) and in elliptical cross-trainer exercise (Dalleck & Kravitz, 2006) in healthy young adults.

The strength of using HR values to predict the energy cost of exercise lies in its simplicity and access to charts that can be used to identify activity-related EE from HR values. Limitations to the method are in the mismatch between the %HR and activity-related EE during low and high exercise intensities, the variability in the HR response to exercise caused by dehydration, ambient heat conditions, aerobic fitness levels, and with some

medications (e.g., β-blockers) that alter the ability of the HR to increase with increased exercise intensity. The variability in the HR response to exercise between persons also makes it difficult to generalize a standard HR–EE curve for all persons.

Body temperature and ventilation reflect the relationships between activity-related EE and increases in the core body temperature and ventilation during exercise. Predicting EE from the volume of air expired each minute (VE) was first proposed by Durnin and Edwards in 1955. While the relationship is modified by sex, age, race, and aerobic fitness levels in adults, the equation of EE (kcal/min) = 0.210VE was developed to predict activity-related EE. Datta and Ramanathan (1969) showed high correlations (r = 0.8–0.99) between VE and activity-related EE in repeated exercise studies with Indian adults. However, the percentage error around the predicted activity-related EE is wide (5–21%), making the method unsuitable for estimating the energy cost of exercise and sports from VE values.

The body temperature can be used to estimate activity-related EE in laboratory and clinical settings, but this method is impractical due to the delay in the increase in body temperature following the onset of exercise. Commercial sensors (e.g., SenseWear Pro Armband™) incorporate body temperature measures into a multimodal body sensor device to predict EE from a variety of feedback, including body heat generated during exercise.

Movement monitors are used primarily to assess physical activity, but they can also be used to estimate the energy costs of exercise. Types of movement monitors used most frequently are pedometers, accelerometers, and multimodal sensors. A detailed discussion of the validity and reliability of movement monitors is provided elsewhere (Bassett & Dinesh, 2010).

Pedometers are low-cost, battery-operated digital step counters that have gained widespread popularity in physical activity studies (Tudor-Locke et al., 2011), physical activity promotion programs (VanWormer et al., 2006), and clinical practice (Stovitz et al., 2005). While many pedometers have been manufactured, only a few (e.g., Yamax Digi-walker SW-2000, Omron HJ-303 and HJ-105 New Lifestyles NL-2000, Kenz Lifecorder) have acceptable accuracy when compared with actual steps taken (Schneider et al., 2004). Pedometers generally are worn at the waist and are triggered by the vertical accelerations of the hip that cause a horizontal spring-suspended level arm circuit. Tudor-Locke et al. (2005) used pedometers to compare the step rate (steps/min) with VO_2 determined by indirect calorimetry to identify the relationship between a pedometer-determined steps/min and ambulatory-related EE. They developed regression equations to estimate MET values for males and females with high predictive validity as shown here:

Men: METs = $-7.065 + (0.105 \times \text{steps/min})$ ($R^2 = 0.8$)
Women: METs = $-8.805 + (0.110 \times \text{steps/min})$ ($R^2 = 0.83$)

This translates into the following steps/min for graded intensities of exercise as follows:

	Steps/min	
	Men	Women
Light (≤2.99 METs)	<96	<107
Moderate (3–5.99 METs)	96–124	107–135
Hard (8–8.99 METs)	125–153	136–162
Very hard (≥9 METs)	>153	>162

Using these step rates, 30 minutes of walking at a moderate intensity corresponds to about 3000 steps for men and women.

Accelerometers are small, battery-operated electronic motion sensors that measure the rate and magnitude of displacement of the body's center of mass during movement. The placement of accelerometers varies with the brand and model. Most are worn on the waist, wrist, or upper arm. Types of accelerometers include uniaxial models that detect movement in the vertical plane and triaxial models that detect movement in the vertical and horizontal planes. The value of triaxial models is that movements involving climbing activities that increase the energy cost of an activity, such as stair climbing and hiking, can be factored into ambulatory-related EE equations whereas uniaxial accelerometers are unable to detect the added energy cost of such activities. The most common types of accelerometers used to assess movement and to estimate

ambulatory-related EE in field settings are the ActiGraph (ActiGraph LLC, Pensacola, FL, USA) and the Actical (Respironics, Philips, The Netherlands) models. The ActiGraph accelerometer was first marketed in the 1990s under the name Computer Science Applications (CSA). This early uniaxial accelerometer detected movement intensity, duration, and steps taken but had limited battery life and memory to store data. With advances in technology, the ActiGraph in use today uses a micro-electro-mechanical system triaxial accelerometer (GT3X+ with 512 MB memory) with a 31-day battery life and memory capable of storing raw movement data for 40 days. The ambulatory data are sampled at a user-specified rate up to 100 Hz that can be aggregated and stored in epochs (sampling intervals) as frequent as 1 second or longer. Output data are downloaded using proprietary software and stored in a computer database. Exercise intensities have been determined by correlating the intensity-related movement output (counts) with VO_2 measured during walking and running on laboratory treadmills and during free-living movements. Freedson et al. (1998) developed the first count values associated with exercise intensity levels ranging from zero (no movement) to >5725 counts (vigorous intensity). Because the Freedson cut-points were developed during treadmill walking and running, others have developed cut-points to reflect physical activity intensities during lifestyle activities in adults (Hendelman et al., 2000; Swartz et al., 2000) and in children (Trost et al., 2006). Efforts to estimate the energy costs of movement from ActiGraph accelerometer data have yielded numerous equations. The equation cited most often for adults is the simple, single regression model developed by Freedson et al. (1998):

$$\text{kJ/min} = [(0.00094 \times \text{counts/min}) + (0.1346 \times \text{mass in kg})] \times 4.186$$

The precision of the equation is $R^2 = 0.82$ (SEE = 1.4 kcal/min). However, it has been noted that regression equations developed from walking and jogging overestimate the energy cost of many walking, light-, and moderate-intensity lifestyle activities. In 2006, Crouter et al. developed a 2-regression model for the ActiGraph accelerometer that distinguishes the activity-related EE between continuous walking, running, and intermittent lifestyle activities. In 2010, Crouter et al. revised the equation to better reflect the intermittent nature of movement as detected by the ActiGraph accelerometer. They noted that people seldom perform activities continuously without starting and stopping for brief moments. Thus the investigators developed a METs prediction equation using a 10-second movement recording epoch that accounts for the variable nature of human movement as presented in the following:

1. If the ActiGraph counts·10/second are ≤8, the EE = 1.0 MET.
2. If the ActiGraph counts·10/second are >8
   a. and the coefficient of variation (CV) of the counts·10/second is ≤10, then EE (METs) = 2.2949395 × [exp(0.00084679 × ActiGraph counts·10/second)] $R^2 = 0.739$; SEE = 0.250;
   b. or the CV of the counts·10/second is >10, then EE (METs) = 0.749395 + [0.716431 × (ln(ActiGraph counts·10/second)) − (0.179874 × (ln(ActiGraph counts·10/second))^3)] R^2 = 0.84; SEE = 0.863.
3. Once a MET value has been calculated for each 10-second epoch within a minute on the ActiGraph clock, the average MET value for six consecutive 10-second epochs within each minute is calculated to obtain the average MET value for that minute.

The CV is defined as the ratio of the standard deviation of the counts·10/second to the mean of the counts·10/second (standard deviation/mean). The activity-related EE can be measured from METs using the following equation:

$$\text{Activity-related EE in kJ/min} = (\text{METs} \times \text{fraction of hours of movement} \times \text{body mass in kg}) \times 4.186$$

The strengths of estimating the activity-related EE from the ActiGraph accelerometer is that the ActiGraph is used worldwide and the equations have reasonable predictive validity. However, a downside is that the equations are complex and computer programs are needed to make the calculations. Thus, the ActiGraph is often used to identify

the time spent in varying intensities of physical activity but seldom used to estimate the energy cost of exercise.

The Actical is another accelerometer used frequently to assess the frequency, duration, and intensity of physical activity. The Actical is a small, triaxial monitor that is worn on the wrist. Operating on similar principles to the ActiGraph, the Actical uses prediction models to reflect the time spent in inactivity and varying intensities of physical activity. Various regression equations have been developed to estimate activity-related EE in adults with varying precision (Lyden et al., 2011). A regression model with good predictive validity has been developed by Crouter et al. (2011) to estimate EE during free-living physical activities. Using a 15-second epoch to aggregate time spent in varying intensities, the following procedures are followed to estimate EE expressed as METs in adults:

1. If the counts·15/second are <35, EE = 1 METs.
2. If the counts·15/second are >35 but <85, EE = 1.83 METs.
3. If the counts·15/second are ≥85
   a. and the CV of the counts·15/second is ≤13%, the EE (METs) = 2.522276 × [exp(0.00055462 × Actical counts·15/second)]R^2 = 0.925; SEE = 0.135;
   b. and the CV of the counts·15/second is >13%, the EE (METs) = 2.1724798 + (0.0072286 × Actical counts·15/second)R^2 = 0.797; SEE = 1.092.
4. Once a MET value has been calculated for each 15-second epoch within a minute on the Actical clock, the average MET value of four consecutive 15-second epochs within each minute is calculated to obtain the average MET value for that minute.

As with the ActiGraph, calculating the activity-related EE from the Actical involves a series of steps that require laboratory settings and computational skills.

Multimodal sensors include monitors that use physiological and movement data to estimate EE. Popular multimodal sensors include the Actiheart (CamNtech, Cambridge, UK) and SenseWear Pro Armband™ (Body Media, Pittsburgh, PA, USA). The Actiheart is a small monitor worn around the chest that records HR, inter-beat-interval from a single electrocardiogram lead, and physical activity from an accelerometer. The Actiheart can record up to 440,000 heart beats using 1-minute epoch integration duration and can store movement and HR data for 21 days. Using proprietary software, the Actiheart can compute REE, activity-related EE, TEF, TEE, and the physical activity level (PAL). The PAL is computed as the ratio of the TEE and REE (TEE/REE) and is an indicator of daily PALs (FAO/WHO/UNU, 2001).

Activity level	Example	PAL
Extremely inactive	Mostly sitting/reclining	<1.4
Low active	Low-intensity lifestyle activity	1.4–1.69
Moderately active	Moderate-vigorous exercise >30 min/day	1.7–1.99
Very active	Moderate-vigorous exercise >1 h/day	2–2.4
Extremely active	Competitive endurance athlete	>2.4

Crouter et al. (2008) tested the accuracy of the activity-related EE values computed by the Actiheart manufacturer's algorithm in 48 men and women aged 35 ± 11 years. Activity-related EE was measured by VO_2 and HR monitoring. Values were within 0.09 kJ·kg/min difference during 18 structured activities of varied intensities. While the Actiheart is accurate in estimating the energy cost of exercise, wearing the device around the chest may be cumbersome for some individuals with excessive body mass. Further, loss of conductivity for the chest electrode can result in a loss of the signal needed to compute activity-related EE.

The SenseWear Pro Armband™ contains a triaxial accelerometer that measures motion and steps taken, galvanic monitor that measures changes in the skin's electrical conductivity in response to sweat and emotional stimuli, an electronic thermometer that measures skin temperature, and a heat flux sensor that measures the amount of heat dissipated from the body. Worn on the upper arm,

data are integrated and downloaded to a web site that computes activity-related EE using proprietary software. A validation study by Koehler et al. (2011) in male endurance athletes showed acceptable validity against DLW (r = 0.73) but the limits of agreement between EE values were wide (–5746 to +5200 kJ/day) and unacceptable. Further, the SenseWear Pro Armband™ has been shown to underestimate activity-related EE during running and bicycling activities (Brazeau et al., 2011; Drenowatz & Eisenmann, 2010). This underestimation increases with increasing exercise intensity. Thus, while integrated sensors have utility in estimating the energy cost of exercise, individual variation in the movement economy and physiological responses to movement make it difficult to identify common activity-related EE prediction equations that can be applied to all people with sufficient precision.

Summary

Several direct and indirect methods are available to measure the energy costs of exercise and sports under controlled laboratory conditions and in free-living settings. These measurements exist along a continuum of having high precision yet low feasibility to having high feasibility yet lower precision in estimating activity-related EE. Laboratory measures involving direct and indirect calorimetry have high precision to estimate activity-related EE, but limit movement patterns due to their use of small metabolic chambers and/or the need for laboratory equipment that may alter activity movement patterns. On the other end of the continuum, field methods used to measure activity-related EE are increasing in precision and ease of use, but still rely on prediction equations to estimate EE that have considerable error when applied to individuals.

References

ACSM (2009) *ACSM's Guidelines for Exercise Testing and Prescription*. Lippincott Williams & Wilkins, Riverwoods, IL.

Ainsworth, B., Haskell, W., Herrmann, S., et al. (2011) 2011 Compendium of Physical Activities: a second update of codes and MET values. *Medicine & Science in Sports & Exercise* 43, 1575–1581.

Bassett Jr, D.R. & Dinesh, J. (2010) Use of pedometers and accelerometers in clinical populations: validity and reliability issues. *Physical Therapy Reviews* 15, 135–142.

Brazeau, A.-S., Karelis, A., Mignault, D., et al. (2011) Accuracy of the SenseWear Armband™ during ergocycling. *International Journal of Sports Medicine* 32, 761–764.

Brockway, J. (1987) Derivation of formulae used to calculate energy expenditure in man. *Human Nutrition: Clinical Nutrition* 41C, 463–471.

Crouter, S.E., Clowers, K.G., & Bassett, D.R. (2006) A novel method for using accelerometer data to predict energy expenditure. *Journal of Applied Physiology* 100, 1324–1331.

Crouter, S., Churilla, J., & Bassett, D.J. (2008). Accuracy of the Actiheart for the assessment of energy expenditure in adults. *European Journal of Clinical Nutrition* 62, 704–711.

Crouter, S.E., Kuffel, E., Haas, J.D., et al. (2010) A refined 2-regression model for the ActiGraph accelerometer. *Medicine & Science in Sports & Exercise* 42, 1029–1037.

Crouter, S., Della Valle, D., Horton, M., et al. (2011) Validity of the Actical for estimating free-living physical activity. *European Journal of Applied Physiology and Occupational Medicine* 111, 1381–1389.

Dalleck, L. & Kravitz, L. (2006) Relationship between %heart rate reserve and $\%VO_2$ reserve during elliptical crosstrainer exercise. *Journal of Sports Science and Medicine* 5, 662–671.

Datta, S. & Ramanathan, N. (1969) Energy expenditure in work predicted from heart rate and pulmonary ventilation. *Journal of Applied Physiology* 26, 297–302.

Drenowatz, C. & Eisenmann, J. (2010) Validation of the SenseWear Armband at high intensity exercise. *European Journal of Applied Physiology* 111, 883–887.

Durnin, J. & Edwards, R. (1955) Pulmonary ventilation as an index of energy expenditure. *Quarterly Journal of Experimental Physiology and Cognate Medical Sciences* 40, 370–377.

FAO/WHO/UNU (2001) *Human Energy Requirements: Report of a Joint FAO/WHO/UNU Expert Consultation*. FAO/WHO/UNU, Rome.

Freedson, P.S., Melanson, E., & Sirard, J. (1998) Calibration of the Computer Science and Application, Inc. accelerometer. *Medicine & Science in Sports & Exercise* 30, 777–781.

Garber, C.E., Blissmer, B., Deschenes, M.R., et al. (2011) Quantity and quality of exercise for developing and maintaining cardiorespiratory, musculoskeletal, and neuromotor fitness in apparently healthy adults: guidance for prescribing exercise. *Medicine & Science in Sports & Exercise* 4, 1334–1359.

Hendelman, D., Miller, K., Baggett, C., et al. (2000) Validity of accelerometry for the assessment of moderate intensity physical activity in the field. *Medicine & Science in Sports & Exercise* 32, S442–S449.

Heymsfield, S., Lohman, T., Wang, Z.-M. et al. (2005) *Human Body Composition*. Human Kinetics, Champaign, IL.

Koehler, K., Braun, H., de Marées, M., et al. (2011) Assessing energy expenditure in male endurance athletes: validity of the SenseWear Armband. *Medicine & Science in Sports & Exercise* 43, 1328–1333.

Kozey, S., Lyden, K., Staudenmayer, J., et al. (2010) Errors in MET estimates of physical activities using 3.5 ml × kg(–1) × min(–1) as the baseline oxygen consumption. *Journal of Physical Activity and Health* 7, 508–514.

Livingstone, M.B., Prentice, A.M., Coward, W.A., et al. (1990) Simultaneous measurement of free-living energy expenditure by the doubly labeled water method and heart-rate monitoring. *American Journal of Clinical Nutrition* 51, 59–65.

Lyden, K., Kozey, S.L., Staudenmeyer, J.W., et al. (2011) A comprehensive evaluation of commonly used accelerometer energy expenditure and MET prediction equations. *European Journal of Applied Physiology* 111, 187–201.

McArdle, W.D., Katch, F.I., & Katch, V.L. (1991) The respiratory quotient. In *Exercise Physiology: Energy, Nutrition, and Human Performance*, 3rd edn. Lea & Febiger, Philadelphia, PA.

McMurray, R.G. & Ondrak, K.S. (2008) Energy expenditure of athletes. In *Sports Nutrition: Energy Metabolism and Exercise* (eds. I. Wolinsky & J. Driskell), pp. 127–159. CRC Press, Boca Raton, FL.

MRC (2011) *Physical Activity Assessment—Heart Rate Monitoring* [Online]. Available: http://www.dapa-toolkit.mrc.ac.uk/physical-activity-assessment/methods/heart-rate-monitoring/index.html (accessed November 14, 2011).

Pettee, K., Tudor-Locke, C., & Ainsworth, B. (2008) The measurement of energy expenditure and physical activity. In *Sports Nutrition: Energy Metabolism and Exercise* (eds. I. Wolinsky & J. Driskell), pp. 159–193. CRC Press, Boca Raton, FL.

Schneider, P.L., Crouter, S., & Bassett, D.R. (2004) Pedometer measures of free-living physical activity: comparison of 13 models. *Medicine & Science in Sports & Exercise* 36, 331–335.

Schutz, Y. (1995) The basis of direct and indirect calorimetry and their potentials. *Diabetes/Metabolism Research and Reviews* 11, 383–408.

Seale, J. & Rumpler, W. (1997) Comparison of energy expenditure measurements by diet records, energy intake balance, doubly labeled water and room calorimetry. *European Journal of Clinical Nutrition* 51, 856–863.

Spurr, G.B., Prentice, A.M., Murgatroyd, P.R., et al. (1988) Energy expenditure from minute-by-minute heart-rate recording comparison with indirect calorimetry. *American Journal of Clinical Nutrition* 48, 552–559.

Stovitz, S.D., VanWormer, J.J., Center, B.A., et al. (2005) Pedometers as a means to increase ambulatory activity for patients seen at a family medicine clinic. *Journal of the American Board of Family Medicine* 18, 335–343

Swain, D. & Leutholtz, B. (1997) Heart rate reserve is equivalent to %VO_2reserve, not to %VO_2max. *Medicine & Science in Sports & Exercise* 29, 410–414.

Swain, D., Leutholtz, B., King, M., et al. (1998) Relationship between % heart rate reserve and %VO_2reserve in treadmill exercise. *Medicine & Science in Sports & Exercise* 30, 318–321.

Swartz, A., Strath, S., Bassett, D.J., et al. (2000) Estimation of energy expenditure using CSA accelerometers at hip and wrist sites. *Medicine & Science in Sports & Exercise* 32, S450–S456.

Trost, S.G., Way, R., & Okely, A. (2006) Predictive validity of three ActiGraph energy expenditure equations for children. *Medicine & Science in Sports & Exercise* 38, 380–387.

Tudor-Locke, C., Sisson, S., Collova, T., et al. (2005) Pedometer-determined step count guidelines for classifying walking intensity in a young ostensibly healthy population. *Canadian Journal of Applied Physiology* 30, 666–676.

Tudor-Locke, C., Craig, C., Cameron, C., et al. (2011) Canadian children's and youth's pedometer-determined steps/day, parent-reported TV watching time, and overweight/obesity: the CANPLAY Surveillance Study. *International Journal of Behavioral Nutrition and Physical Activity* 8, 66.

VanWormer, J., Pronk, N.P., & Boucher, J.L. (2006) Experience analysis of a practice-based, online pedometer program. *Diabetes Spectrum* 19, 197–200.

Weir, J.B. (1949) New methods for calculating metabolic rate with special reference to protein metabolism. *Journal of Physiology* 109, 1–9.

Wilmore, J.H. & Haskell, W.L. (1971) Use of the heart rate-EE relationship in the individualized prescription of exercise. *American Journal of Clinical Nutrition* 24, 1186–1192.

Chapter 5

Energy Balance and Energy Availability

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Goals of Athletic Training

Athletes strive to achieve bodies with the mass, composition, and mix of fuel stores that qualify them to compete and then optimize their performance on the day and at the time of competition. In this sense, all athletes are bodybuilders. Figure 5.1 is a photo of 22-year-old identical twins Otto and Ewald Spitz who had both been distance runners until they were 18 when Ewald changed his diet and exercise habits to compete in field events (discus, shot put, hammer throw, etc.) while Otto remained a runner (Kono, 2001).

Nutrition plays a co-equal role with exercise in achieving such bodybuilding objectives, which vary from sport to sport and from position to position in team sports. Furthermore, the most effective diets for achieving particular sets of these objectives are no more traditional, intuitive, or obvious than the most effective exercises. This chapter discusses the most general nutritional requirement affecting an athlete's body mass, composition, and fuel stores, namely energy. Necessarily, this discussion of energy quickly focuses on the special need of humans to derive energy from glucose. This special need imposes strict limitations on an athlete's diet and exercise behavior. These limitations are violated at the cost of health and performance, due to hormone-mediated effects on diverse physiological systems.

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Complicating the athlete's nutritional challenge is our lack of a strong biological drive to match energy intake to activity-induced energy expenditure. As a result, athletes must learn to eat by discipline instead of appetite. Unfortunately, efforts to develop optimal, coordinated diet and exercise disciplines are hampered by recently recognized ambiguities about whether various physiological responses to athletic training are effects of exercise or of energy deficiency. Many responses long thought to be effects of exercise disappear with increases in dietary energy (Braun & Brooks, 2008). As a result, considerable research remains to be done before dieticians will be able to manage the energy requirements of athletes with confidence.

Distinctions between Energy Balance and Energy Availability

Traditionally, dieticians have studied the effects of dietary energy on the health and performance of athletes in terms of energy balance, defined as dietary energy intake minus total energy expenditure (EB = EI – TEE). Conceptually, energy balance is the amount of dietary energy added to or lost from the body's energy stores after all of the body's physiological systems have completed their work for the day. As such, energy balance is an *output* from those systems.

Figure 5.2a shows a simple model of an athlete's energy expenditure (EE) over a 24-hour day. Although EE fluctuates greatly from minute to minute during waking hours, it is represented in Figure 5.2a as three average levels: a low aver-

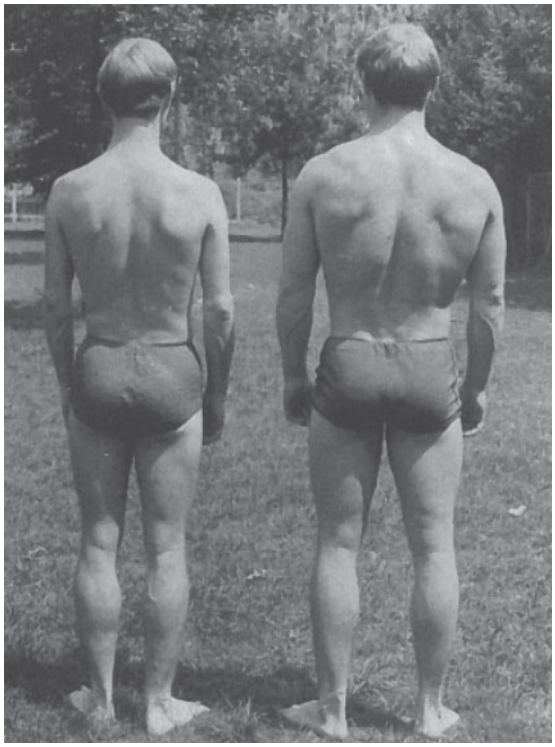

Figure 5.1 Identical twins, 22 years old, who had trained for different sports, Otto for long-distance running and Ewald for field events (discus, shot put, hammer throw, etc.). Source: From Kono (2001), with permission.

age during sleep, an intermediate average during routine waking activities other than exercise, and a high average during exercise. TEE is the sum of EE over the entire 24 hours. To manage EB, EI is controlled to equal TEE plus a chosen value of EB: EI = TEE + EB. In practice, this is difficult to do for two reasons. First, controlling EI is challenging; and second, TEE is much larger than EB, and uncertainty in the measurement of TEE is large compared to the desired value of EB.

By contrast, biologists traditionally study the effects of dietary energy on the health and reproduction of animals facing their special environmental challenges in terms of energy availability (Wade & Schneider, 1992), defined as dietary energy intake minus the energy expended in overcoming a particular challenge. Conceptually, energy availability is the amount of dietary energy remaining for all other physiological systems after the challenge has been overcome. As such, energy availability is an *input* to those systems.

For example, biologists investigating the effects of cold exposure, or of the energy cost of foraging for food, on animal reproduction have defined, quantified, and controlled energy availability as dietary energy intake minus the energy expended in thermogenesis, or foraging. Inspired by their example, physiologists investigating the effects of exercise on the reproductive function of female athletes have defined energy availability as dietary energy intake minus the energy expended in exercise (EA = EI – EEE).

The concept of energy availability recognizes that dietary energy is expended in several fundamental physiological processes, including cellular maintenance, thermoregulation, growth, reproduction, immunity, and locomotion, and that the energy expended in one of these processes is not available for the others (Wade & Jones, 2004). As a result, the health of an animal (i.e., the proper functioning of these physiological processes) depends not on energy balance (i.e., on the amount of weight the animal gains or loses each day) but rather on the amount of dietary energy that remains as an input for its physiological systems after the animal has coped with its special environmental challenge(s); e.g., *after* foraging for food, or *after* keeping warm, or *after* mounting an immune defense, or in the case of athletes *after* exercise training.

Figure 5.2b shows a slightly more elaborate model of an athlete's EE over a 24-hour day. In Figure 5.2b, the low level of EE during sleep is extended throughout the day, because the physiological processes occurring during sleep continue through the day. The intermediate level of EE during routine waking activities is extended through the exercise training period, because that is the level of EE on a non-exercising day. The remaining EE is the amount specifically associated with exercise (EEE). To calculate EA, EEE is measured as the difference between total energy expenditure during exercise and total energy expenditure in routine waking activities during the same time period ($EEE = TEE_X - TEE_W$). To manage EA, EI is controlled to equal EEE plus a chosen value of EA: EI = EEE + EA. (To account for individual differences, all these quantities are

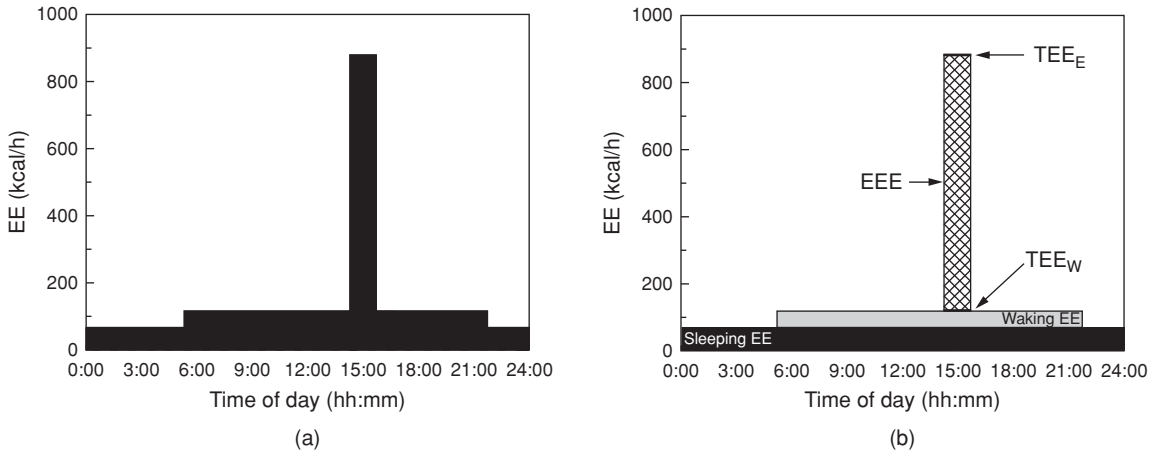

Figure 5.2 Twenty-four-hour energy expenditure (EE) of an athlete. Fluctuating EE is approximated by three average levels: a low average during sleep, an intermediate average during routine waking activities other than exercise, and a high average during exercise. (a) For calculating energy balance, total energy expenditure is estimated as the sum of all energy expended in 24 hours. (b) For calculating energy availability, exercise energy expenditure (EEE) is calculated as the total energy expended during exercise (TEE_E) minus the total energy expended during the same period of a day when not exercising (TEE_W). (1 kcal = 4.18 kJ).

normalized to body size.) In practice, EA can be managed more accurately than EB, even though the difficulty of controlling EI remains, because EEE is smaller than EA, and uncertainty in the measurement of EEE is very small compared to the desired value of EA.

The contrast between EB and EA is illustrated in Figure 5.3, which shows the data collected while eight lean, untrained men lived in a room calorimeter for a week (Stubbs et al., 2004). During that week, their EI = 2770 kcal/day (11.6 MJ/day), EEE = 840 kcal/day (3.5 MJ/day), and EA = EI − EEE = 2770 − 840 = 1930 kcal/day (11.6 − 3.5 = 8.1 MJ/day) were held constant by their voluntary behavior. Nevertheless, their EB = EI − TEE = 2770 − 4500 = −1730 kcal/day (11.6 − 18.8 = −7.2 MJ/day) on day 1 declined toward 0 during the week, because TEE declined at a rate of ~90 kcal/day (375 kJ/day) as various involuntary physiological processes slowed down. At this rate, the men would have recovered EB = 0 kcal/day—a *pathological* state of energy balance achieved by the involuntary and imperceptible suppression of physiological systems—in 3 weeks, while they remained in severely low energy availability. Figure 5.3 also illustrates a common error in the field of dietetics, which is to estimate energy requirements by measuring energy expenditure. Measurements of energy expenditure contain no information about whether physiological systems are functioning in a healthy manner.

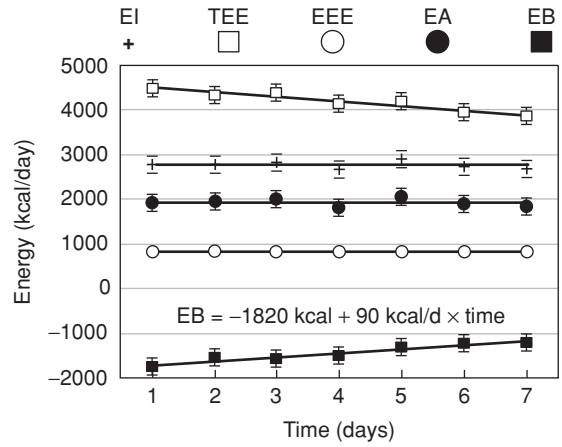

Figure 5.3 Suppression of energy expenditure by low energy availability. As eight lean men lived in a room calorimeter for 1 week, their energy intake (EI), exercise energy expenditure (EEE), and low energy availability (EA) were constant. Meanwhile, their total energy expenditure (TEE) declined toward EI and negative energy balance (EB) increased toward 0 as physiological processes were suppressed. (1 kcal = 4.18 kJ).

Hormones regulating physiological processes respond to energy availability, not to the intake or expenditure of energy and not to the "stress" of exercise (Loucks, 2009). For example, Figure 5.4a shows the diet and exercise treatments administered for 4 days to six groups of healthy young women (Loucks & Callister, 1993). Three groups were administered energy availability A = 30 kilocalories per kilogram of body mass per day (kcal/kgBW/day) (125 kJ/kgBW/day) and three others were administered 8 kcal/kgBW/day (33 kJ/kgBW/day). At each energy availability, one group (ZERO) performed no exercise, while two other groups expended E = 22 kcal/kgBW/day (92 kJ/kgBW/day) in exercise. One of these groups (L) exercised at a low intensity of 40% VO_2 max and the other at a high intensity (H) of 70% VO_2 max. Figure 5.4b shows that the resulting suppression of triiodothyronine (T_3) was associated with low energy availability (A) and not with low dietary energy intake (I), exercise energy expenditure (E), or exercise intensity (Zero, H, L). Other hormones respond similarly (Loucks et al., 1998).

Hormone Responses to Low Energy Availability

Animals have evolved neuroendocrine mechanisms centered in the brain that promote survival through periods of energy deficiency. The human brain accounts for only ~2% of body weight, but this is very large compared to other animals. The brain also has a very high metabolic rate compared to other tissues, accounting for ~20% of resting metabolism in adults and ~50% in children. Furthermore, under normal circumstances neurons are fueled almost exclusively by glucose. As a result, the human brain oxidizes ~100 g/day of glucose. Because the brain stores very little glucose in the form of glycogen (~1 g), its primary source of energy is the glucose circulating in the blood. We are here because our brain has evolved special neurons that sense the amount of glucose circulating in the blood along with several afferent hormone signals of energy deficiency from peripheral tissues, and because the brain has evolved other special neurons that secrete efferent hormone signals that cause glucose to be released

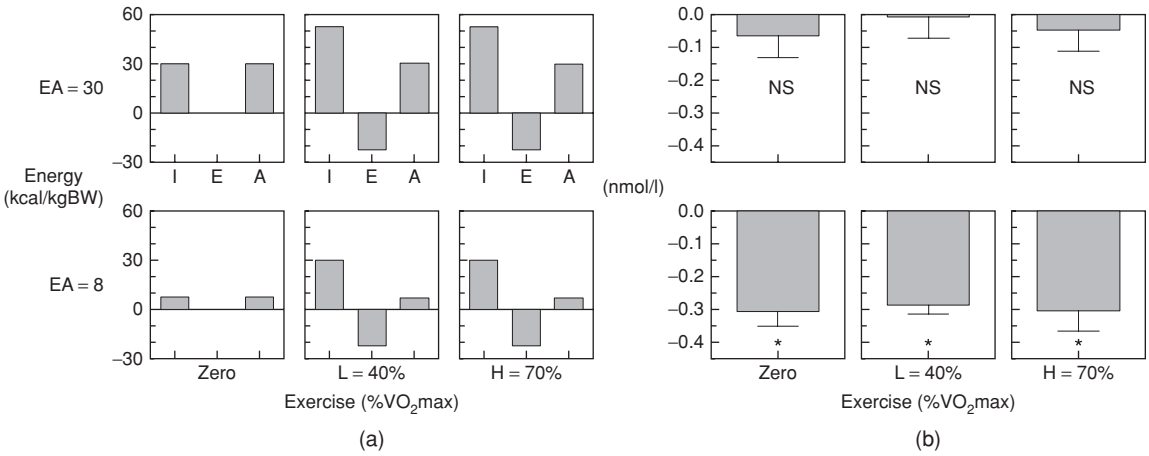

Figure 5.4 Suppression of triiodothyronine. (a) Experimental design: for 4 days, various diet and exercise treatments were administered to six groups of healthy young women. Energy intake (I) and exercise energy expenditure (E) were controlled to administer a balanced (B = 30 kcal/kgBM/day = 125 kJ/kgBM/day) energy availability (A) to three groups and a deficient (D = 8 kcal/kgFFM/day = 33 kJ/kgBM/day) energy availability to three other groups. At each energy availability, one group performed zero exercise (Z) while two groups expended 22 kcal/kgBM/day = 92 kJ/kgBM/day in exercise at either a low (L = 40% VO_2 max) or high (H = 70% VO_2 max) intensity. (b) Effects on T_3 (mean ± SE). Suppression of T_3 was associated with energy availability and not with energy intake, exercise energy expenditure, or exercise intensity.

into the blood from glycogen stores elsewhere in the body, that reduce the oxidation of glucose for energy by peripheral tissues, and that stimulate appetite to increase the absorption of glucose into the blood from the gut.

By contrast, skeletal muscle accounts for 50% of body weight and stores an enormous amount of glucose as glycogen (about 300–750 g). However, skeletal muscle lacks the enzyme glucose-6-phosphatase that is necessary for returning that glucose to the bloodstream. The amounts of pyruvate, lactate, and alanine that are released from skeletal muscle during exercise yield no more than a few grams of glucose by hepatic gluconeogenesis (Ahlborg et al., 1974). Furthermore, these gluconeogenic precursors are derived in part from skeletal muscle glucose uptake rather than glycogenolysis. In addition, hepatic gluconeogenesis consumes a third of the energy content of the glucose molecule that is produced. As a result, almost none of the energy stored as glycogen in skeletal muscle is available to the brain. The only other substantial store of glycogen (about 80–120 g) is in the liver, which does contain glucose-6-phosphatase. Consequently, liver glycogen is the principal store of glucose available to the brain, but it is only a 1-day supply and half of that is depleted during the overnight fast. This is why we need to replenish our liver glycogen stores every day.

Meanwhile, in addition to depriving the brain of its own glycogen stores, working skeletal muscle competes aggressively against the brain for glucose circulating in the blood and stored in the liver. For example, working skeletal muscle oxidizes as much glucose during a marathon race as the brain will need for the next week—and when the race is over, the recovering skeletal muscle takes hundreds of additional grams of glucose to replenish its glycogen stores. This is why athletes in endurance sports need especially large quantities of carbohydrate in their diets.

Indeed, liver glycogen stores establish a milestone in the duration of energy deficiency. Deficiency persisting longer than the time required to deplete liver glycogen stores can be regarded as chronic, while more transitory deficiencies are regarded as acute.

Effects of Chronic Low Energy Availability

Figure 5.5a shows the dose-dependent effects of energy availability on plasma glucose during the feeding phase of the day in women who had expended EEE = 15 kcal/kgFFM/day (63 kJ/kgFFM/day) by walking at 70% VO_2 max on a treadmill (a workload similar to a 7-mile run) each day for 5 days (Loucks & Thuma, 2003). Meanwhile, their intake of a typical Western mixture of macronutrients (55% carbohydrates, 30% fats, and 15% protein) had been controlled to set their EA at 45 kcal/kgFFM/day (188 kJ/kgFFM/day) in one trial and at either 10, 20, or 30 kcal/kgFFM/day (42, 84, or 125 kJ/kgFFM/day) in another trial. The resulting effects would have been somewhat different with other combinations of macronutrients and with other intensities of exercise (at which working skeletal muscle would have utilized other combinations of metabolic fuels), but the general trend would have been similar.

The data in Figure 5.5a (and in Figures 5.7, 5.8, 5.9, and 5.12) are represented as percentages of the mean level at 45 kcal/kgFFM/day (188 kJ/kgFFM/day). They show that as energy availability was reduced to 20 kcal/kgFFM/day (84 kJ/kgFFM/day), effects on plasma glucose were minimized by multiple neuroendocrine glucoregulatory mechanisms, some of which are described below. When EA was further reduced to 10 kcal/kgFFM/day (42 kJ/kgFFM/day), however, carbohydrate intake was reduced to ~155 g/day, of which skeletal muscle oxidized ~85 g/day during exercise. This left a carbohydrate availability of only ~70 g/day. Within 5 days, liver glycogen stores became depleted and the body's glucoregulatory mechanisms were unable to maintain normal plasma glucose levels.

Figure 5.5b shows observational data on plasma glucose during the feeding phase of the day in amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS) (Laughlin & Yen, 1996). For comparison to Figure 5.5a, the data in Figure 5.5b (and in Figures 5.7, 5.8, 5.9, and 5.12) have been normalized to the level in ES. The low plasma glucose level in AmA is indicative of chronic low energy availability, and more specifically low carbohydrate availability.

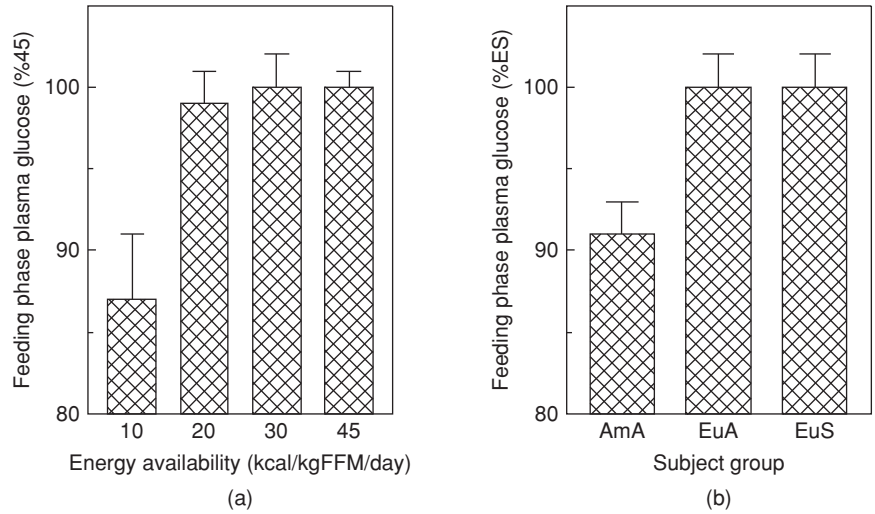

Figure 5.5 Plasma glucose levels (mean ± SE) during the feeding phase of the day. (a) In exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at EA = 45 kcal/kgFFM/day. Below EA = 20 kcal/kgFFM/day, glucoregulatory mechanisms failed to maintain normal plasma glucose levels. (b) In amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS). Data are normalized to levels in EuS. Low plasma glucose levels in AmA suggest low EA.

An important mechanism that helps to maintain plasma glucose levels during prolonged energy deficiency is a shift in the selection of metabolic fuels by working skeletal muscle. Reducing EA from 45 to 10 kcal/kgFFM/day (188–42 kJ/kgFFM/day) caused the respiratory quotient during exercise to decline from 0.91 to 0.85, reflecting a reduction from 71% to 51% in the amount of energy derived by working skeletal muscle from the oxidation of carbohydrates (Loucks & Thuma, 2003). This shift from glucose to fatty acid oxidation increased the availability of glucose to the brain by ~34 g/day. Thus, energy deficiency, like a high fat diet, shifts working skeletal muscle from glucose to fat oxidation.

Another important mechanism helping to maintain plasma glucose levels during prolonged energy deficiency is ketogenesis. The mobilization of fatty acids from adipose tissue increases their uptake by the liver as well as skeletal muscle. As EA declines, the β-oxidation of fatty acids in the liver produces acetyl-CoA faster than the TCA cycle can accept it. The excess is converted to ketone bodies (primarily β-hydroxybutyrate and acetoacetate) and released into the blood. Unlike fatty acids, ketone bodies can cross the blood–brain barrier and be oxidized by the brain for energy. Indeed, the uptakes of glucose and ketone bodies by the human brain are proportional to their concentrations in the blood (Hasselbalch et al., 1995). Figure 5.6 shows the ratio of fasting plasma β-hydroxybutyrate, the most abundant ketone body, to fasting plasma glucose in exercising women as EA was experimentally reduced from 45 to 10 kcal/kgFFM/day (188 to 42 kJ/kgFFM/day) for 5 days (Loucks & Thuma, 2003). As EA was reduced, the increasing availability of ketone bodies substantially reduced the brain's need for plasma glucose. (Corresponding data on female athletes are not available.)

A third mechanism helping to maintain plasma glucose levels during prolonged energy deficiency is hepatic gluconeogenesis from non-carbohydrate carbon substrates, i.e., the amino acids obtained from the breakdown of skeletal muscle and other body proteins and the glycerol obtained from the breakdown of triglycerides. Elevated levels of cortisol are necessary but not sufficient for accelerating muscle protein breakdown. In working skeletal muscle, protein damage, the release of Ca^{2+} into the cytosol, and elevated concentrations of reactive

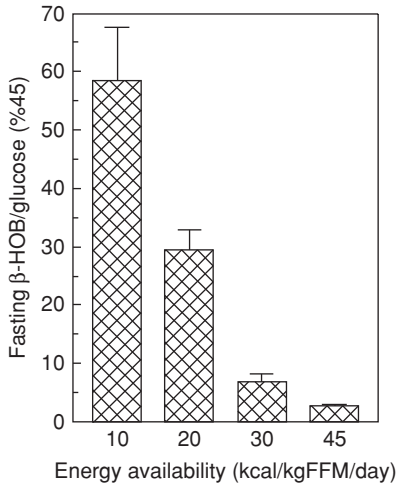

Figure 5.6 Ketogenesis at low energy availability. Ratio of fasting plasma β-hydroxybutyrate (β-HOB) to fasting plasma glucose (mean ± SE) in exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at 45 kcal/kgFFM/day. As EA declined, levels of plasma β-HOB approached those of plasma glucose.

oxygen species from incompletely oxidized metabolic fuels all activate proteases that make myofibrillar proteins available for ubiquitination (Loucks, 2012). So, too, do low insulin levels (Mitch et al., 1999). Thus, the cortisol/insulin ratio is an index of proteolytic drive. Energy deficiency accelerates proteolysis by increasing the cortisol/insulin ratio. Figure 5.7a shows cortisol/insulin ratios during the feeding phase of the day in exercising women as EA was reduced from 45 to 10 kcal/kgFFM/day (188 to 42 kJ/kgFFM/day) for 5 days (Loucks & Thuma, 2003). The observational data in Figure 5.7b show cortisol/insulin ratios during the feeding phase of the day in AmA, EuA, and EuS (Laughlin & Yen, 1996). The elevated cortisol/insulin ratios in both groups of athletes are evidence of accelerated proteolysis.

Insulin and growth hormone (GH), along with the catecholamines, regulate the cycling of fatty acids in adipose tissue. Briefly, insulin inhibits and GH stimulates lipolysis so that the GH/insulin ratio is an index of lipolytic drive. Energy deficiency accelerates lipolysis by increasing the GH/insulin ratio.

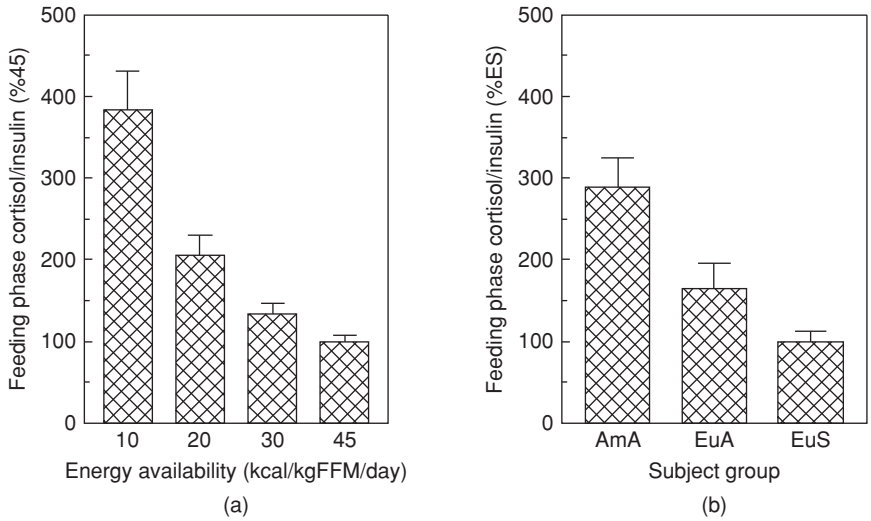

Figure 5.7 The ratio of cortisol to insulin (mean ± SE) as an index of proteolytic drive. (a) In exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at EA = 45 kcal/kgFFM/day. (b) In amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS). Data are normalized to levels in EuS. Elevated ratios in the athletes suggest elevated rates of proteolysis for supporting plasma glucose levels through gluconeogenesis.

Figure 5.8a shows 24-hour mean GH/insulin ratios in exercising women as EA was reduced from 45 to 10 kcal/kgFFM/day (188 to 42 kJ/kgFFM/day) for 5 days (Loucks & Thuma, 2003). Figure 5.8b shows observational data on 24-hour mean GH/insulin ratios in AmA, EuA, and EuS (Laughlin & Yen, 1996). The elevated GH/insulin ratios in both groups of athletes are evidence of accelerated lipolysis.

Chronic glucose deficiency also lowers thyroid hormone levels. T_3 stimulates mitochondrial biogenesis and ATP production in mitochondria and has profound effects on skeletal muscle protein turnover, fiber type expression, and Ca^{2+} uptake. Through these mechanisms, low T_3 levels reduce the ability of skeletal muscle to produce mechanical work and power (Loucks, 2012). Figure 5.9a shows T_3 levels in exercising women as EA was reduced from 45 to 10 kcal/kgFFM/day (188 to 42 kJ/kgFFM/day) (Loucks & Thuma, 2003). Figure 5.9b compares observational data on T_3 levels in AmA, EuA, and EuS (Loucks et al., 1992). Low T_3 levels in the athletes suggest that their muscular endurance, strength, and power may be impaired by energy deficiency.

Effects of Acute Low Energy Availability

Energy availability can also fluctuate widely within an hour. Figure 5.10a shows the 24-hour rhythm of insulin in eight men and women who ate three 500 kcal (2.1 MJ) meals at 6-hour intervals (8 a.m., 2 p.m., and 8 p.m.). Figure 5.10b shows the associated cortisol/insulin ratios. The rate of exercise-stimulated protein synthesis varies with the availability of amino acids in the blood, but only when insulin concentrations are above ~80 pM. Below that level, the ability of exercise to stimulate protein synthesis is impaired by insulin deficiency (Fedele et al., 2000). In Figure 5.10a insulin levels fell below ~80 pM about 4 hours after a meal. Similarly, insulin has little effect on proteolysis at rest when its concentrations are above ~150 pM, but below that level proteolysis accelerates as insulin levels decline (Louard et al., 1992). In Figure 5.10a, the concentration of insulin is >150 pM for only ~1 hour beginning ~40 minutes after each meal. This is why resistance exercise is more effective at stimulating muscle growth when it is performed shortly after a meal, and why athletes should eat snacks between meals.

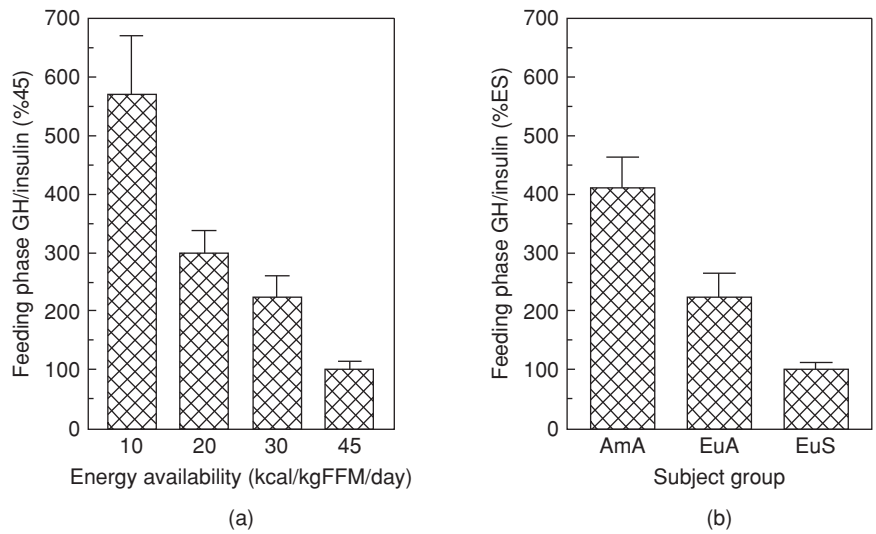

Figure 5.8 The ratio of growth hormone (GH) to insulin (mean ± SE) as an index of lipolytic drive. (a) In exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at EA = 45 kcal/kgFFM/day. (b) In amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS). Data are normalized to levels in EuS. Elevated ratios in the athletes suggest elevated rates of lipolysis for supporting plasma glucose levels by shifting metabolic fuel utilization from glucose to fatty acids.

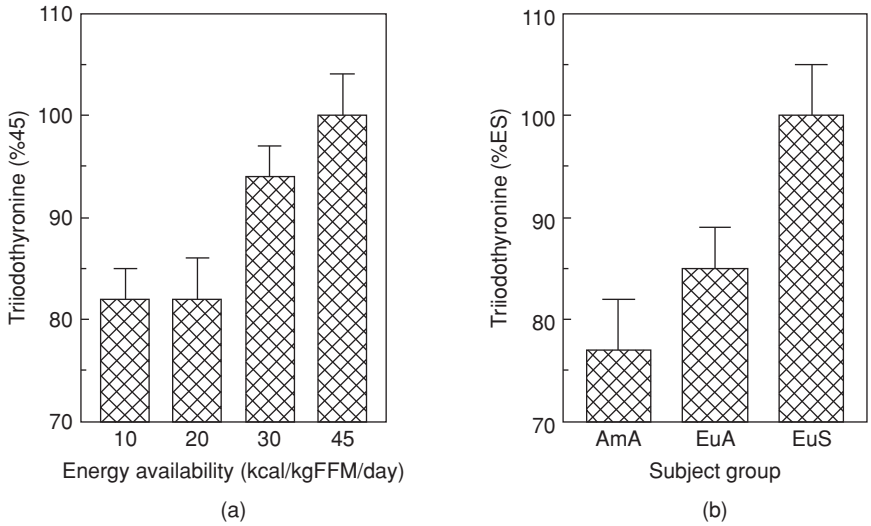

Figure 5.9 Triiodothyronine (T_3) levels (mean ± SE). (a) In exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at EA = 45 kcal/kgFFM/day. (b) In amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS). Data are normalized to levels in EuS. Low T_3 levels in the athletes suggest a generalized reduction in the rates of energy-consuming processes.

Glucoregulatory hormone concentrations change during a prolonged exercise bout, as well. Figure 5.11 shows data on the cortisol/insulin ratio with and without an intravenous infusion of glucose during 2 hours of exercise at 70% peak VO_2 under early morning postabsorptive conditions (MacLaren et al., 1999). Initially, the cortisol/insulin ratio was similar to the peak postabsorptive values in Figure 5.10b. Without the glucose infusion, the ratio rose even higher to more than 30,000. With glucose infusion, the ratio declined to 2000, similar to the levels shortly after meals in Figure 5.10b. Thus, the rise in the

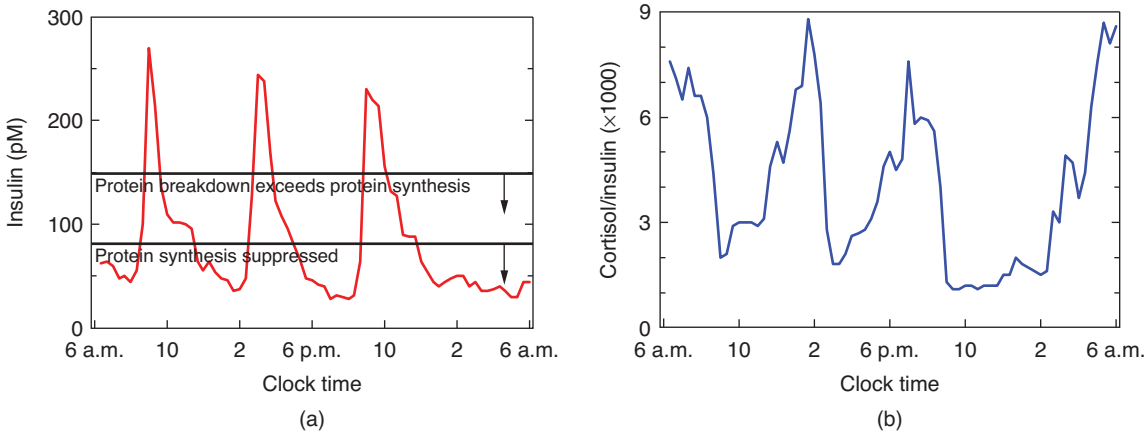

Figure 5.10 Acute effects of meals on insulin and cortisol in eight men and women consuming three 500 kcal meals at 6-hour intervals (8 a.m., 2 p.m., and 8 p.m.). (a) Insulin levels only exceeded 150 pM for ~1 hour and 80 pM for ~4 hours. (b) The ratio of cortisol to insulin. This eating style creates narrow windows of opportunity for optimum responses to resistance exercise.

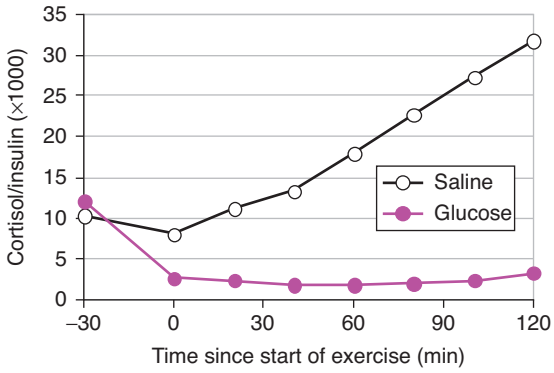

Figure 5.11 Cortisol to insulin ratios in eight men during 2 hours of exercise in the postabsorptive state at 70% VO_2 max with glucose and saline infusions. High and rising ratios with saline infusion suggest elevated rates of proteolysis for supporting plasma glucose levels through gluconeogenesis. Glucose infusion prevented this.

cortisol/insulin ratio during prolonged exercise is caused not by exercise, but rather by the failure to eat during exercise.

Athletes participating in sports that emphasize leanness display an increased incidence of upper respiratory tract infections caused by viruses (Hagmar et al., 2008). Our defense against viruses is type 1 immunity. Prolonged exercise has been reported to suppress type 1 immunity by a mechanism mediated, in part, by the rise in cortisol during prolonged exercise. In an experiment involving the expenditure of 2200 kcal (9.2 MJ) of energy during 2.5 hours of exercise at 65% VO_2 max, participants consumed a carbohydrate beverage providing just 23% of that energy in one trial and a placebo beverage in another. The carbohydrate beverage reversed the sign of the cortisol response (–33% vs. +32%) and attenuated the suppression of type 1 immune parameters by an average of 65% (Lancaster et al., 2005). The conclusion to be drawn from these few examples is that athletes need to manage acute as well as chronic energy availability to protect their health, promote their growth, and optimize their performance.

The Low Energy Availability Threshold

Some harmful effects of chronic energy deficiency are evident in the female athlete triad. Ovarian function depends critically upon the frequency with which the pituitary gland secretes pulses of luteinizing hormone (LH) into the blood. This frequency, in turn, depends upon the frequency with which pulses of gonadotropin-releasing hormone (GnRH) are secreted by certain neurons in the hypothalamus of the brain into a portal system of vessels that carries GnRH to the pituitary. GnRH pulse frequency is regulated by several hormonal and neural signals carrying information about energy availability to the brain.

Figure 5.12a shows LH pulse frequency in exercising women as EA was reduced from 45 to 10 kcal/kgFFM/day (188 to 42 kJ/kgFFM/day) (Loucks & Thuma, 2003). The observational data in Figure 5.12b compare LH pulse frequencies reported in two studies of AmA, EuA, and EuS (Laughlin & Yen, 1996; Loucks et al., 1989). Figure 5.12a shows that LH pulse frequency is robust against reductions of EA to 30 kcal/kgFFM/day (125 kJ/kgFFM/day), but declines as EA is reduced further. Figure 5.12b shows low LH pulse frequencies in both AmA and EuA, with a more extreme reduction in AmA. Although EuA menstruated very regularly, their luteal function after ovulation was found to be suppressed and abbreviated. This imperceptible subclinical menstrual disorder can cause infertility by preventing the implantation of a fertilized egg in the uterus. The 3-month incidence of such subclinical menstrual disorders among eumenorrheic runners has been found to be 78% (De Souza et al., 1998).

The threshold of EA below which LH pulse frequency was suppressed in exercising women (30 kcal/kgFFM/day = 125 kJ/kgFFM/day) corresponds closely to measurements of resting metabolic rate in male and female athletes (for a citation list, see Nattiv et al., 2007). In Figure 5.13, this EA threshold is plotted as a line through the origin. Also plotted in Figure 5.13 is the regression line relating sleeping metabolic rate (SMR) measured by indirect calorimetry to FFM in young adult men and women: SMR = 2.270 + 0.091 MJ/kgFFM/day × FFM (i.e., 540 kcal/day + 22 kcal/kgFFM/day × FFM) (Westerterp, 2003). Subthreshold EA provides most women with less energy than they need just to sleep. The proximity of the two lines in the vicinity of the data in Figure 5.13 also suggests that the SMR regression line may provide a more quantitatively accurate definition of the EA threshold over a wider range of FFM.

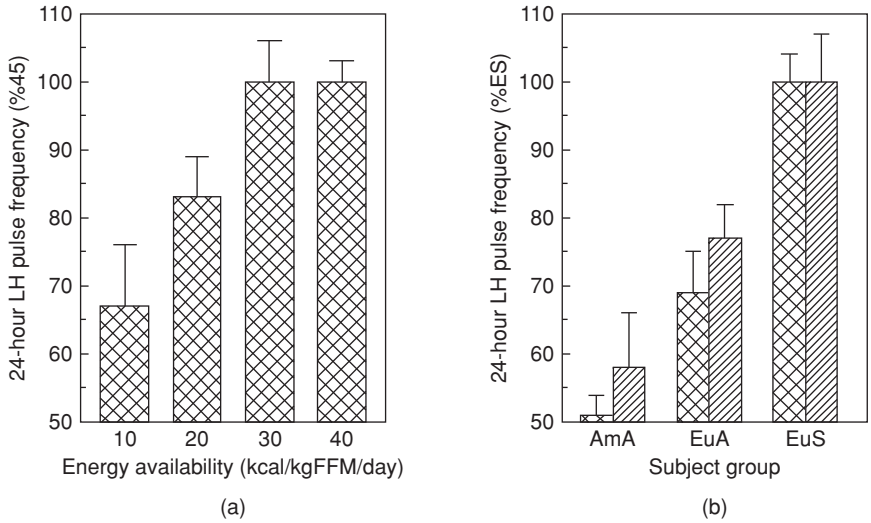

Figure 5.12 Luteinizing hormone (LH) pulse frequency (mean ± SE) over 24 hours. (a) In exercising women after 5 days at energy availabilities (EA) of 45, 30, 20, and 10 kcal/kgFFM/day (i.e., 188, 125, 84, and 42 kJ/kgFFM/day). Data are normalized to levels at EA = 45 kcal/kgFFM/day. Below EA = 30 kcal/kgFFM/day, LH pulse frequency declined. (b) In amenorrheic athletes (AmA), eumenorrheic athletes (EuA), and eumenorrheic sedentary women (EuS). Data are normalized to levels in EuS. Low LH pulse frequencies in athletes mediate their clinical (AmA) and subclinical (EuA) menstrual disorders. ⊠, Laughlin and Yen (1996) and Loucks et al. (1989); ▨, Laughlin and Yen (1996).

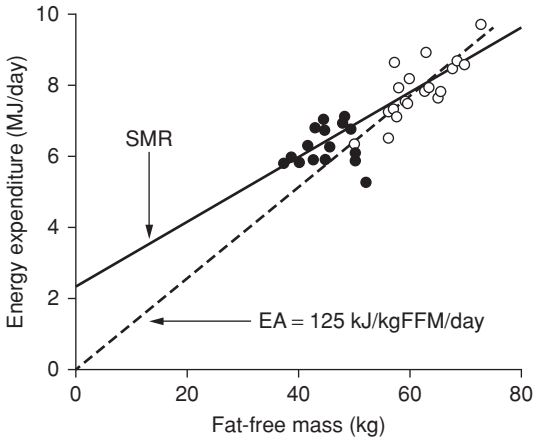

Figure 5.13 The low energy availability (EA) threshold. The dashed line is EA = 125 kJ/kgFFM/day = 30 kcal/kgFFM/day). The solid line is the regression of sleeping metabolic rate (SMR) on fat-free mass (FFM): SMR = 2.27 MJ/day + 0.091 kJ/kgFFM/day × FFM = 540 kcal/day + 22 kcal/kgFFM/day × FFM. Closed circles = females; open circles = males. EA <125 kJ/kgFFM/day provides most women with less energy than they need during sleep.

Conceptually, one would expect the same low energy availability threshold to apply to other basic physiological functions as well. For example, thermoregulation depends in part on the degree to which the respiratory chain in the inner wall of the mitochondrion in skeletal muscle is coupled to ATP formation. That coupling is strongly dependent on T_3 (Moreno et al., 2003). When T_3 is suppressed by low glucose availability, energy expenditure declines and cold tolerance is impaired despite high fatty acid availability.

Nevertheless, this low energy availability threshold should not be overinterpreted as applying to every physiological process. Nor should all physiological processes be assumed to exhibit threshold behavior. It seems likely that growth also depends not on the intake or expenditure but rather on the availability of energy after exercise training, but the optimum level of energy availability may be >45 kcal/kgFFM/day (>188 kJ/kgFFM/day). Glycogen replenishment and carbohydrate loading also likely depend on the availability of carbohydrate, defined as dietary carbohydrate intake minus carbohydrate oxidation during

exercise. High carbohydrate intake raises concern about *de novo* lipogenesis, but this is negligible in the liver (Aarsland et al., 1997; Hellerstein et al., 1991) and inefficient in adipose tissue (Macdonald, 1999) until glycogen storage capacity has been saturated (Acheson et al., 1988). Thus, there may be a threshold of high carbohydrate availability below which *de novo* lipogenesis can be avoided.

Causes of Low Energy Availability

Effective treatment of an athlete's low energy availability requires knowledge of the origin of the low energy availability. Low energy availability appears to derive from four different origins. Some athletes reduce energy availability intentionally in a rational but misguided pursuit of their desired body size, body composition, and mix of energy stores. Objectives of athletic training may be complex, such as reducing fat mass while increasing muscle mass and maximizing glycogen stores and also improving motor skills, speed, and endurance. Achieving such complex objectives requires disciplined adherence to a sophisticated and shifting diet and exercise regimen. Early encouraging results of a moderate reduction in energy availability may induce impatient athletes to reduce energy availability below 30 kcal/kgFFM/day (125 kJ/kgFFM/day). This may be followed by discouraging results, but athletes and coaches may interpret these disappointments as evidence that even more extreme reductions are needed to achieve further improvements. These extreme reductions may require athletes to adopt disordered eating behaviors, such as skipping meals, vomiting, and using laxatives. Nevertheless, for athletes whose low energy availability is merely the effect of unwise advice, professional guidance about nutrition, intermediate and ultimate goals, schedules, and techniques may be sufficient to correct unhealthy diet and exercise behavior.

In other athletes, low energy availability originates in an eating disorder, in particular anorexia nervosa. Anorexia nervosa is a serious clinical mental illness, usually accompanied by other mental illnesses (for a review, see Chapter 42, and see also Klump et al., 2009). In addition to nutritional counseling, anorexia nervosa requires psychiatric treatment, sometimes unwilling inpatient treatment with forced feeding (Carney et al., 2008), because anorexia nervosa has one of the highest risks of premature death of any mental illness (Harris & Barraclough, 1998). Diagnosed by current DSM-IV criteria, anorexia nervosa has a standardized mortality ratio of ~10 compared to age- and sex-matched peers (Birmingham et al., 2005). Sixty percent of deaths in anorexia nervosa are due to natural causes (i.e., to the medical consequences of the disease), for which the mortality risk is increased four times (Harris & Barraclough, 1998). The risk of death due to unnatural causes, (i.e., accident, misadventure, homicide, and suicide) is increased 11 times, and the specific risk of suicide is increased 32 times (Harris & Barraclough, 1998). From the date of diagnosis with anorexia nervosa, a patient's life expectancy is reduced by more than 20 years (Harbottle et al., 2008).

Because the mortality of anorexia nervosa is so high, sports organizations need to institutionalize procedures that identify undernourished athletes who may have anorexia nervosa and refer them for psychiatric evaluation and care. Those who encounter undernourished athletes (i.e., coaches, trainers, nutritionists, parents, and physicians in family practice, orthopedics, and sports medicine) may have difficulty distinguishing athletes with anorexia nervosa from athletes who are merely overly zealous in their rational efforts to lose weight, because both groups may practice disordered eating behaviors. Athletes with anorexia nervosa will not comply with professional recommendations to modify their behavior.

The third origin of low energy availability in athletes is the suppression of appetite. Over the past 15 years, many studies have found appetite to be suppressed by prolonged exercise (for reviews, see Loucks, 2004; Loucks et al., 2011). Yet, appetite remains a largely neglected topic in the field of sports nutrition. Indeed, the word "appetite" appears only once in the joint position stand of the American Dietetic Association, the Dietitians of Canada, and the American College of Sports Medicine on nutrition and athletic performance (Rodriguez et al., 2009).

Briefly, food deprivation increases hunger, but the same energy deficit produced by exercise does not. Appetite suppression by prolonged exercise has been demonstrated in controlled experiments with

protocols ranging from a few hours to 12 weeks. The effect is mediated by appetite-regulating hormones. The orexigenic (hunger) hormone ghrelin induces us to begin eating, and several anorexigenic (satiety) hormones (including peptide YY, glucagon-like peptide 1, and pancreatic polypeptide) induce us to stop eating. Exercise does not raise ghrelin concentrations but does raise the concentrations of anorexigenic hormones. As a result, *ad libitum* energy intake is reduced relative to energy expenditure. In prolonged 12-week experiments, some participants did gradually develop ghrelin responses to exercise and increases in hunger and *ad libitum* energy intake that prevented weight and fat loss, but others did not. For those who did not, "there is no strong biological imperative to match energy intake to activity-induced energy expenditure" (Blundell & King, 1999).

Athletes in endurance sports often eat diets containing high percentages of carbohydrates, because the consumption of high carbohydrate diets for a few days before a performance test has been shown to delay fatigue. Such carbohydrate loading is useful and appropriate as a pre-race tactic, but not as a lifestyle. The suppression of appetite by diets containing high percentages of carbohydrates has been demonstrated in experimental protocols ranging from a week (Stubbs et al., 2004) to a month (Horvath et al., 2000). As the percentage of carbohydrates in the diet was reduced, *ad libitum* energy intake spontaneously increased. Moreover, as the percentage of carbohydrates in the diet was reduced from 67% to 55%, the resulting increase in *ad libitum* energy intake preserved the actual amount of carbohydrate consumed. The mechanism of this effect has not yet been identified, but may involve the greater bulk and fiber content of carbohydrate-rich foods.

The appetite-suppressive effects of prolonged exercise and diets with high percentages of carbohydrates are large and additive (Stubbs et al., 2004), so that together they can reduce energy availability below 30 kcal/kgFFM/day (125 kJ/kgFFM/day). Consequently, appetite is not a reliable indicator of energy requirements for endurance athletes. To avoid unintentional low energy availability, athletes in endurance sports need to eat by discipline (i.e., planned amounts of selected foods at scheduled times) instead of appetite. However, the large amount of energy expended in endurance training greatly increases the amount of food required to achieve any target energy availability. For some athletes, the will to eat may limit the amount of exercise they can do at their target energy availability.

The fourth origin of low energy availability among female athletes is unrelated to sport. Around the world, about twice as many young women as young men at every decile of body mass index perceive themselves to be overweight (Wardle et al., 2006). The numbers actively trying to lose weight are even more disproportionate, and this disproportion *increases* as BMI declines, so that almost nine times as many lean women as lean men are actively trying to lose weight (Wardle et al., 2006). Indeed, more young female athletes report improvement of appearance than improvement of performance as a reason for dieting (Martinsen et al., 2010). Thus, social issues unrelated to sport may need to be addressed to persuade female athletes to eat *beyond* their appetites.

Management of Energy Availability

General Procedure

The energy availability of athletes is managed in six steps: (1) the athlete's FFM is measured; (2) the athlete's daily EEE is measured; (3) a value of EA appropriate for the athlete's current training goal is chosen; (4) the necessary daily energy intake is calculated as EI = FFM × (EEE + EA); (5) meals and snacks providing EI are selected; and (6) the athlete consumes these meals by discipline, regardless of appetite. Appropriate ranges of EA for various athletic purposes are listed in Table 5.1.

Table 5.1 Appropriate ranges of energy availability for various athletic purposes

>45 kcal/kgFFM/day (>188 kJ/kgFFM/day)	Gain of body mass, muscle hypertrophy, carbohydrate loading
~45 kcal/kgFFM/day (188 kJ/kgFFM/day)	Maintenance of body size and mass; focus on skill development
30–45 kcal/kgFFM/day (125–188 kJ/kgFFM/day)	Loss of body mass or fat

New Measurement Techniques

In addition to traditional methods, new technologies for monitoring diet and exercise programs are developing faster than they can be validated by independent research. Scores of manufacturers now make several different types of devices in various price ranges for estimating energy expenditure (TEE_X, TEE_W). Care is needed to select devices that estimate energy expended rather than work done. Energy expenditure data can be downloaded from these devices to software on personal computers (e.g., TrainingPeaks WKO+) or uploaded to cloud-based websites (e.g., TrainingPeaks.com, basic service free). Smartphone apps have become available for scanning barcodes to download or upload detailed nutritional data on hundreds of thousands of commercially packaged foods (e.g., FoodScanner, TrainingPeaks Mobile). Such tools increase the convenience, if not the accuracy, with which athletes, coaches, and dieticians can obtain diet and exercise data to calculate energy availability. These technologies can be expected to advance and proliferate rapidly in the coming years.

A Cautionary Note about the Calculation of EEE

In the recommended definition of EA illustrated in Figure 5.2b, the energy expenditure specifically attributable to exercise is calculated as EEE = $TEE_X - TEE_W$. Not making this subtraction results in an alternative definition and quantitatively larger value of exercise energy expenditure (EEE*) and in an alternative definition and quantitatively lower value of energy availability (EA* = EI – EEE*), illustrated in Figure 5.14. The greater convenience of the latter definition warrants consideration of its limitations.

If exercise intensity is high and duration is not more than an hour or so, as illustrated in Figure 5.14a, the convenience of EEE* may outweigh its small positive bias (~2 kcal/kgFFM/day) for management purposes. Calculations of EA* for cross-country runners, for example, could still be compared to EA = 30 kcal/kgFFM/day × FFM or to SMR to assess qualitatively whether their energy availability was dangerously low. On the other hand, if exercise intensity is low or intermittent and continues for several hours (H) as illustrated in Figure 5.14b, then the convenience of EEE* would

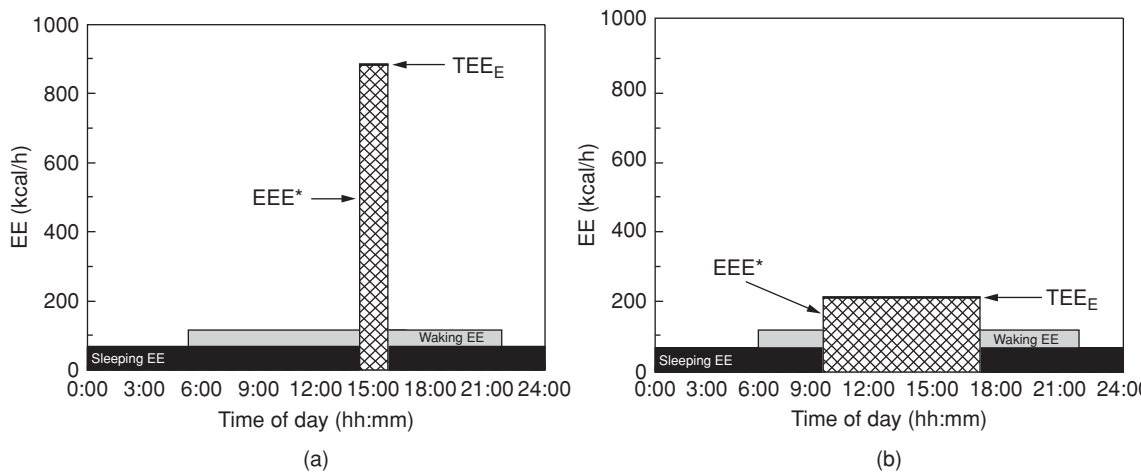

Figure 5.14 Estimation of exercise energy expenditure (EEE*) without adjustment for waking energy expenditure (TEE_W). (a) When exercise intensity is high and duration is short, EEE* = TEE_E is biased slightly high and the resulting EA* = EI – EEE* is biased slightly low compared with EEE and EA in Figure 2b. For qualitative comparison to EA = 30 kcal/kgFFM/day, the errors are negligible. (b) When exercise intensity is low or intermittent and duration is long, EEE* = TEE_E is biased very high and the resulting EA* = EI – EEE* is biased very low compared with EEE and EA in Figure 2b. EA* will lead to many false-positive assessments of low EA.

not outweigh the associated large positive bias (~2 × H kcal/kgFFM/day). It would not be appropriate, for example, to assess whether the energy availability of dancers who train for 8 hours per day was dangerously low by comparing their EA* to EA = 30 kcal/kgFFM/day × FFM or to SMR.

Summary

Conceptually, energy availability is the amount of dietary energy remaining after exercise training for all other physiological processes. As such, energy availability has several advantages over energy balance for managing coordinated diet and exercise programs to optimize the health and performance of athletes. One of these advantages is that the calculation of energy availability involves the estimation of exercise energy expenditure instead of total energy expenditure, which has a much greater measurement uncertainty. Dose–response research has identified a threshold of energy availability below which reproductive health is impaired. The same threshold may apply to other pathological conditions associated with energy deficiency, but other thresholds not yet identified may apply to pathological conditions associated with energy excess. Moreover, some physiological processes, such as protein synthesis, may vary linearly with energy availability.

Because of the uniquely high requirement of the human brain for glucose, apparent effects of low energy availability may actually be effects of low carbohydrate availability. Neuroendocrine responses to low energy availability mobilize metabolic fuels from storage depots throughout the body. The mobilization of amino acids for gluconeogenesis rather than for the resynthesis of new proteins may be counterproductive for athletic objectives, as may also be the depletion of liver glycogen stores.

A program to increase the energy availability of an energy-deficient athlete needs to address the specific cause(s) of the energy deficiency in that particular athlete. Sports management programs should institutionalize procedures to distinguish athletes whose low energy availability originates in an eating disorder requiring psychiatric treatment from those whose low energy availability has other origins. New technologies are becoming available to help athletes, coaches, and dieticians worldwide to monitor energy availability.

References

Aarsland, A., Chinkes, D., & Wolfe, R.R. (1997) Hepatic and whole-body fat synthesis in humans during carbohydrate overfeeding. *American Journal of Clinical Nutrition* 65, 1774–1782.

Acheson, K.J., Schutz, Y., Bessard, T., et al. (1988) Glycogen storage capacity and de novo lipogenesis during massive carbohydrate overfeeding in man. *American Journal of Clinical Nutrition* 48, 240–247.

Ahlborg, G., Felig, P., Hagenfeldt, L., et al. (1974) Substrate turnover during prolonged exercise in man: splanchnic and leg metabolism of glucose, free fatty acids and amino acids. *Journal of Clinical Investigation* 53, 1080–1090.

Birmingham, C.L., Su, J., Hlynsky, J.A., et al. (2005) The mortality rate from anorexia nervosa. *International Journal of Eating Disorders* 38, 143–146.

Blundell, J.E. & King, N.A. (1999) Physical activity and regulation of food intake: current evidence. *Medicine & Science in Sports & Exercise* 31, S573–S583.

Braun, B. & Brooks, G.A. (2008) Critical importance of controlling energy status to understand the effects of "exercise" on metabolism. *Exercise and Sport Sciences Reviews* 36, 2–4.

Carney, T., Tait, D., Richardson, A., et al. (2008) Why (and when) clinicians compel treatment of anorexia nervosa patients. *European Eating Disorders Review* 16, 199–206.

De Souza, M.J., Miller, B.E., Loucks, A.B., et al. (1998) High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *Journal of Clinical Endocrinology and Metabolism* 83, 4220–4232.

Fedele, M.J., Hernandez, J.M., Lang, C.H., et al. (2000) Severe diabetes prohibits elevations in muscle protein synthesis after acute resistance exercise in rats. *Journal of Applied Physiology* 88, 102–108.

Hagmar, M., Hirschberg, A.L., Berglund, L., et al. (2008) Special attention to the weight-control strategies employed by Olympic athletes striving for leanness is required. *Clinical Journal of Sports Medicine* 18, 5–9.

Harbottle, E.J., Birmingham, C.L., & Sayani, F. (2008) Anorexia nervosa: a survival analysis. *Eating and Weight Disorders* 13, e32–e34.

Harris, E.C. & Barraclough, B. (1998) Excess mortality of mental disorder. *British Journal of Psychiatry* 173, 11–53.

Hasselbalch, S.G., Knudsen, G.M., Jakobsen, J., et al. (1995) Blood–brain barrier permeability of glucose and ketone bodies during short-term starvation in humans. *American Journal of Physiology: Endocrinology and Metabolism* 268, E1161–E1166.

Hellerstein, M.K., Christiansen, M., Kaempfer, S., et al. (1991) Measurement of de novo hepatic lipogenesis in humans using stable isotopes.

Journal of Clinical Investigation 87, 1841–1852.

Horvath, P.J., Eagen, C.K., Ryer-Calvin, S.D., et al. (2000) The effects of varying dietary fat on the nutrient intake in male and female runners. *Journal of the American College of Nutrition* 19, 42–51.

Klump, K.L., Bulik, C.M., Kaye, W.H., et al. (2009) Academy for eating disorders position paper: eating disorders are serious mental illnesses. *International Journal of Eating Disorders* 42, 97–103.

Kono, T. (2001) *Weightlifting, Olympic Style*, pp. 194–197. Hawaii Kono Company, Oahu, HI.

Lancaster, G.I., Khan, Q., Drysdale, P.T., et al. (2005) Effect of prolonged exercise and carbohydrate ingestion on type 1 and type 2 T lymphocyte distribution and intracellular cytokine production in humans. *Journal of Applied Physiology* 98, 565–571.

Laughlin, G.A. & Yen, S.S.C. (1996) Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. *Journal of Clinical Endocrinology and Metabolism* 81, 4301–4309.

Louard, R.J., Fryburg, D.A., Gelfand, R.A., et al. (1992) Insulin sensitivity of protein and glucose metabolism in human forearm skeletal muscle. *Journal of Clinical Investigation* 90, 2348–2354.

Loucks, A.B. (2004) Energy balance and body composition in sports and exercise. *Journal of Sports Sciences* 22, 1–14.

Loucks, A. (2009) Is stress measured in joules? *Military Psychology* 21, S101–S107.

Loucks, A.B. (2012) The endocrine system: integrated influences on metabolism, growth, and reproduction. In *ACSM's Advanced Exercise Physiology* (eds. P.A. Farrell, M.J. Joyner, & V.J. Caiozzo), pp. 466–506. Wolters Kluwer, Philadelphia, PA.

Loucks, A.B. & Callister, R. (1993) Induction and prevention of low-T_3 syndrome in exercising women. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology* 264, R924–R930.

Loucks, A.B. & Thuma, J.R. (2003) Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *Journal of Clinical Endocrinology and Metabolism* 88, 297–311.

Loucks, A.B., Mortola, J.F., Girton, L., et al. (1989) Alterations in the hypothalamic-pituitary-ovarian and the hypothalamic-pituitary-adrenal axes in athletic women. *Journal of Clinical Endocrinology and Metabolism* 68, 402–411.

Loucks, A.B., Laughlin, G.A., Mortola, J.F., et al. (1992) Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. *Journal of Clinical Endocrinology and Metabolism* 75, 514–518.

Loucks, A.B., Verdun, M., & Heath, E.M. (1998) Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *Journal of Applied Physiology* 84, 37–46.

Loucks, A.B., Kiens, B., & Wright, H.H. (2011) Energy availability in athletes. *Journal of Sports Sciences* 29(Suppl. 1), S7–S15.

Macdonald, I.A. (1999) Carbohydrate as a nutrient in adults: range of acceptable intakes. *European Journal of Clinical Nutrition* 53(Suppl. 1), S101–S106.

MacLaren, D.P., Reilly, T., Campbell, I.T., et al. (1999) Hormonal and metabolic responses to maintained hyperglycemia during prolonged exercise. *Journal of Applied Physiology* 87, 124–131.

Martinsen, M., Bratland-Sanda, S., Eriksson, A.K., et al. (2010) Dieting to win or to be thin? A study of dieting and disordered eating among adolescent elite athletes and non-athlete controls. *British Journal of Sports Medicine* 44, 70–76.

Mitch, W.E., Bailey, J.L., Wang, X., et al. (1999) Evaluation of signals activating ubiquitin-proteasome proteolysis in a model of muscle wasting. *American Journal of Physiology: Cell Physiology* 276, C1132–C1138.

Moreno, M., Lombardi, A., De Lange, P., et al. (2003) Fasting, lipid metabolism, and triiodothyronine in rat gastrocnemius muscle: interrelated roles of uncoupling protein 3, mitochondrial thioesterase, and coenzyme Q. *FASEB Journal* 17, 1112–1114.

Nattiv, A., Loucks, A.B., Manore, M.M., et al. (2007) American College of Sports Medicine position stand: the female athlete triad. *Medicine & Science in Sports & Exercise* 39, 1867–1882.

Rodriguez, N.R., DiMarco, N.M., & Langley, S. (2009) Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *Journal of the American Dietetic Association* 109, 509–527.

Stubbs, R.J., Hughes, D.A., Johnstone, A.M., et al. (2004) Rate and extent of compensatory changes in energy intake and expenditure in response to altered exercise and diet composition in humans. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology* 286, R350–R358.

Wade, G.N. & Jones, J.E. (2004) Neuroendocrinology of nutritional infertility. *American Journal of Physiology: Regulatory, Integrative and Comparative Physiology* 287, R1277–R1296.

Wade, G.N. & Schneider, J.E. (1992) Metabolic fuels and reproduction in female mammals. *Neuroscience and Biobehavioral Reviews* 16, 235–272.

Wardle, J., Haase, A.M., & Steptoe, A. (2006) Body image and weight control in young adults: international comparisons in university students from 22 countries. *International Journal of Obesity (London)* 30, 644–651.

Westerterp, K. (2003) Energy metabolism and body composition: general principles. *European Respiratory Monograph* 24, 1–10.

Chapter 6

Assessing Body Composition

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Introduction

Body composition is an important health and athletic performance variable. Body fat may act as ballast in biomechanical terms, but adipose tissue is a vital endocrine organ in terms of general health. While the storage and metabolism of body fat have undoubtedly played a key role in our evolutionary past, the different performance and health imperatives have presented a conflict for athletes, for whom risks of eating disorders associated with aiming for extremes of body composition are exacerbated.

Quantification of total body fat content has often been the prime focus of attention, but many coaches and scientists working with elite athletes recognize that assessment of the amount and distribution of lean tissues like bone and muscle can be just as important in determining sports performance. Coaches may assume that increased muscle mass will enhance power production, thereby justifying resistance training for athletes. However, more is not necessarily better when it comes to strength and body bulk, as the athlete must bear the burden of additional weight in the form of muscle and its biomechanical consequences for movement.

In certain weight-sensitive sports, many athletes use extreme methods to reduce mass rapidly or maintain a low body mass in order to gain a competitive advantage (Ackland et al., 2012). Consequently, athletes with very low body mass, extreme mass changes due to dehydration or eating disorders, extremely low percent body fat (% fat), or insufficient bone mineral density are becoming common issues in many sports (Nattiv et al., 2007; Sundgot-Borgen & Torstveit, 2010). Contrary to what many coaches believed until recently, weight change is not simply a matter of gaining muscle and shedding pure fat, but involves a variety of tissues dependent upon initial body composition, rate of weight loss, and other factors (Hall, 2007).

Assessing human body composition has formed a central part of medical research for the best part of a century. While progress has been significant with the use of new and combined analytical methods, indisputable ethical and methodological limitations have prevented the identification of an absolute standard against which other methods can be compared. As a consequence, while accurate assessment of body fatness has been a major goal of body composition research over the past 50 years, much of the work to validate new and old methods is indirect. Despite considerable advances in methods, today there is still no gold standard for body fat assessment with accuracy better than 1% across a range of body types.

Together with the development of multicomponent assessment methods over the last three decades, we have also witnessed an increase in research on elite athletes from a whole range of sports. As training methods have become more sophisticated, each athletic group has become more specialized, modifying its typical physique imperatives away

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from general morphological norms. As a consequence, many of the assumptions on which some techniques rely are no longer valid for athletes. For example, elite athletes who had undergone resistance training were estimated to have –12% fat using densitometry (Adams et al., 1982) and negative fat on the torso using dual-energy X-ray absorptiometry (DXA) (Stewart & Hannan, 2000). At the same time, the practicalities of making measurements have become increasingly challenging as athletes may be reluctant to interrupt what for many is a full-time occupation for the sake of body composition assessment, thereby making the more time-consuming laboratory techniques less appealing. These factors all conspire against the scientist seeking to make accurate measurements on athletes, with the inevitable consequence that data may be misleading, misinterpreted, or perhaps used inappropriately. This reality has forced researchers to consider acceptable surrogate measures for fatness, such as a sum of skinfolds, without recourse to quantifying tissue mass.

The choice of body composition technique often depends on the intended purpose for which data are to be used, as well as the available technology. In regard to high-performance sport, assessment of body composition may define a performance or selection criterion, be used to assess the effectiveness of an exercise or dietary intervention, or be used to monitor the health status of an athlete. Individual body composition goals should be identified by trained health-care personnel (e.g., an athletic trainer, physiologist, nutritionist, or physician) and body composition data should be treated in the same manner as other personal and confidential medical information.

A Framework for Comparing Techniques

Though many assessment techniques exist for describing the constituent components of the body, in practice, the techniques in current use fall into reference, laboratory, and field method categories (Ackland et al., 2012), which include both the chemical (molecular) or anatomical (tissue/systems) approaches (Figure 6.1). Within these approaches,

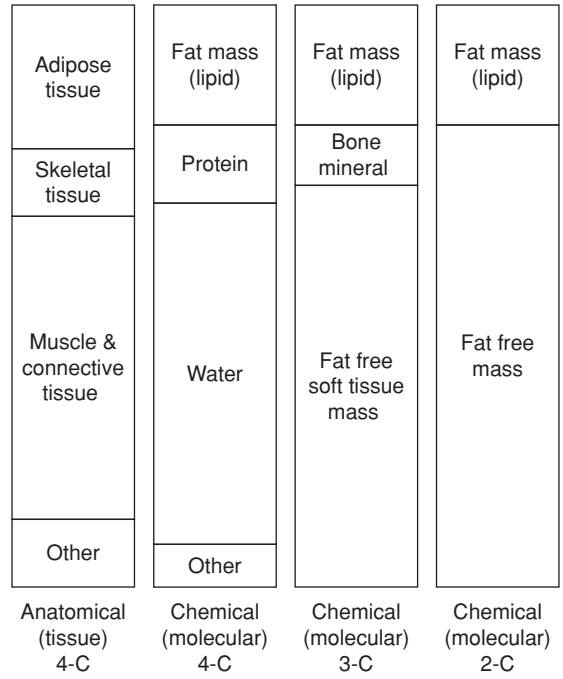

Figure 6.1 Anatomical and chemical body composition models. 4-C, 3-C, and 2-C refer to 4-, 3-, and 2-component models, respectively.

we must also understand that techniques can be categorized as being direct—for example, via cadaver dissection, indirect—where a surrogate parameter is measured to estimate tissue or molecular composition, or doubly indirect—where one indirect measure is used to predict another indirect measure (i.e., via regression equations). The use of regression equations to make predictions from composition measurements also means that these latter approaches are sample-specific.

In both the chemical and anatomical approaches, we may also employ multicomponent models (Figure 6.1). Thus, it has been common for authors to refer to 2-component models (fat mass (FM) and fat-free mass (FFM)), 3-component models (fat, bone mineral, and lean content), or 4-component models (adipose, bone, muscle, and other tissues).

A review of body composition methods must also consider the implications of techniques that merely sample the body as opposed to those which attempt to assess the whole body. Several commonly

employed methods (e.g., skinfolds) sample the subcutaneous adipose tissue (SAT) at standardized sites and assume that there is some fixed and direct relationship between this compartment and fat depots deep within the body. Furthermore, it is assumed in these methods that the standardized sites provide a representative estimate of the total SAT in the body.

Finally, mention must be made regarding individual versus group results. Some techniques that supposedly assess body composition (e.g., body mass index (BMI)) are often cited as being significantly correlated with important health indicators or values from other assessment procedures. Practitioners need to be mindful that demonstration of a strong association at the population level is not the same as a technique providing accurate, precise, and reliable body composition data for an individual.

Body Composition Assessment Techniques

The reference methods are, by definition, the most accurate techniques for assessing body composition and have often been employed as the criterion against which other techniques are compared. Nevertheless, these reference methods may have limited applicability for monitoring athletes. Limitations include feasibility (e.g., cadaver dissection), time and financial costs involved (e.g., MRI scanning), a lack of published normative data (e.g., multicomponent models), and unnecessary radiation exposure (e.g., CT scanning). There are also questions regarding sensitivity of some of the accepted reference methods. Since these methods are not commonly employed for the assessment of athletes, we will not consider them further in this chapter.

Laboratory Methods

Laboratory methods are often used for assessing body composition of athletes, but there exists wide variation in their accuracy and precision. So named, these methods generally require the athlete to attend a facility where the equipment is located. In this section, we will review the accuracy, reliability, and utility of DXA, densitometry, hydrometry (or body water), ultrasound (US), and 3D photonic scanning.

Dual-Energy X-Ray Absorptiometry DXA was originally developed as a scanning system for investigating bones from earlier photon absorptiometry methods that used gamma-emitting radioisotopes. When X-rays pass through an absorbing substance, they attenuate according to the atomic number of the atoms in their path and, using two energies, there is a steeper attenuation at the lower energy level. As a result, the ratio of the attenuation at the two energies (the *R* value) varies between tissues and can be used to measure tissue composition.

With the participant lying motionless on the scanning table wearing a hospital gown or sports clothing, the machine maps the mass and composition of each pixel in terms of bone mineral, fat, and fat-free soft tissue (Figure 6.2). Depending on the scanner type (fan or pencil beam), a whole-body scan can take between 4 and 15 minutes. For athletes, DXA measurement has several advantages over other reference and laboratory techniques, due to its speed and convenience and because the measurement is minimally influenced by fluctuations in body water content. Readers are referred to Pietrobelli et al. (1996) for a comprehensive summary on the principles of DXA.

With a low radiation dose (which varies according to the scanner type and beam configuration), this method is viewed as a laboratory reference method and contributes to the bone mineral assessment for multicomponent models. DXA's utility and widespread proliferation in current practice is due to the convenience of acquiring regional composition data without recourse to medical imaging techniques. However, we caution against using DXA on multiple occasions (perhaps no more than four times per annum), not only due to the cumulative radiation dose, but also due to the error of measurement which limits the ability to detect small body composition changes over time.

Perhaps the biggest methodological weakness is DXA's inability to measure soft tissue composition in the approximately 40% of pixels that contain bone in a typical healthy adult. Fat and fat-free soft tissue are predicted according to the gradient of differing

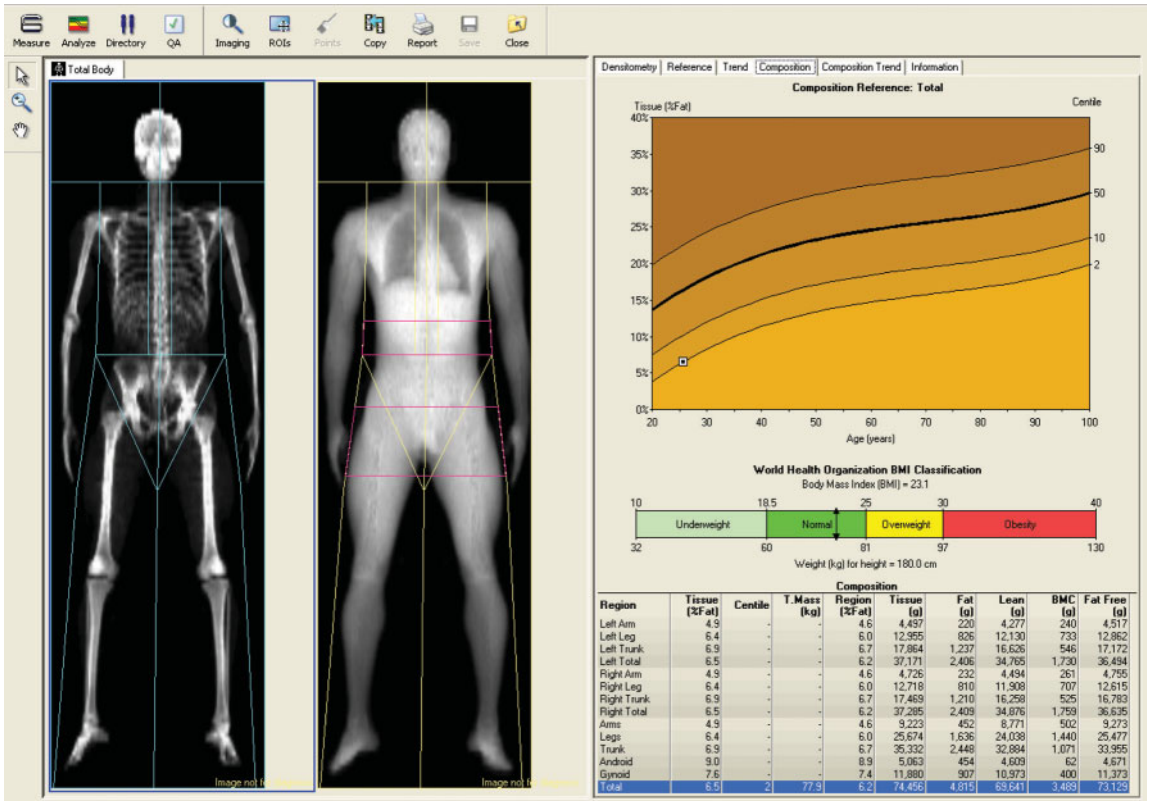

Figure 6.2 DXA (Lunar Prodigy) body composition analysis screen for a male athlete.

composition in "soft tissue shells" and are projected behind the bone "shadow" (Nord & Payne, 1995). Lean individuals usually have relatively fewer non-bone pixels from which this prediction can be made.

The size of individuals can also be a limitation in two ways. First, in larger individuals, the tissue depth is a factor attenuating the X-ray beam, and for those with chest depth greater than 25 cm, composition predictions can be unreliable (Jebb et al., 1993). Second, scanning tables are generally limited to 190 cm in length. In practice, the head and neck can be scanned separately from the torso and the data combined retrospectively (Prior et al., 1997). Newer scanners have wider scanning tables with heavier payloads and will accommodate greater tissue depths, but tall or heavy athletes may still require to be measured in sequential scans, which may be less accurate than a single whole-body scan.

DXA has been used to derive regional and total fat estimates which outperformed densitometry relative to a 4-component model (Prior et al., 1997). This has led some authors to use it as a reference method in preference to densitometry, yielding FM and FFM predictions from other indirect methods such as skinfolds. In a study of male athletes, standard error of the estimate (SEE) predicting DXA-derived FM from skinfolds was 1.7 kg, although the seven leanest athletes showed negative fat on the torso (Stewart & Hannan, 2000). The high muscle mass and low FM of these individuals appear to fall beyond the calibrated range. Of some considerable concern, then, is the ever-increasing access to DXA by commercial sports organizations that seek to measure incrementally lean and muscular individuals, meaning the scope for misinterpretation of data is also increasing.

In summary, DXA is a reasonably precise whole-body assessment method, but has some limitations in producing accurate soft tissue estimates for lean athletes. Using multicomponent models as a reference method to validate DXA, Lohman et al. (2000) reported an SEE of 2–3% for predicting % fat. Finally, despite efforts at cross-calibration between equipment manufacturers, to date, the results remain specific to each scanner type and software version.

Densitometry Densitometry or underwater weighing (UWW) involves measuring body mass and volume, then calculating their ratio to derive whole-body density. This method partitions the body into FM and FFM, assumes a constant density of each, then relates the measured whole-body density to a % fat (Siri, 1956). Assessment of body volume is generally made using either UWW or air displacement plethysmography (ADP). The major limitation of this technique is, however, that variations in water and bone mineral content of the FFM among populations and individuals affect its density (Going, 2005), thereby violating the central assumption of constant FFM tissue density, with profound consequences for estimating fatness.

UWW requires a participant (on a submersible seat suspended from an autopsy scale or load cell) to exhale maximally during submersion. Calculating body density relies on dividing body mass by the measured volume (the latter being derived by the apparent weight loss of the submerged body). Although less subject involvement is required using ADP, both methods require estimation of residual lung volume with additional equipment and expertise. In UWW, this is routinely performed using an oxygen dilution technique (Wilmore et al., 1980) and should be done while the participant is immersed to the neck in the water because of the influence of hydrostatic pressure on measured lung volumes.

ADP measures body volume in a sealed air capsule rather than under water. By comparison, ADP is rapid, does not require water confidence, and is suitable for a wider range of individuals. Currently, the available ADP technology is referred to as the BOD POD (Life Measurement Inc., Concord, CA, USA). In this system, a measuring chamber and a reference chamber (beneath the seat) are linked by a flexible airtight diaphragm which is perturbed to induce small pressure changes between both chambers (Figure 6.3). Using Poisson's law, the pressure–volume relationship at a fixed temperature is used to calculate the volume of the participant in the measuring chamber. After the system has been calibrated with a known volume, the participant is weighed wearing swimwear and cap and then occupies the measuring chamber for ~2 minutes for volumetric measurement. Breathing normally during measurement, the participant is then prompted to execute a breathing maneuver for residual gas calculation. Adjustments for thoracic gas volume and skin surface area are necessary because of the behavior of the air inside the chamber.

Despite its advantages over UWW, in participant acceptability and throughput, several

Figure 6.3 A volunteer undergoing the BOD POD measurement procedure.

methodological issues require consideration. Moisture on the skin or hair affects compressibility of air next to the body surface, leading to an underestimation of % fat. The behavior of air close to the skin surface is predicted by a surface area artifact, based on estimated body surface area, the accuracy of which may be of some concern. Clothing is also important with swimwear being recommended; wearing gym apparel reduces test–retest reliability and leads to an underestimation of fatness. Peeters and Claessens (2011) also demonstrated that a lycra cap compresses the hair less effectively than a silicon cap, not fully eliminating the effect of isothermal air trapped in scalp hair, which also results in an underestimation of fatness. While such issues of participant presentation are easily addressed, more problematic may be the location of the capsule, which requires a separate room, with closely regulated temperature and humidity. Changes in ambient pressure through windows or doors may cause the system to require regular recalibration.

Variations in % fat have been reported for gender between UWW and ADP. Compared with results from UWW, ADP underestimated % fat (in absolute terms) by 8% in lean female athletes (Vescovi et al., 2002), underestimated % body fat at lower fat values and overestimated at higher fat values in boys (Claros et al., 2005), and underpredicted % fat by an average 2% in male college football players (Collins et al., 1999). In summary, despite the popularity of densitometry techniques over many decades, both UWW and ADP techniques adopt the 2-component model that assumes density of FFM to be constant. This assumption is clearly violated in many groups of athletes. Therefore, caution is essential when interpreting body fat results from these methods, especially for lean athletes.

Hydrometry Among athletes, water is the single largest component of the body, typically accounting for 50–70% of the total mass. Different lean tissues generally have between 70% and 80% water, while adipose tissue has between 10% and 20% water (Bender & Bender, 1997; Institute of Medicine, 2005). Total body water can be used to estimate both FM and FFM, assuming a constant hydration of 72–73%, but variation in hydration levels among subjects is the main limitation of this method for athletes. The primary approach for body water measurement is the deuterium dilution method, which was well described by Schoeller (2005). Body water assessment relies on purchase of deuterium oxide (a stable isotope), expert measurement skills, and expensive laboratory equipment, so is not generally available for widespread body composition assessments.

An understanding of hydration status, though, has implications for all body composition assessment techniques. Although the rate of turnover of body water is typically about 2–3 l/day, it can be much higher than this, with sweat losses in excess of 3 l/h being sustained for relatively short periods during physical activity in hot environments. Daily fractional turnover can therefore reach 20–30% of total body water. Acute changes in body water may confound the use of standard methodologies for the assessment of body composition. All measures of body composition should therefore be made under standardized conditions of hydration status (e.g., after fasting, prior to an exercise bout, with an empty bladder).

Ultrasound US imaging is a relatively new technique for body composition assessment. Operating in brightness or "B-mode," US images can be assessed to determine the thickness of the SAT at various positions beneath the skin surface. The precision of SAT measurements was found to be excellent (Bellisari et al., 1993), while Ishida et al. (1992) found B-mode US to be a highly reliable method for the measurement of both fat and muscle thickness.

Considerable skill and anatomical knowledge may be needed to identify correctly the interfaces of some deep tissues, but the SAT is comparatively easy to find as it forms a continuous layer underneath the skin which is bounded by the muscle fascia at the deep margin. Horn and Müller (2010) compared SAT measurements in excised pig tissue using a semiautomatic US image evaluation procedure with vernier caliper measurements (0.01 mm resolution); the correlation was very high ($r = 0.998$; $n = 140$) and SEE was 0.21 mm. The regression coefficient was 0.98 when the standard sound velocity of 1540 m/s was used. In this technique, it is important for the investigator to control visually

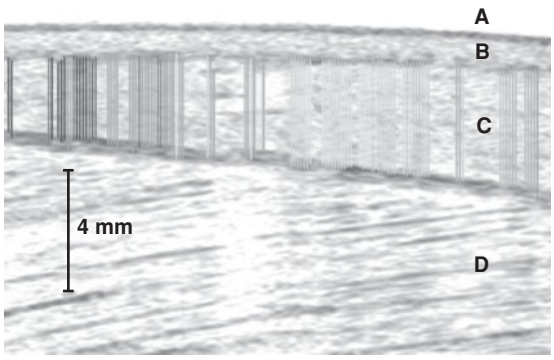

Figure 6.4 Semiautomatic ultrasound B-mode image evaluation. A, electrode gel; B, dermis; C, subcutaneous adipose tissue; D, muscle. Adapted from Ackland et al. (2012).

the output of automatic edge detection algorithms so as to prevent erroneous image interpretations. An example for SAT measurements using recently developed semiautomatic evaluation software is shown in Figure 6.4.

Many thickness measurements can be obtained from a single US image, resulting in a very low standard error of the mean. Furthermore, many measurements can easily be made at a given site where SAT thickness varies greatly and mean values can be used instead of single point measurements. US is well suited to analyze fat patterning, and total SAT may be determined by combining a series of US measurements with body surface area measurement techniques like three-dimensional (3D) laser scanning. In summary, it can be expected that US measurements of adipose, muscle, and other tissue thicknesses will become an important body composition assessment technique because of the high measurement accuracy. Future studies are needed, however, to derive standard reference ranges applicable to controls and athletes, once measurement sites and protocols are established.

3D Photonic Scanning 3D photonic scanning enables fast and accurate external profiling of the body. Its development over the past two decades for the clothing and automotive industries has included approaches using class 1 (eye-safe) lasers, structured light, and millimeter-wave technologies. Scanners measure the participant in form-fitting clothing and take approximately 10 seconds to capture the whole-body shape, with outputs including linear and curved distances, cross-sectional areas, surface areas, and segment volumes (Figure 6.5).

3D scanning has been validated for assessing body volume and subsequent % fat prediction following appropriate accounting for lung volumes (Wang et al., 2006), although this includes the same assumptions for FM and FFM as ADP regarding the 2-component model. The requirement for participants to wear form-fitting clothing, which can be a severe limitation in obesity or body image research, is of little impediment for monitoring athletes, many of whom are required to wear such clothing in training and competition (Schranz et al., 2010).

In summary, this novel methodology is not routinely used to quantify fatness, but is a potentially

Figure 6.5 A volunteer preparing for 3D whole-body scanning.

useful adjunct to existing techniques, which may be important for athlete monitoring. Despite its limitations, the rapid profiling capability enables great numbers of athletes to be surveyed and its use, in combination with other measurement modalities such as US and DXA, will undoubtedly represent a major advance in future body composition research.

Field Methods

Field methods are most often employed for monitoring body composition in sport science applications, but with varying degrees of validity. The equipment is generally portable and measurements can be made at training or competition venues. In this section, we will review the accuracy, reliability, and utility of techniques such as anthropometry, bioelectrical impedance analysis (BIA), and weight–height indices such as the ubiquitous BMI.

Anthropometry Anthropometry involves measures taken on the surface of the body, including skinfolds, girths, and skeletal lengths and breadths. Despite the simplicity of concept, anthropometric measures may serve as useful surrogates for adiposity, muscularity, and frame size.

To date, well over 100 body fat prediction equations have been developed from skinfold and other measurements, but their highly variable outcomes result from differences in the populations sampled and a lack of rigor in standardizing the technique. For instance, varying the skinfold location by as little as 1 cm produces significantly different results, even when experienced practitioners measure the same participant (Hume & Marfell-Jones, 2008). Therefore, precise definitions for measurement sites in combination with a standardized technique are of fundamental importance for this method. We recommend using the International Society for the Advancement of Kinanthropometry (ISAK) protocol of eight skinfolds where possible (all on the right side of the body) so as to provide a representative sample of SAT deposition and thereby account for interindividual differences in adipose tissue patterning. One measurement from each site should be made in a predetermined order and then repeated to record either the mean of two or the median of three trials.

Segmental girths are often used as a surrogate measure of adiposity, particularly in obese individuals for whom skinfold testing is not possible. In contrast, for very muscular athletes, girth measures permit monitoring of muscularity or the robustness of physique. When measuring girths, a metal tape must lie against the skin (spanning any concavities), but not drawn so tightly as to compress it. The reader is referred to Stewart et al. (2011) for full descriptions of anthropometric measurements.

A further problem is created when prediction equations are employed to transform skinfolds, circumferences, and breadths to an estimate of FM or % fat, whereby the assumptions made in treating the data are of questionable validity. While many generalized equations have been cross-validated for specific samples, their use in determining fatness in athletes relies on conforming to the assumptions both of anthropometry and of either densitometry or DXA, depending on the criterion measure upon which the equation was created. Out of 18 such equations, only 3 were found to be reliable for use in male athletes when predicting densitometry-determined fat (Sinning et al., 1985).

Instead of converting skinfolds to % fat values, Kerr and Stewart (2009) recommended using a skinfold sum as a valid proxy for adiposity. Individual and sum of skinfolds can be compared to published norms (Table 6.1) for Olympic or International-level athletes (Ackland et al., 1997, 1998, 2001; Carter & Ackland, 1994; Carter et al., 2005; Kerr et al., 2007; Ridge et al., 2007). The rationale for proposing the skinfold thickness as a valid measure in its own right, without conversion to FM or % fat, centers on the avoidance of a series of assumptions which are known to be invalid—especially so in an athletic sample. These include assumptions of constant skin thickness, uniform SAT compressibility, consistent relative adipose tissue distribution, constant fat fraction within adipose tissue, constant internal to external distribution of fat, and above all, the assumed constancy of the FFM density.

Various regimens have summed values from different measurement sites in an attempt to capture a representative surface adiposity. While it is clear that certain sites such as the thigh and the iliac crest tend to be larger than others, such a pattern may

Table 6.1 Normative data for international-level athletes

			Skinfold sum (mm)[a]		
Sport	Level	Position/event	*N*	Mean ± SD	Range
Female athletes					
Basketball[b]	International	Guard	64	76.6 ± 22.2	36.4–143.5
		Forward	65	76 ± 20.1	40.9–131.7
		Center	47	88 ± 21.1	45.7–146.8
Diving[c]	International		39	65.6 ± 17	32.1–114.3
Slalom paddlers[d]	Olympic	Kayak	12	68.9 ± 13.9	46–99
Sprint paddlers[e]	Olympic		20	75.2 ± 16.3	51.5–103.6
Rowers[e]	Olympic	Lightweight	14	59.5 ± 11.9	40.1–77.9
		Open	73	89 ± 23.2	56.5–135.3
Triathlon[f]	International		19	62.8 ± 13.4	40.3–98.4
Swimming[c]	International		170	72.6 ± 19.6	37.9–147.1
Synchronized swimming[c]	International		137	81.7 ± 22.1	37.5–145.8
Waterpolo[c]	International		109	89.8 ± 23.8	39.7–151.6
Male athletes					
Diving[c]	International		43	45.9 ± 11.4	28–79.7
Slalom paddlers[d]	Olympic	Kayak	12	45.8 ± 9	32.4–63.5
	Olympic	Canoe	19	57.1 ± 9.4	38.3–73.7
Sprint paddlers[e]	Olympic		50	55.4 ± 16.2	39–89.3
Rowers[e]	Olympic	Lightweight	56	44.8 ± 8	31.2–62.3
		Open	153	65.4 ± 17.3	42.6–97.4
Triathlon[f]	International		19	48.3 ± 10.2	36.8–85.9
Swimming[c]	International		231	45.8 ± 9.5	26.6–99.9
Waterpolo[c]	International		190	62.5 ± 17.7	27.9–112.1

[a]Sum of eight skinfolds (unless otherwise indicated) = triceps, subscapular, biceps, iliac crest, supraspinale, abdominal, front thigh, medial calf.
[b]Sum of seven skinfolds from Ackland et al. (1997) = triceps, subscapular, biceps, supraspinale, abdominal, front thigh, medial calf.
[c]Sum of six skinfolds from Carter and Ackland (1994) = triceps, subscapular, supraspinale, abdominal, front thigh, medial calf.
[d]Ridge et al. (2007).
[e]Ackland et al. (2001).
[f]Ackland et al. (1998).

alter with increasing leanness. This introduces a further level of complexity (explaining why generalized formulae may not be valid for athletes) and affords the opportunity to track skinfold patterns, means, or ratios with leanness. The first of these is best depicted in a radial plot known as the skinfold map (Figure 6.6), where the profile can be used for tracking individual change or comparing an individual to group data.

Using the data presented by Kerr and Stewart (2009), the average skinfold magnitude across sites assessed by ISAK-qualified practitioners varies considerably by sport and is generally lower in males than females as depicted in Figure 6.7.

Using the same approach and comparing these grouped athlete profiles with those of a cohort of adult anorexic patients (Orphanidou et al., 1997), we see that female gymnasts have lower, but all other female athletes slightly higher, scores. Having said this, we should add that applying group data to individuals requires a degree of caution. Therefore, it is important to recognize the probability of individuals

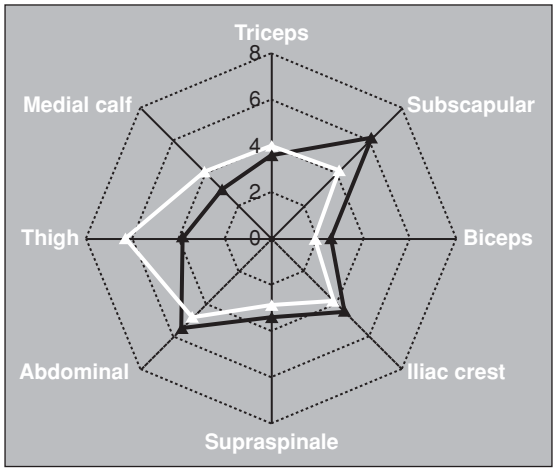

Figure 6.6 A skinfold map illustrating extreme leanness in elite adult male (black) and female (white) endurance athletes with a similar skinfold sum. Measurements in millimeters.

displaying different minimum skinfold thresholds before health or performance deteriorates.

In participants exhibiting extreme leanness, the characteristic fat patterning associated with males and females becomes less distinct with reduced variability in skinfold magnitude across sites. Nevertheless, some distinctiveness remains, with the male profile having the highest value at the subscapular site and the female profile at the thigh skinfold. This can be seen in Figure 6.6, where the male and female athletes have almost identical skinfold totals, but comparisons may be further elucidated by considering skinfold ratios. These have been used extensively in tracking fat patterning during childhood growth, but may have a role in identifying minimum fatness in athletes. Figure 6.8 depicts the leanest of an athletic sample for selected skinfold ratios (the same individuals as in Figure 6.6) and also the equivalent mean values for 106 male

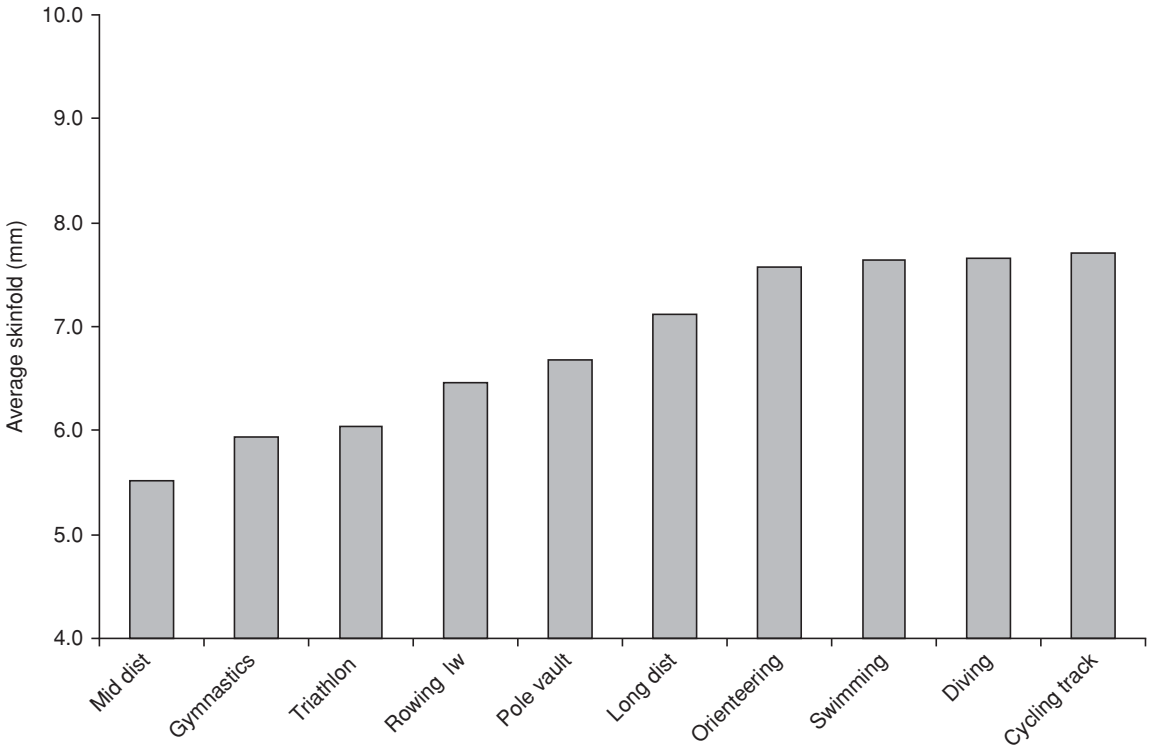

Figure 6.7 Average skinfold depth across seven or eight sites for male athlete groups. Data calculated from summary presented in Kerr and Stewart (2009). Mid dist, middle-distance track runners; lw, lightweight; Long dist, long-distance runners.

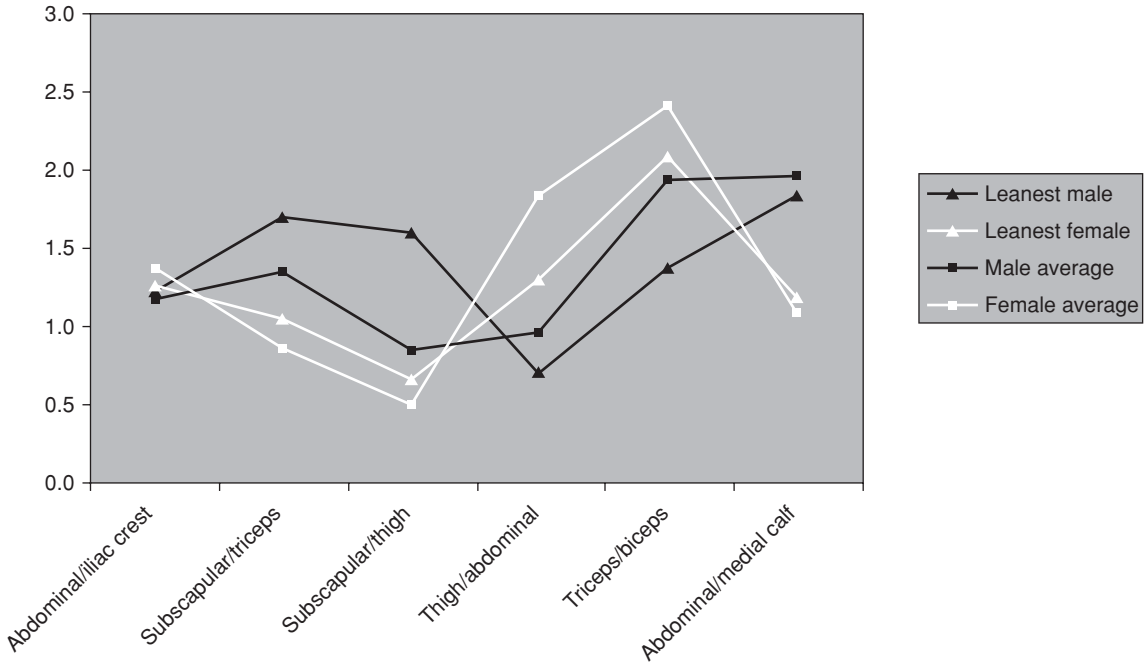

Figure 6.8 Skinfold ratios showing extremely lean male and female endurance athletes, compared with mean values from 106 male and 30 female athletes across a range of sports from Stewart (1999). Note some ratios depict a physique gradient (e.g., thigh/abdominal), while others depict only sexual dimorphism (e.g., abdominal/medial calf). The abdominal/iliac crest ratio shows neither.

and 30 female elite athletes from a range of sports (Stewart, 2003).

Some interesting observations can be made from Figure 6.8, including that subscapular:triceps, thigh:abdominal, and triceps:biceps skinfold ratios all appear to exhibit a difference between the leanest and mean values, thereby suggesting a "physique gradient" of fat patterning. Conversely, the abdominal:medial calf skinfold ratio displays no such gradient, although gender differences appear preserved. The abdominal:iliac crest skinfold ratio appears to depict neither a physique gradient nor sexual dimorphism. While caution may be advised in the use of ratios as opposed to absolute values as a result of error propagation, ratios appear ubiquitously throughout exercise science for monitoring athletes, and a combination of absolute and ratio scores (in conjunction with health and performance measures) might best serve scientists seeking to use skinfolds to establish a system of flagging inappropriately low body fat levels.

In summary, anthropometry provides a simple and highly portable field method for estimating body composition via surrogate measures for fatness and muscularity. Of all techniques, anthropometry has the best established international protocol for use and is accessible to those who routinely assess athletes. Provided the measurer is properly trained to minimize error and follow a standard protocol, the assumptions of the technique are acknowledged, and the data treatments are not confounded with additional sources of error (i.e., conversion to % fat), the anthropometric techniques have widespread utility for monitoring athlete body composition.

Bioelectrical Impedance Analysis The BIA device introduces a source of alternating current and detects it at a different body location. Because water and the salts contained therein conduct electricity well, whereas other molecules (principally lipid) are poor conductors, BIA predicts the water content of the body and "translates" this into an estimate of

tissue composition by subtraction from scale mass. Though BIA has been used widely to estimate body composition and many equations have been produced (Chumlea & Sun, 2005), its accuracy is limited for estimating body water and body fatness.

These devices typically employ a tetrapolar arrangement where the source and detection electrodes are placed on the dorsal surfaces of the right hand and foot approximately 5 cm apart. Other techniques use electrodes on both sides of the arms and legs (Malavolti et al., 2003). BIA assumes that a body segment comprises an isotropic material and that the body can be represented by limb and torso cylinders of similar impedance (the head is discounted). In practice, body tissues, especially in the torso, are considerably more complex in both morphology and composition and thus in conductance.

BIA also requires numerous assumptions to be made concerning the hydration of tissues and their proportions, but athlete training programs introduce factors which make it difficult for participants to avoid violating these assumptions. For example, exercise elevates temperature and, therefore, affects specific electrical resistivity, whilst also causing wide fluctuations in hydration status. In addition, the impedance assessment is not properly representative of the whole body since approximately 97% of the total impedance comes from the limbs, but only 3% from the torso (where 50% of body fat may be typically located). Newer devices that involve hand-to-hand or foot-to-foot electrode arrangements sample only a portion of the body. Finally, and of greatest concern, is that the prediction models use gender, age, stature, and body mass as input data to collectively account for up to 85% of the variation in body fat prediction, which has led some scientists to question its validity. In addition, some devices have optional "athletic status" inputs, which, if selected, can alter the predicted % fat by up to 5%. While BIA may have applications in some clinical settings, it has a range of difficulties when applied to athletes which frustrate attempts to derive meaningful data for whole-body composition.

Body Mass Index Weight–height indexes have been used for many years in an attempt to determine the "ideal weight" for an individual. The best known of these is the BMI or Quetelet's index, which relates body mass to height via the equation $\text{BMI} = m/h^2$. All such indexes provide a measure of ponderosity, which is not the same as measuring fatness. For an individual of any given stature, body mass will vary according to the amount and density of lean body mass as well as adipose tissue mass. The BMI does not distinguish the body composition or structure of individuals, so misclassification (particularly of heavily muscled individuals being assessed as over-fat) is a problem especially among athletes. In addition, diurnal fluctuation in stature and mass combine to alter BMI by more than one unit per day in most adults, confirming the absurdity of using it for individual prediction.

Summary and Implications

All body composition techniques have some inherent problems, whether in methodology, in interpreting the data, or in the assumptions that are made. Limitations in both the 2-component model (accuracy, assumptions) and multicomponent model (practicality, cost) highlight the pressing need for an economical laboratory or field approach to body composition assessment that is both accurate and reliable.

In the absence of such a criterion technique, several methods can be employed (with due caution) to play a useful role in monitoring body composition of athletes. For example, where the body composition assessment is used as a performance or selection criterion, then technique accuracy and reliability are of paramount importance. Ackland et al. (2012) suggest that the multicomponent model might be employed here provided the selected model accounts for the variability of the density of FFM in its computation. In this case, coaches and sport science support staff must give due consideration to the technical error of measurement and not apply an absolute criterion or threshold value for selection unilaterally. This is of particular importance when extremely lean athletes are examined.

If the athlete's body composition is being monitored to assess the effectiveness of an exercise or dietary intervention, then the use of some laboratory or field method may be more practical.

Laboratory techniques such as DXA, ADP, and US could be employed, provided the same equipment and a standardized protocol are used on each occasion. Similarly, a field method such as anthropometry offers a cost-effective means of monitoring SAT, provided the operator has the necessary training. BIA and BMI are not supported for assessing or monitoring body composition, nor are those methods that rely on an assumed constant density of FFM in their computation of % fat.

Regardless of the method favored, it is important to appreciate that athletes need to present for measurement in a standardized manner. Their adherence to protocols such as fasting, the amount of exercise in the past 12–24 hours, and maintaining hydration can greatly affect the veracity of recorded body composition data.

Ultimately, many sports have physique imperatives for elite performance, and body composition measurement casts light on the process of optimizing these. However, two further points are worthy of mentioning. First, two individuals of similar body composition may have different performance and the tolerance of an extremely lean physique is likely to differ considerably between individuals. Second, while an ideal body composition may pose a biomechanical advantage, a degree of performance variability will occur independently of body composition, so composition measures may not be as useful for diagnostic, selective, or predictive purposes as is often anticipated.

References

Ackland, T.R., Schreiner, A.B., & Kerr, D.A. (1997) Absolute size and proportionality characteristics of world championship female basketball players. *Journal of Sports Sciences* 15(5), 485–490.

Ackland, T., Blanksby, B., Landers, G., et al. (1998) Anthropometric profiles of elite triathletes. *Journal of Science and Medicine in Sport* 1(1), 52–56.

Ackland, T., Kerr, D., Hume, P., et al. (2001) *Anthropometric normative data for Olympic rowers and paddlers*. Proceedings of the Annual Conference of Sports Medicine Australia (SMA), October 23–27, 2001, Perth, Australia.

Ackland, T., Lohman, T., Sundgot-Borgen, J., et al. (2012) Current status of body composition assessment in sport. *Sports Medicine* 42, 227–249.

Adams, J., Mottola, M., Bagnall, K.M., et al. (1982) Total body fat content in a group of professional football players. *Canadian Journal of Applied Sport Sciences* 7, 36–40.

Bellisari, A., Roche, A.F., & Siervogel, R.M. (1993) Reliability of B-mode ultrasonic measurements of subcutaneous adipose tissue and intra-abdominal depth: comparisons with skinfold thickness. *International Journal of Obesity and Related Metabolic Disorders* 17(8), 475–480.

Bender, D.A. & Bender, A.E. (1997) *Nutrition: A Reference Handbook*. Oxford University Press, Oxford.

Carter, J. & Ackland, T. (1994) *Kinanthropometry in Aquatic Sports*. Human Kinetics, Champaign, IL.

Carter, J.E., Ackland, T.R., Kerr, D.A., et al. (2005) Somatotype and size of elite female basketball players. *Journal of Sports Sciences* 23(10), 1057–1063.

Chumlea, W. & Sun, S. (2005) Bioelectrical impedance analysis. In *Human Body Composition*, 2nd edn (eds. S.B. Heymsfield, T.G. Lohman, Z.M. Wang, et al.), pp. 79–87. Human Kinetics, Champaign, IL.

Claros, G., Hull, H.R., & Fields, D.A. (2005) Comparison of air displacement plethysmography to hydrostatic weighing for estimating total body density in children. *BMC Pediatrics* 5, 37.

Collins, M.A., Millard-Stafford, M.L., Sparling, P.B., et al. (1999) Evaluation of the BOD POD for assessing body fat in collegiate football players. *Medicine & Science in Sports & Exercise* 31(9), 1350–1356.

Going, S. (2005) Hydrodensitometry and air displacement plethysmography. In *Human Body Composition*, 2nd edn (eds. S.B. Heymsfield, T.G. Lohman, Z.M. Wang, et al.), pp. 17–33. Human Kinetics, Champaign, IL.

Hall, K.D. (2007) Body fat and fat-free mass inter-relationships: Forbes's theory revisited. *British Journal of Nutrition* 97, 1059–1063.

Horn, M. & Müller, W. (2010) Towards an accurate determination of subcutaneous adipose tissue by means of ultrasound [abstract WCB-A01448-02611]. Sixth World Congress of Biomechanics, ISB, August 1–6, 2010, Singapore.

Hume, P. & Marfell-Jones, M. (2008) The importance of accurate site location for skinfold measurement. *Journal of Sports Sciences* 26, 1333–1340.

Institute of Medicine (2005) *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride, and Sulfate*, pp. 73–185. National Academic Press, Washington, DC.

Ishida, Y., Carroll, J., Pollock, M., et al. (1992) Reliability of B-mode ultrasound for the measurement of body fat and muscle thickness. *American Journal of Human Biology* 4, 511–520.

Jebb, S., Goldberg, G., & Elia, M. (1993) DXA measurements of fat and bone mineral density in relation to depth and adiposity. In *Human Body Composition* (eds. K.J. Ellis & J.D. Eastman), pp. 115–119. Plenum Press, New York.

Kerr, D. & Stewart, A. (2009) Body composition in sport. In *Applied Anatomy and Biomechanics in Sport*, 2nd edn (eds. T.R. Ackland, B.C. Elliott, & J. Bloomfield), pp. 67–86. Human Kinetics, Champaign, IL.

Kerr, D.A., Ross, W.D., Norton, K., et al. (2007) Olympic lightweight and open-class rowers possess distinctive physical and proportionality characteristics. *Journal of Sports Sciences* 25(1), 43–53.

Lohman, T.G., Harris, M., Teixeira, P.J., et al. (2000) Assessing body composition and changes in body composition. Another look at dual-energy X-ray absorptiometry. *Annals of the New York Academy of Sciences* 904, 45–54.

Malavolti, M., Mussi, C., Poli, M., et al. (2003) Cross-calibration of eight-polar bioelectrical impedance analysis versus

dual-energy X-ray absorptiometry for the assessment of total and appendicular body composition in healthy subjects aged 21–82 years. *Annals of Human Biology* 30, 380–391.

Nattiv, A., Loucks, A.B., Manore, M.M., et al. (2007) American College of Sports Medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise* 39, 1867–1882.

Nord, R. & Payne, R. (1995) Body composition by dual energy X-ray absorptiometry—a review of the technology. *Asia Pacific Journal of Clinical Nutrition* 4, 167–171.

Orphanidou, C.I., McCargar, L.J., Birmingham, C.L., et al. (1997) Changes in body composition and fat distribution after short term weight gain in patients with anorexia nervosa. *American Journal of Clinical Nutrition* 65, 1034–1041.

Peeters, M.W. & Claessens, A.L. (2011) Effect of different swim caps on the assessment of body volume and percentage body fat by air displacement plethysmography. *Journal of Sports Sciences* 29, 191–196.

Pietrobelli, A., Formica, C., Wang, Z., et al. (1996) Dual-energy X-ray absorptiometry body composition model: review of physical concepts. *American Journal of Physiology* 271, E941–E951.

Prior, B.M., Cureton, K.J., Modelsky, C.M., et al. (1997) *In vivo* validation of whole body composition estimates from dual-energy X-ray absorptiometry. *Journal of Applied Physiology* 83, 623–630.

Ridge, B., Broad, E., Kerr, D., et al. (2007) Morphological characteristics of Olympic slalom canoe and kayak paddlers. *European Journal of Sport Science* 7(2), 107–113.

Schoeller, D. (2005) Hydrometry. In *Human Body Composition*, 2nd edn (eds. S.B. Heymsfield, T.G. Lohman, Z.M. Wang, et al.), pp. 35–49. Human Kinetics, Champaign, IL.

Schranz, N., Tomkinson, G., Olds, T., et al. (2010) Three-dimensional anthropometric analysis: differences between elite Australian rowers and the general population. *Journal of Sports Sciences* 28, 459–469.

Sinning, W.E., Dolny, D.G., Little, K.D., et al. (1985) Validity of "generalised" equations for body composition analysis in male athletes. *Medicine & Science in Sports & Exercise* 17, 124–130.

Siri, W. (1956) The gross composition of the body. In *Advances in Biological and Medical Physics* (eds. J.H. Lawrence & C.A. Tobias). Academic Press, London.

Stewart, A. (1999)Body composition in athletes assessed by DXA and other methods. PhD thesis, University of Edinburgh, Edinburgh.

Stewart, A. (2003) Anthropometric fat patterning in male and female subjects. In *Kinanthropometry VIII: Proceedings of the 8th International Society for the Advancement of Kinanthropometry Conference* (eds. T. Reilly & M. Marfell-Jones), pp. 195–234. Routledge, London.

Stewart, A.D. & Hannan, W.J. (2000) Prediction of fat and fat free mass in male athletes using dual X-ray absorptiometry as the reference method. *Journal of Sports Sciences* 18, 263–274.

Stewart, A., Marfell-Jones, M., Olds, T., et al. (2011) *International Standards for Anthropometric Assessment*. International Society for the Advancement of Kinanthropometry, Potchesfstroom, South Africa.

Sundgot-Borgen, J. & Torstveit, M.K. (2010). Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine & Science in Sports* 20 (Suppl. 2), 112–121.

Vescovi, J.D., Hildebrandt, L., Miller, W., et al. (2002) Evaluation of the BOD POD for estimating percent fat in female college athletes. *Journal of Strength and Conditioning Research* 16, 599–605.

Wang, J., Gallagher, D., Thornton, J.C., et al. (2006) Validation of a 3-dimensional photonic scanner for the measurement of body volumes, dimensions, and percentage body fat. *American Journal of Clinical Nutrition* 83, 809–816.

Wilmore, J.H., Vodak, P.A., Parr, R.B., et al. (1980) Further simplification of a method for determination of residual lung volume. *Medicine & Science in Sports & Exercise* 12, 216–218.

Chapter 7

Carbohydrate Needs of Athletes in Training

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Introduction

Carbohydrate plays a central role in the performance of sport, providing an important and versatile substrate for regeneration of ATP to fuel the exercising muscle. Its versatility is manifested by its availability from endogenous and exogenous sources and its ability to be metabolized by both oxygen-dependent and independent mechanisms that can support either high rates of power production for shorter periods, or lower but more sustained power outputs. A further example of the importance of carbohydrate is its role as a fuel for the brain and central nervous system, underpinning performance-influencing factors such as pacing, perceptions of fatigue, motor skill, and concentration.

Body stores of carbohydrate are limited in relation to the requirements of exercise; in fact, they may be less than the fuel demands of a single sporting event. The availability of carbohydrate stores is limiting for the performance of prolonged continuous or intermittent sporting events, and is permissive for the performance of sustained high-intensity sports. As such, a key issue in promoting optimum performance is the matching of body carbohydrate stores with the fuel demands of the athlete's training or competition program.

Carbohydrate has also played a central role in the recognition of sports nutrition, with major breakthroughs in the science and practice of sports nutrition involving activities related to carbohydrate and exercise (see Table 7.1). The aim of this review is to summarize current guidelines related to the role of carbohydrate in the athlete's everyday diet, and in particular to optimize the adaptation to training. Special attention will be focused on new areas of knowledge or interest, and to acknowledge that each athlete has unique and changing requirements for carbohydrate based on his or her periodized training program.

Daily Carbohydrate Requirements for Training

The aim of training is to achieve optimum performance on the day of competition via three processes or paradigms: "training hard" to create the required training stimulus, "training smart" to maximize adaptations to the training stimulus, and "training specifically" to fine-turn the behaviors or physiology needed for competition strategies (Maughan & Burke, 2011). Each athlete needs to undertake a periodized training program, based on the principles of progressive overload, to develop the various physiological, biomechanical, metabolic, psychological, and other characteristics that underpin successful outcomes in his or her event. In some cases, the nutritional strategies needed to achieve the various paradigms of training are different from, and even opposite to, each other, so athletes also need to periodize their nutrition.

Training Harder—Refueling on a Daily Basis

Generally, the principle of supporting the athlete to train intensively without succumbing to illness,

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Table 7.1 Examples of the role of carbohydrate in the evolution of sports nutrition

Breakthroughs in scientific knowledge		Breakthroughs in athletic practice	
Techniques	Associated outcomes	Issue	Outcome
1960s: percutaneous biopsy techniques applied to skeletal muscle	Enhanced understanding of the role of muscle glycogen as a muscle fuel: • Pre-exercise glycogen stores are associated with exercise capacity • Strategies of exercise and diet can increase local muscle glycogen content	1969: Ron Hill uses carbohydrate loading technique to win European marathon championships in an impressive "come from behind" victory	Carbohydrate loading is popularized and becomes synonymous with marathon nutrition as well as being used in preparation for many endurance and ultra-endurance sports
1980s: tracer technology used to track oxidation of exogenous carbohydrate during exercise	• Recognition of carbohydrates consumed during exercise as additional muscle fuels capable of extending exercise performance	1980s: sports drinks initially manufactured to replace fluids and electrolytes become recognized as a fuel source for prolonged exercise	A range of sports drinks and other carbohydrate-containing products such as bars (1990s) and gels (2000s) become popular aids for fueling prolonged sporting events
2000s: tracer technology used to track muscle oxidation of different types and mixtures of carbohydrate	• Recognition that intestinal limits to glucose absorption can be overcome by providing mixtures of carbohydrates absorbed via different transporters • Recognition that high rates of carbohydrate intake are associated with better performance and reduced gut discomfort during exercise lasting longer than about 3 h	2000s: recognition that successful athletes in many prolonged sports (e.g., ironman triathlons and cycling stage races) consume carbohydrate at rates greater than the guidelines	• Guidelines for carbohydrate intake during exercise are updated to encourage higher intakes of carbohydrate during sports >3 h duration • Sports products are manufactured using multiple forms of carbohydrate with different absorption characteristics (multiple transportable carbohydrates) to allow athletes to consume large amounts of carbohydrate during ultra-endurance sports with reduced risk of GI upsets
2000s: functional magnetic resonance imaging (fMRI) used to track changes in brain activity following carbohydrate intake	• Confirmation that mouth rinsing with carbohydrates enables receptors in mouth to communicate with reward centers in central nervous system promoting well-being and allowing the athlete to pace at a higher intensity	2000s: manufacture and use of new products such as sports confectionery allow athletes to practice mouth tasting of carbohydrate during a range of non-endurance sports	• Guidelines for carbohydrate intake during exercise are updated to acknowledge that intake of small amounts of carbohydrate—even as mouth rinse—can enhance performance of sports of 45–75 min

injury, and chronic fatigue is underpinned by the provision of adequate muscle fuel, particularly, muscle glycogen. The restoration of muscle and liver glycogen is a fundamental goal of recovery between training sessions, particularly when the athlete undertakes multiple workouts within a condensed time period. The 2010 IOC Consensus on Nutrition for Sport provided an opportunity to review the guidelines for daily carbohydrate intake to provide adequate fuel for workouts or competition and to refuel between sessions (Burke et al., 2011). The updated principles of eating to promote glycogen storage are summarized in Table 7.2, with a brief explanation of the evidence to support these strategies. Overall, the prevailing theme of the athlete's everyday diet remains that carbohydrate is

Table 7.2 Updated principles for refueling after exercise

Principles	Supporting evidence
The most important dietary factor in determining glycogen storage is the amount of carbohydrate consumed. Daily requirements for carbohydrate should track glycogen requirements for immediate training or to refuel for future sessions.	There is a direct relationship between the quantity of dietary carbohydrate and post-exercise glycogen storage, at least until the muscle storage capacity or threshold has been reached (Burke et al., 2004; Costill et al., 1981).
When the period between exercise sessions is <8 h, the athlete should consume carbohydrate as soon as practical after the first workout to maximize the effective recovery time between sessions.	Glycogen storage is most rapid when carbohydrate is consumed in the hours immediately after exercise, but most importantly, in the absence of carbohydrate intake, refueling is ineffective (Ivy et al., 1988).
Early post-exercise recovery may be enhanced by a higher rate of carbohydrate intake, especially when consumed in frequent small feedings.	Highest rates of glycogen synthesis have been reported during first 4 h of recovery when high rates of carbohydrate intake are consumed in serial feedings (Jentjens et al., 2001; van Hall et al., 2000; van Loon et al., 2000).
During longer recovery periods (24 h) when the athlete can consume adequate energy and carbohydrate, the types, pattern, and timing of carbohydrate-rich meals and snacks can be chosen according to what is practical and enjoyable.	When conditions support optimal refueling, there is no difference in glycogen synthesis between liquid or solid forms of carbohydrate (Keizer et al., 1986), or if carbohydrate is spread over large meals or frequent smaller snacks (Burke et al., 1996; Costill et al., 1981). When there is adequate time for refueling, it does not appear to matter if carbohydrate intake is delayed for a couple of hours (Parkin et al., 1997) to suit issues of food availability or appetite.
Carbohydrate-rich foods with a moderate high glycemic index (GI) provide a readily available source of substrate for glycogen synthesis. This may be important in situations where maximum glycogen storage is required in the hours after an exercise bout.	Foods with a low glycemic index appear to be less effective in promoting glycogen storage (Burke et al., 1993). This may be partly due to poor digestibility that overestimates actual carbohydrate intake (Jozsi et al., 1996) and may be compensated by additional intake of these foods, or the addition of foods with a high GI to meals and snacks.
Nutrient-rich carbohydrate foods or other foods added to recovery meals and snacks can provide a good source of protein and other nutrients.	Other nutrients may be important in recovery processes and should also be consumed during the recovery period. As well as aiding protein synthesis, protein consumed during recovery may promote glycogen storage when carbohydrate intake is sub-optimal (for review, see Betts & Williams, 2010).
Adequate energy intake is needed to optimize glycogen storage; restrained eating practices of some athletes, interferes both with meeting targets for carbohydrate intake and optimizing glycogen storage from this intake.	When energy intake is restricted, lower glycogen storage may be expected from the same carbohydrate intake (Tarnopolsky et al., 2001).
Although there are small differences in glycogen storage across the menstrual cycle, females can store glycogen as effectively as male athletes if they consume adequate carbohydrate and energy.	Greater storage of glycogen occurs during the luteal phase rather than the follicular phase (Hackney et al., 1994; Nicklas et al., 1989). Weight-conscious female athletes are at high risk of lower rates of glycogen storage due to sub-optimal intakes of carbohydrate and energy (Tarnopolsky et al., 1995, 1997). However, when carbohydrate and energy intake are adequate, females can store glycogen as effectively as males (Tarnopolsky et al., 1997, 2001).

Principles	Supporting evidence
Athletes should follow sensible practices regarding alcohol intake at all times, but particularly in the recovery period after exercise.	Excessive intake of alcohol after exercise may directly inhibit glycogen storage while blood alcohol concentration is elevated. The most important effects of alcohol intake on refueling, however, is via a reduced ability, or interest, to achieve dietary goals—i.e., optimum amount and timing of carbohydrate intake (Burke et al., 2003).

Source: Modified from Burke et al. (2011).

important for performance and that daily intake should track with the muscle's acute fuel requirements or the need to prepare glycogen stores for a future exercise session. Indeed, although some studies fail to show benefits (Cox et al., 2010; Sherman et al., 1993), perhaps in part due to methodological issues (see Burke, 2010), others show that when carbohydrate intake is higher and better matched to muscle fuel needs, the athlete can train harder (Costill et al., 1988; Simonsen et al., 1991) or perform better at the end of an intensive training block than a trial or group consuming a lower carbohydrate intake (Achten et al., 2004; Halson et al., 2004; Simonsen et al., 1991). Nevertheless, the language and the intention of the latest sports nutrition guidelines (Burke et al., 2011) have distanced themselves in several ways from the rhetoric of previous decades in which blanket recommendations of a "high carbohydrate diet" for all athletes were common.

First, the updated guidelines recognize that adequate glycogen stores are most relevant when it is important to train hard and perform well, particularly for high-intensity exercise. However, they also recognize that the elements and goals of the training of athletes are different from each other, and vary from day to day (i.e., the microcycle) and across macrocycles, according to the principle of periodization. Therefore since each athlete's carbohydrate requirements are individual and changing rather than uniform and static, his or her daily carbohydrate intake may vary both in quantity and importance. A second recommendation from more recently devised guidelines (Burke et al., 2004) is that targets for carbohydrate intake should not be provided in terms of percentage of total energy intake (e.g., carbohydrate should provide more than 55% of energy intake). This "% energy" terminology is not easily translated into practice, but more importantly, it can give a misleading account of absolute amounts of carbohydrate since it is confounded by the energy denominator (Burke et al., 2001). Instead, targets for daily carbohydrate intake are usefully given in amounts (grams) scaled to body mass (as a proxy for the volume of active muscle) and exercise load (intensity and duration). The suggested targets for an adequate carbohydrate supply for exercise are summarized in Table 7.3 with the caveat that these should be fine-tuned with individual consideration of total energy needs, specific training needs, and feedback from training performance.

A final update recommended a replacement of generic descriptions of "high carbohydrate diets" or "low carbohydrate diets" with a more specific consideration of how well an athlete's carbohydrate intake addresses their individual fuel needs for exercise (Burke et al., 2011). Just as "energy availability" has been coined to define an athlete's energy intake in relation to the energy costs of their specific exercise program, "carbohydrate availability" is a preferable way to discuss carbohydrate intake. An athlete's carbohydrate status can be judged in terms of whether their total daily intake and the timing of its consumption in relation to exercise maintains an adequate supply of carbohydrate substrate for the muscle and central nervous system ("high carbohydrate availability") or whether carbohydrate fuel sources are depleted or limiting for the daily exercise program ("low carbohydrate availability"). This approach would mean that two athletes who consume the same amount of carbohydrate might differ in the assessment of how suitable these intakes are, or that an individual athlete with a relatively low

Table 7.3 2010 IOC guidelines for carbohydrate intake in the training diet

	Situation	Carbohydrate targets	Comments on type and timing of carbohydrate intake
TOTAL DAILY NEEDS FOR FUEL AND RECOVERY			
• When it is important to train hard or with high intensity, daily carbohydrate intakes should match the fuel needs of training and glycogen restoration • These general recommendations should be fine-tuned with individual consideration of total energy needs, specific training needs, and feedback from training performance • Since training commitments change due to periodization in sport, an athlete's daily carbohydrate intake should also be varied			
Light	• Low intensity or skill-based activities	3–5 g/kg of athlete's body mass (BM)/day	• Intake may be timed to provide fuel intake around training sessions in the day. Otherwise, as long as total fuel needs are provided, the pattern of meals/snacks may simply be guided by convenience and individual choice • Carbohydrate foods or meal combinations that are protein and nutrient rich will allow the athlete to meet other acute or chronic sports nutrition goals
Moderate	• Moderate exercise program (i.e., ~1 h/day)	5–7 g/kg/day	
High	• Endurance program (e.g., 1–3 h/day moderate high intensity exercise)	6–10 g/kg/day	
Very high	• Extreme commitment (i.e., >4–5 h/day moderate high intensity exercise	8–12 g/kg/day	
SPECIAL TIMING OF INTAKE TO SUPPORT KEY TRAINING SESSIONS			
• Consuming carbohydrate around key training sessions to include pre-workout intake, and refueling during and after the session is likely to enhance performance and recovery • A focus on eating around training sessions helps the athlete to automatically match their energy and carbohydrate intake with changing needs			
Pre-exercise fueling	1–4 g/kg consumed 1–4 h before training		• Timing, amount, and type of carbohydrate foods and drinks should be chosen to suit individual preferences/experiences • It may be useful to experiment with intended competition practices in some training sessions to fine time plans
Refueling during training	45–75 min duration	Small amounts throughout training (including "mouth rinsing")	• Even when supplementary muscle fuel is not needed, the brain responds to mouth contact with carbohydrate; the athlete may feel better and train harder. This may be useful if the athlete is "training low" with low muscle carbohydrate stores
	1–2.5 h	30–60 g/h	• Practicing with intended competition intake strategies will allow the plan to be fine tuned and for the athlete to adapt to it
	2.5–3 h (when simulating optimum competition practices and/or to support high daily energy needs)	Up to 90 g/h	• As for events of 1–2.5 h • Products providing multiple transportable carbohydrates (Glucose:fructose mixtures) will achieve high rates of oxidation of carbohydrate consumed during exercise
Post-exercise (especially when there is <8 h recovery between fuel-demanding sessions)	1–1.2 g/kg/h in first hour		• Nutrient-dense forms of carbohydrate (i.e., carbohydrate-rich foods and food combinations that also provide protein and micronutrients) can promote other goals of recovery as well as nutritional goals for overall health and well-being

Source: From Burke et al. (2011).

intake of carbohydrate on a rest day or during a light training phase might be considered to have adequate carbohydrate availability but would require a larger intake on days/periods of higher volume training.

Athletes are encouraged to adjust their daily carbohydrate intakes by adopting eating patterns in which meals/snacks providing carbohydrate (and other important nutrients) are consumed strategically around exercise sessions. This means that additional carbohydrate-rich foods/fluid is automatically consumed when the athlete undertakes an exercise session, allowing carbohydrate and energy intake to track with the changes in the fuel cost of the athlete's exercise commitments. Benefits include better performance and recovery from key workouts due to enhanced carbohydrate availability as well as opportunities to fine tune and adapt to intended race day practices. This will be discussed in the following sections.

Training Specifically—Practicing Competition Nutrition Strategies

Another goal of training is to prepare the athlete to undertake the nutritional strategies that will be important in their competition eating plan. Although training prepares the athlete to get to the starting line or opening phase of their event, performance can be further fine-tuned by nutritional practices undertaken before, during, or after (between) competition bouts. These competition nutrition strategies may include the targeted intake of carbohydrate in the days and hours prior to the event, as well as during exercise. Where a series of bouts is required to determine the outcome of competition, the intake of carbohydrate may be part of promoting recovery between events.

Practicing competition strategies during the training phase is an important part of event preparation. While many athletes consume carbohydrate before and during training sessions to suit the logistics of the workout or to provide the minimal amount needed to prevent frank fatigue, optimum competition strategies generally require a more proactive intake and may be dictated by rules of the sport or event. Guidelines for carbohydrate intake before and during events are also summarized in Table 7.3 and discussed in more detail in other chapters of this book. Part of the purpose of simulating these strategies in training may be adapting the plan to the athlete, so that competition fluid and food intake is adjusted to suit individual tolerance and opportunities for carbohydrate intake during the event. However, there is also an opportunity to adapt the athlete to the plan, to learn the behavioral skills associated with obtaining and consuming fluids and foods during exercise, and to train the gut to tolerate or process the ingested nutrients. Indeed, consuming carbohydrate during training sessions has been shown to increase the rate of oxidation of these exogenous carbohydrate sources, with the adaptation occurring presumably at the level of intestinal absorption (Cox et al., 2010).

Training Smarter—Should Athletes Train with Low Carbohydrate Availability?

The chronic changes in the muscle and other body systems achieved by training can be seen as a cumulative effect of the series of transient increases in intracellular signaling and gene expression that occur during the recovery from each single workout. Our previous paradigm was that these were best produced by intensive training. However, it has been uncertain whether it is a *lack* or a *surplus* of substrate that triggers and promotes the training adaptation process (Coyle, 2000). More recent insights into the molecular basis of training have identified various interactions of nutrition and exercise that can promote greater adaptations from the same training stimulus (i.e., "train smarter"), including altering carbohydrate status.

Studies have shown that compared with conditions of high carbohydrate availability, a bout of endurance exercise commenced with low muscle glycogen or after an overnight fast results in a greater transcriptional activation of enzymes involved in carbohydrate metabolism and an increase in adaptive responses favoring fat metabolism (see Chapter 13). This information underpins the recently described "train low, compete high" protocol—training with low carbohydrate availability to enhance the training response, but competing with high fuel availability to promote performance. Great interest in this concept was generated by a study by Hansen et al. (2005) in which untrained males undertook a 10-week program of

leg knee extensor "kicking" exercise. In a clever experimental design, each subject's legs undertook the same training program (5 hours per week) but received a different daily schedule: whereas one leg was trained daily, the contralateral leg undertook two training sessions close together on a single day followed by a rest day. The consumption of a carbohydrate-rich diet (~8 g/kg/day) meant that one leg exercised with full restoration of glycogen each day, while the "twice a day" leg undertook the second session with depleted muscle glycogen stores. The leg which undertook half of its sessions with low carbohydrate availability had a greater increase in resting glycogen content and citrate synthase activity and an almost twofold greater increase in one-legged exercise time to fatigue, despite a similar increase in maximum power (Hansen et al., 2005).

There is a misconception that "train low" techniques require chronic adherence to a low carbohydrate diet. In fact, there are a number of protocols that can be used to reduce the availability of exogenous and/or endogenous carbohydrate stores (see Table 7.4). Indeed, the

Table 7.4 Strategies to reduce carbohydrate availability to alter the molecular responses to endurance-based training sessions

Exercise-diet strategy	Main outcomes
Chronically low carbohydrate diet (carbohydrate intake less than fuel requirements for training)	• Chronic reduction in muscle carbohydrate availability (endogenous and potentially exogenous sources) for all training sessions, depending on degree of fuel mismatch • Chronic whole-body effects of low carbohydrate availability including impairment of immune system and central nervous system function
Twice-a-day training (low endogenous carbohydrate availability for the second session in a day achieved by limiting the duration and carbohydrate intake in recovery period after the first session)	• Reduction in endogenous and exogenous carbohydrate availability for the muscle during the second training session • Acute reduction in carbohydrate availability for immune and central nervous systems depending on duration of carbohydrate restriction and muscle fuel requirements of second session
Training after an overnight fast	• Reduction in exogenous carbohydrate availability for the muscle for the specific session • Potential reduction in endogenous carbohydrate availability if there is inadequate glycogen restoration from previous day's training • Acute reduction in carbohydrate availability for immune system and central nervous system depending on duration of carbohydrate restriction and fuel requirements of the session
Prolonged training with or without an overnight fast and/or withholding carbohydrate intake during the session	• Reduction in exogenous carbohydrate sources for the muscle for the specific session • Acute reduction in carbohydrate availability for immune and central nervous systems depending on duration of carbohydrate restriction and fuel requirements of the session
Withholding carbohydrate during the first hours of recovery	• Could provide adequate fuel availability for the specific session but amplify post-exercise signaling due to the short but targeted time of low carbohydrate availability—theoretically achieves both "training harder" and "training smarter" effects • Could interfere with refueling for subsequent training sessions if total carbohydrate intake is reduced rather than just delayed. Given limits in the total rate of glycogen synthesis, delaying the timing of intake may reduce the potential for total glycogen storage between two sessions that are < 8 h apart, regardless of total carbohydrate intake • May reduce immune system function or accentuate the immune-suppression that occurs after exercise

Source: From Burke (2010).
Note that permutations and combinations of these strategies could alter exogenous and endogenous carbohydrate supplies independently or interactively.

strategies that have been investigated include selective placement of some training sessions with low carbohydrate availability within an otherwise carbohydrate-adequate diet; for example, the two-a-day training protocol or undertaking prolonged exercise after an overnight fast while consuming only water (Burke, 2010). Although the findings have significant scientific merit and possible application for exercise programs targeting metabolic improvements and health outcomes, there are some concerns in applying the technique to sports performance. In fact, studies in trained populations have failed to find any enhancement of performance gains from the "two-a-day training" model of low glycogen training (Hulston et al., 2010; Yeo et al., 2008) or training in fasted/water fed state (Cox et al., 2010) over conventional training even when there was evidence of enhancement of the metabolic adaptations associated with training.

Several disadvantages are associated with train low techniques including an increased risk of illness and injury, and impairment of the ability to train at high intensities. This latter finding from several "train low" studies (Hulston et al., 2010; Yeo et al., 2008) is not insignificant since training at high speeds/power outputs/intensities is a cornerstone of the principles of preparation for elite sport (Burke, 2010). A pragmatic commentary on practices in the field is that most athletes already periodize the carbohydrate availability for their training sessions. By design or by accident, some workouts are undertaken with reduced carbohydrate availability (the second or third session of a day during high volume periods, early morning sessions undertaken before breakfast, training during a period of energy restriction for weight loss) while others are undertaken with good carbohydrate support (quality sessions scheduled during lower volume periods, sessions undertaken after a meal). Thus, the real question is not whether there is a role for dietary periodization with carbohydrate availability, but whether it should be exploited in a different way (Burke et al., 2011).

For the moment, athletes should focus on good carbohydrate availability for sessions requiring high intensity or high levels of technique and skill, while noting that it is less important during lower intensity workouts or conditioning sessions at the beginning of a season. Future studies should involve more intricate designs to investigate the role of "training low" in the periodized training program. In addition, evidence-based strategies such as caffeine supplementation (Burke, 2008) or carbohydrate mouth rinsing (Jeukendrup & Chambers, 2010) might be able to "rescue" the decrements in training intensity associated with training with low carbohydrate availability to overcome one of its apparent disadvantages.

Promoting Glycogen Storage with Suboptimal Carbohydrate Intakes

Although adequate intakes of carbohydrate and energy will promote optimum glycogen storage, there are a number of situations in which these conditions may not be achievable. This includes the reduced post-exercise intake of food/fluid associated with a suppressed appetite, as well as the restricted energy intake and dietary restraint associated with achieving or maintaining low body mass/body fat levels. This is particularly common among female athletes and athletes in weight-division sports. Therefore, there is practical interest in developing strategies to promote better glycogen synthesis when total carbohydrate intake is below the amount needed for maximum glycogen storage. Possible strategies include altering the timing and pattern of intake of the suboptimal carbohydrate amounts. Unfortunately, no studies of this issue are available.

Some opportunities apparently exist, however, to enhance glycogen storage from a given amount of carbohydrate. For example, the use of a high-molecular-weight glucose polymer (Piehl-Aulin et al., 2000), the co-ingestion of large amounts of caffeine (Pedersen et al., 2008), and prior creatine loading (Robinson et al., 1999; van Loon et al., 2004) have all been shown to increase glycogen storage. The practical implications of these findings warrant consideration. For example, the reliance on substantial amounts of a glucose polymer to provide a substantial proportion of total energy intake will reduce the nutrient density of the diet and may reduce the athlete's ability to meet other nutritional goals. Meanwhile, there may be side effects associated with large doses (9 mg/kg) of caffeine, such as sleep interference and the risk of tremors and elevated heart rates. Even if further studies can show that the

glycogen storage effects of caffeine occur at lower doses, individual sensitivity may prevent it from being routinely used in such a manner. Equally, side effects associated with creatine use, such as weight gain, may mean that some athletes may not want to take advantage of any benefits on refueling strategies. Therefore, these strategies may be useful only in situations such as competition, where glycogen supercompensation or enhanced refueling during brief recovery periods may make glycogen storage a priority over other issues. Further work is needed.

To date, the only dietary strategy of true value in increasing glycogen storage from suboptimal carbohydrate intakes is the addition of protein to carbohydrate-rich meals or snacks. The effects of protein intake on glycogen storage has been an issue of debate over the past two decades, with studies showing conflicting results of either a positive (Ivy et al., 2002; Zawadzki et al., 1992) or absent (Jentjens et al., 2001; van Hall et al., 2000) effect. However, a recent meta-analytical approach to this literature shows clear evidence that when carbohydrate intake is below the targets for optimum glycogen storage during the first 4 hours of recovery (i.e., <1 g/kg/h), the co-ingestion of about 20–25 g protein can enhance glycogen storage (Figure 7.1) (Betts & Williams, 2010). Further studies are needed to investigate whether this effect extends to longer time periods, although it should be recognized that the athlete gains other benefits from consuming well-timed doses of protein of similar amount in the recovery after exercise (see Chapter 11).

Summary and Future Directions for Research

Carbohydrate continues to be an important nutrient in the athlete's everyday diet. An athlete's daily carbohydrate intake and the timing of its intake in relation to a workout should be considered in terms of how well it can supply adequate substrate to the muscle and central nervous system during exercise: whether it meets fuel needs ("high carbohydrate availability") or whether carbohydrate stores become depleted ("low carbohydrate availability"). In preparation for competition, the athlete must periodize workouts between the goals of training hard, training smart, and training specifically. Each aspect may require different manipulations of carbohydrate intake: to provide high carbohydrate availability to promote the intensity and quality of some ses-

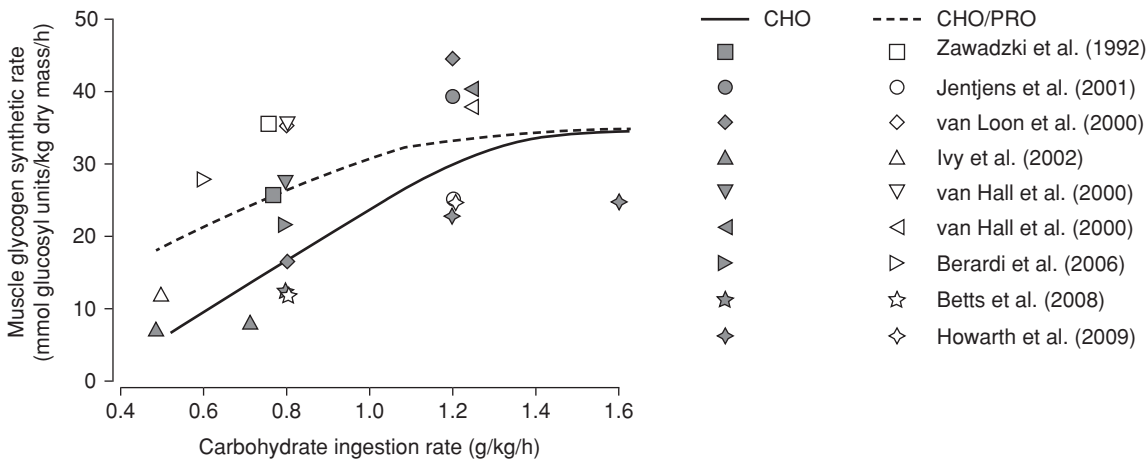

Figure 7.1 Reported rates of muscle glycogen resynthesis across nine studies that have compared muscle glycogen storage over >2–6 hours post-exercise with varied rates of carbohydrate (CHO) intake, with or without co-ingestion with protein (PRO). All studies have matched either for energy intake or for carbohydrate intake. From Betts and Williams (2010), with permission from Springer.

sions, to (perhaps) reduce carbohydrate availability to promote metabolic adaptations to other sessions, and finally to habituate with intended competition practices during other workouts. Quantitative and qualitative guidelines for carbohydrate intake, based on our updated knowledge, are summarized in Tables 7.2 and 7.3. Although we can be confident that these guidelines represent enhanced knowledge of the role of carbohydrate in sports nutrition, there are issues that still require further investigation. Future attention should be directed to a more strategic approach to training with low availability of endogenous and/or exogenous carbohydrate to enhance competition performance. Furthermore, for situations where energy restriction or practical challenges to eating cause suboptimal carbohydrate intake, it would be useful to know if manipulations can promote better glycogen storage from a given amount of carbohydrate intake. Such information will continue to promote.

References

Achten, J., Halson, S.H., Moseley, L., et al. (2004) Higher dietary carbohydrate content during intensified running training results in better maintenance of performance and mood state. *Journal of Applied Physiology* 96, 1331–1340.

Berardi, J.M., Price, T.B., Noreen, E.E., et al. (2006) Postexercise muscle glycogen recovery enhanced with a carbohydrate–protein supplement. *Medicine & Science in Sports & Exercise* 38, 1106–1113.

Betts, J.A. & Williams, C. (2010) Short-term recovery from prolonged exercise: exploring the potential for protein ingestion to accentuate the benefits of carbohydrate supplements. *Sports Medicine* 40, 941–959.

Betts, J.A., Williams, C., Boobis, L., et al. (2008) Increased carbohydrate oxidation after ingesting carbohydrate with added protein. *Medicine & Science in Sports & Exercise* 40, 903–912.

Burke, L.M. (2008) Caffeine and sports performance. *Applied Physiology Nutrition and Metabolism*, 33, 1319–1334.

Burke, L.M. (2010) Fueling strategies to optimize performance: training high or training low? *Scandinavian Journal of Medicine and Science in Sports* 20(Suppl. 2), 11–21.

Burke, L.M., Collier, G.R., & Hargreaves, M. (1993) Muscle glycogen storage after prolonged exercise: the effect of the glycemic index of carbohydrate feedings. *Journal of Applied Physiology* 75, 1019–1023.

Burke, L.M., Collier, G.R., Davis, P.G., et al. (1996) Muscle glycogen storage after prolonged exercise: effect of the frequency of carbohydrate feedings. *American Journal of Clinical Nutrition* 64, 115–119.

Burke, L.M., Cox, G.R., Cummings, N.K., et al. (2001) Guidelines for daily CHO intake: do athletes achieve them? *Sports Medicine* 31(4), 267–299.

Burke, L.M., Collier, G.R., Broad, E.M., et al. (2003) The effect of alcohol intake on muscle glycogen storage following prolonged exercise. *Journal of Applied Physiology* 95, 983–990.

Burke, L.M., Kiens, B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22, 15–30.

Burke, L.M., Hawley, J.A., Wong, S.H., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Science* 29(Suppl. 1), S17–S27.

Costill, D.L., Sherman, W.M., Fink, W.J., et al. (1981) The role of dietary carbohydrates in muscle glycogen resynthesis after strenuous running. *American Journal of Clinical Nutrition* 34, 1831–1836.

Costill, D.L., Flynn, M.G., Kirwan, J.P., et al. (1988) Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine & Science in Sports & Exercise* 20, 249–254.

Cox, G.R., Clark, S.A., Cox, A.J., et al. (2010) Daily training with high carbohydrate availability increases exogenous carbohydrate oxidation during endurance cycling. *Journal of Applied Physiology* 109, 126–134.

Coyle, E.F. (2000) Physical activity as a metabolic stressor. *American Journal of Clinical Nutrition* 72, 512S–520S.

Hackney, A.C., McCracken-Compton, M.A., & Ainsworth, B. (1994) Substrate responses to submaximal exercise in the midfollicular and midluteal phases of the menstrual cycle. *International Journal of Sport Nutrition* 4, 299–308.

Halson, S.H., Lancaster, G.I., Achten, J., et al. (2004) Effects of carbohydrate supplementation on performance and carbohydrate oxidation after intensified cycling training. *Journal of Applied Physiology* 97, 1245–1253.

Hansen, A.K., Fischer, C.P., Plomgaard, P., et al. (2005) Skeletal muscle adaptation: training twice every second day vs. training once daily. *Journal of Applied Physiology* 98, 93–99.

Howarth K.R., Moreau, N.A., Phillips, S.M., et al. (2009) Coingestion of protein with carbohydrate during recovery from endurance exercise stimulates skeletal muscle protein synthesis in humans. *Journal of Applied Physiology* 106, 1394–1402.

Hulston, C.J., Venables, M.C., Mann, C.H., et al. (2010) Training with low muscle glycogen enhances fat metabolism in well-trained cyclists. *Medicine & Science in Sports & Exercise* 42, 2046–2055.

Ivy, J.L., Katz, A.L., Cutler, C.L., et al. (1988) Muscle glycogen synthesis after exercise: effect of time of carbohydrate ingestion. *Journal of Applied Physiology* 64, 1480–1485.

Ivy, J.L., Goforth, H.W., Damon, B.D., et al. (2002) Early post-exercise muscle glycogen recovery is enhanced with a carbohydrate–protein supplement. *Journal of Applied Physiology* 93(4), 1337–1344.

Jentjens, R.L., van Loon, L.J.C., Mann, C.H., et al. (2001) Addition of protein and amino acids to carbohydrates does not enhance postexercise muscle glycogen synthesis. *Journal of Applied Physiology* 91, 839–846.

Jeukendrup, A.E. & Chambers E.S. (2010) Oral carbohydrate sensing and exercise performance. *Current Opinion in Clinical Nutrition and Metabolic Care* 13, 447–451

Jozsi, A.C., Trappe, T.A., Starling, R.D., et al. (1996) The influence of starch

structure on glycogen resynthesis and subsequent cycling performance. *International Journal of Sports Medicine* 17(5), 373–378.

Keizer, H.A., Kuipers, H., Van Kranenburg, G. et al. (1986) Influence of liquid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response, and maximal physical working capacity. *International Journal of Sports Medicine* 8, 99–104.

Maughan, R.J. & Burke, L.M. (2011) Practical nutrition recommendations for the athlete. In *Sports Nutrition: More Than Just Calories—Triggers for Adaptation* (eds. R.J. Maughan & L.M. Burke), pp. 131–149. Nestec Ltd/S. Karger AG, Switzerland.

Nicklas, B.J., Hackney, A.C., & Sharp, R.L. (1989) The menstrual cycle and exercise: performance, muscle glycogen, and substrate responses. *International Journal of Sports Medicine* 10, 264–269.

Parkin, J.A.M., Carey, M.F., Martin, I.K., et al. (1997) Muscle glycogen storage following prolonged exercise: effect of timing of ingestion of high glycemic index food. *Medicine & Science in Sports & Exercise* 29, 220–224.

Pedersen, D.J., Lessard, S.J., Coffey, V.G., et al. (2008) High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coingested with caffeine. *Journal of Applied Physiology* 105, 7–13.

Piehl Aulin, K., Soderlund, K., & Hultman, E. (2000) Muscle glycogen resynthesis rate in humans after supplementation of drinks containing carbohydrates with low and high molecular masses. *European Journal of Applied Physiology* 81, 346–351.

Robinson, T.M., Sewell, D.A., Hultman, E., et al. (1999). Role of submaximal exercise in promoting creatine and glycogen accumulation in human skeletal muscle. *Journal of Applied Physiology*, 87,598–604.

Sherman, W.M., Doyle, J.A., Lamb, D.R., et al. (1993) Dietary carbohydrate, muscle glycogen, and exercise performance during 7 d of training. *American Journal of Clinical Nutrition* 57, 27–31.

Simonsen, J.C., Sherman, W.M., Lamb, D.R., et al. (1991) Dietary carbohydrate, muscle glycogen, and power output during rowing training. *Journal of Applied Physiology* 70, 1500–1505.

Tarnopolsky, M.A., Atkinson, S.A., Phillips, S.M., et al (1995) Carbohydrate loading and metabolism during exercise in men and women. *Journal of Applied Physiology* 78, 1360–1368.

Tarnopolsky, M.A., Bosman, M., MacDonald, J.R., et al. (1997) Postexercise protein–carbohydrate and carbohydrate supplements increase muscle glycogen in men and women. *Journal of Applied Physiology* 83(6), 1877–1883.

Tarnopolsky, M.A., Zawada, C., Richmond, L.B., et al. (2001) Gender differences in carbohydrate loading are related to energy intake. *Journal of Applied Physiology* 91, 225–230.

van Hall, G., Shirreffs, S. M., & Calbet, J.A. (2000) Muscle glycogen resynthesis during recovery from cycle exercise: no effect of additional protein ingestion. *Journal of Applied Physiology* 88, 1631–1636.

van Loon, L.J.C., Saris, W.H.M., Kruijshoop, M., et al. (2000) Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *American Journal of Clinical Nutrition* 72, 106–111.

van Loon, L.J.C., Murphy, R., Oosterlaar, A.M., et al. (2004) Creatine supplementation increases glycogen storage but not GLUT-4 expression in human skeletal muscle. *Clinical Science* 106, 99–106.

Yeo, W.K., Paton, C.D., Garnham, A.P., et al. (2008) Skeletal muscle adaptation and performance responses to once a day versus twice every second day endurance training regimens. *Journal of Applied Physiology* 105, 1462–1470.

Zawadzki, K.M., Yaspelkis, B.B., & Ivy, J.L. (1992) Carbohydrate–protein complex increases the rate of muscle glycogen storage after exercise. *Journal of Applied Physiology* 72, 1854–1859.

Chapter 8

The Regulation and Synthesis of Muscle Glycogen by Means of Nutrient Intervention

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Introduction

Glycogen is a branched polymer of glucose, stored predominately in the skeletal muscle and liver, although other tissues such as brain and adipose can store glycogen. The basic polymerization of glycogen is via α-1,4-glycosidic linkages between glucose residues, with branch points introduced by the occurrence of α-1,6-glycosidic linkages about every six to seven glucose residues. This creates a compact molecular structure referred to as a β-particle in mammalian muscle and as an α-particle in liver.

Glycogen stored in the liver provides a carbohydrate reserve for blood glucose whereas muscle glycogen serves as a major fuel source for muscle work. Although muscle glycogen represents less than 4% of total energy storage in the body, it is the most important fuel source during prolonged exercise of moderate to high intensity, high intensity interval exercise, and resistance exercise. Supporting the importance of muscle glycogen are early research findings demonstrating that the capacity for prolonged strenuous exercise is directly related to the pre-exercise muscle glycogen concentration, that perception of effort during prolonged exercise parallels the declining muscle glycogen stores, and that strenuous exercise cannot be maintained once the muscle glycogen concentration is substantially reduced or depleted (Hultman, 1967).

Because of the importance of muscle glycogen as a fuel source, considerable research has been conducted to determine how it is synthesized and regulated, and how best to increase its rate of recovery when depleted. Early research also investigated means of raising the muscle glycogen stores above normal levels, or glycogen supercompensation, in preparation for endurance competitions such as a marathon. The current chapter begins with a brief discussion of the structure of glycogen and its regulation. Next, protocols for glycogen supercompensation are reviewed, followed by means of insuring muscle glycogen replenishment from one day to the next. Since many athletes are involved in multiple training sessions and competitive events on the same day, the final topic discussed is nutritional interventions that maximize the rate of muscle glycogen synthesis during the hours immediately following exercise. The chapter concludes with recommendations for increasing and maintaining muscle glycogen stores for training and competition.

Glycogen Formation and Structure

The initial step in the formation of a glycogen particle is glycosylation of glycogenin on a tyrosine residue. Glycogenin is a uridine diphosphate (UDP)-glucosyltransferase that sits at the core of the glycogen particle. The current model for glycogenin action is for self-glycosylation within a dimer,

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with one subunit transferring glucose residues to its partner forming a glucan chain of ~10 residues. Once an initial glucan chain is formed, synthesis of the glycogen particle comes under the control of glycogen synthase in association with glycogenin. Glycogen synthase catalyzes the transfer of glucose from UDP-glucose to the terminal glucose residue at the non-reducing end of a glucan chain to form an α-1,4-glycosidic linkage similar to the actions of glycogenin. The α-1,4-glycosidic linkages between glucose residues are limited to eight or fewer by amylo (1,4-1,6) transglycosylase. Amylo (1,4-1,6) transglycosylase is a branching enzyme that catalyzes the transfer of a terminal glucan chain of approximately seven glucosyl residues from the end of an α-1,4 glucan chain to the 6-hydroxyl group of a glucose residue on the same or another glucan chain. This forms an α-1,6-glycosidic linkage and creates a compact spherical glycogen particle (Figure 8.1).

Associated with the glycogen particle are a number of enzymes in addition to glycogenin and glycogen synthase. These include, but are not limited to glycogen phosphorylase, debranching enzyme, glycogen phosphorylase kinase, laforin, malin, protein phosphatase-1, and AMP-activated protein kinase (AMPK) (Graham et al., 2010). It is believed that active binding and dissociation of these proteins to the glycogen particle regulate the net balance

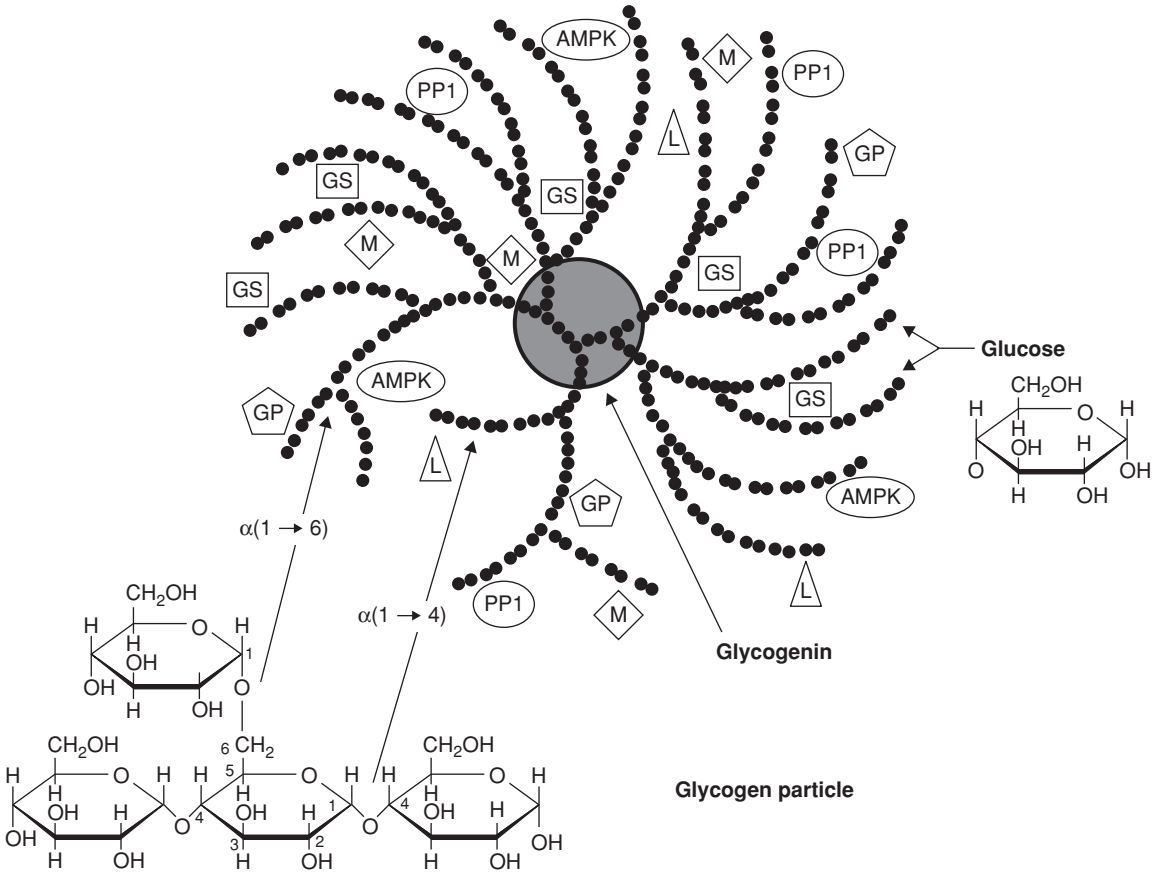

Figure 8.1 Glycogen particle and associated proteins, with a detailed illustration of the α-1,4-glycosidic and α-1,6-glycosidic linkages between glucosyl units. Branch points in the glucan chain start with α-1,6-glycosidic linkages. Proteins associated with the glycogen particle include but are not limited to: AMPK, AMP-activated protein kinase; GP, glycogen phosphatase; GS, glycogen synthase; L, laforin; M, malin; PP1, protein phosphatase-1.

between glycogen synthesis and degradation. Moreover, glycogen particles are found mainly in the subsarcolemmal, intermyofibrillar, and intramyofibrillar spaces of the myofibril, and this distribution is thought to facilitate the energetic requirements of different cellular functions (Graham et al., 2010).

Glycogen Synthase Regulation

Glycogen synthase is a member of a family of glycosyltransferases that catalyze the formation of α-1,4-glycosidic linkages of glycogen using UDP-glucose as substrate. Glycogen synthase activity is regulated by phosphorylation–dephosphorylation and is also allosterically activated by glucose-6-phosphate (G-6-P) (Danforth, 1965). Glycogen synthase can be phosphorylated at nine different sites, with phosphorylation reducing its activity and sensitivity to G-6-P. When dephosphorylated by the action of glycogen protein phosphatase-1, its activity and sensitivity to G-6-P progressively increases with decreasing phosphate content (Prats et al., 2009). Insulin and muscle contraction cause the dephosphorylation of glycogen synthase at several sites and increases enzymatic activity and sensitivity for activation by glucose-6-phosphate. In addition, insulin and contraction increase glycogen synthase affinity for UDP-glucose.

Recent research suggests that phosphorylation of glycogen synthase can affect its intracellular distribution. The phosphorylation of glycogen synthase on site 1b targets its association with intramyofibrillar glycogen particles (Meyer et al., 1970). Prats et al. (2011) reported that during exhaustive exercise, increases in plasma epinephrine directly correlated with increases in glycogen synthase phosphorylation of site 1b, which is phosphorylated by cAMP-dependent protein kinase. Since muscle contraction preferentially hydrolyzes intramyofibrillar glycogen, Prats et al. (2011) hypothesized that the rise in epinephrine during exercise targets glycogen synthase to the depleted glycogen pool by activating cAMP-dependent protein kinase. Therefore, following exercise, and when sufficient glucose and insulin are available, glycogen synthase would be appropriately located and activated for glycogen restoration. Furthermore, phosphorylation of glycogen synthase at site 2 by AMPK targets glycogen synthase to intermyofibrillar glycogen particles (Barre et al., 2007). Prats et al. (2011) suggest that AMPK acts as a sensor of intermyofibrillar glycogen particles, phosphorylating glycogen synthase at site 2 when these are low. This would inactivate the enzyme and target it to intermyofibrillar glycogen particles, whereupon a rise in insulin and increase in muscle glucose transport would reactivate glycogen synthase and restore glycogen in the intermyofibrillar particles. Support for this hypothesis is the finding by Barre et al. (2007) that chronic activation of muscle AMPK leads to intermyofibrillar glycogen accumulation.

Regulation of Glycogen Synthesis After Exercise

Following its depletion by exercise, muscle glycogen synthesis occurs in a biphasic manner. In the first phase, there is a rapid synthesis of glycogen that ranges between 12 and 30 μmol/g wet wt/h, which lasts for about 30–40 minutes and is insulin independent (Price et al., 1994). This phase, however, is activated only after muscle glycogen stores are substantially depleted and if sufficient glucose is available. In the second phase, which is insulin dependent, glycogen synthesis rates fall to 2–3 μmol/g wet wt/h under euglycemic conditions (Price et al., 1994). However, carbohydrate supplementation can increase the rate of synthesis during the slow phase by three- to four-fold (Ivy et al., 1988a), and if supplementation is maintained periodically it can eventually lead to above normal glycogen levels or glycogen supercompensation (Hultman, 1967).

Accounting for the rapid phase of glycogen synthesis following exercise is a high glycogen synthase active state, which is strongly influenced by the muscle glycogen concentration (Bergström et al., 1972). The lower the muscle glycogen concentration after exercise, the greater the activation of glycogen synthase. Conversely, as the glycogen concentration increases, glycogen synthase activity decreases. The reason for the inverse relationship between muscle glycogen content and glycogen synthase activity relates to the binding of both glycogen synthase and

glycogen protein phosphatase to glycogen as part of a glycogen protein complex as previously mentioned. When glycogen concentration decreases, both glycogen synthase and glycogen phosphatase are released, enabling the active phosphatase to catalyze dephosphorylation of glycogen synthase and activate the enzyme. This process is also under the influence of the intracellular G-6-P concentration. It has been proposed that increased binding of G-6-P to glycogen synthase exposes the phosphorylated sites to phosphatase attack. Therefore, a low muscle glycogen and high intracellular G-6-P level could contribute to an increased glycogen synthase activity after exercise. In this regard, it has been observed with ^{13}C-nuclear magnetic resonance (NMR) spectroscopy that the intracellular G-6-P concentration can increase fivefold after an exercise bout that substantially lowers the muscle glycogen stores (Bloch et al., 1994).

An exercise-induced increase in glycogen synthase activity can catalyze the rapid restoration of glycogen only if adequate substrate is available. Therefore, an essential cellular modification that enables the rapid increase in muscle glycogen after exercise is an increase in the permeability of the cell to glucose. In this respect, muscle contractions have a strong and prolonged insulin-like effect on the permeability of muscle to glucose (Ivy & Holloszy, 1981; Richter et al., 1984). The increase in muscle membrane permeability after exercise is due to a protracted increase in the number of glucose transporters incorporated into the plasma membrane (Goodyear et al., 1990). The increase in membrane glucose transporters induced by muscle contractile activity, however, reverses within 30–60 minutes as glucose transporters eventually recede from the plasma membrane in the absence of carbohydrate supplementation (Goodyear et al., 1990). Therefore, the increase in membrane permeability to glucose, together with the activation of glycogen synthase, allows for an initial rapid insulin-independent synthesis of muscle glycogen following exercise that significantly depletes the muscle glycogen stores.

The second phase of glycogen synthesis is characterized by a marked increase in the sensitivity of muscle to insulin. This increased sensitivity can result in muscle glucose uptake and glycogen synthesis with insulin concentrations that normally have no detectable effect on either process (Cartee et al., 1989). Furthermore, this increased sensitivity can be sustained for several days with appropriate carbohydrate consumption and results in glycogen supercompensation (Cartee et al., 1989; Richter et al., 1984). Appropriately, glycogen synthase under these conditions remains sensitive to allosteric activation by G-6-P and has an increased affinity for UDP-glucose. It is also possible that an increased expression of the insulin-regulated glucose transporter, GLUT4, contributes to muscle glycogen synthesis after exercise when synthesis is sustained for several days (Kuo et al., 1999).

Glycogen Supercompensation

Because muscle glycogen is the primary fuel source during intense exercise and its depletion is directly related to fatigue, methods of glycogen supercompensation prior to competition have been practised for many years. Initial research suggested that the most effective means of glycogen supercompensation was to first deplete muscle glycogen with vigorous exercise and consume a carbohydrate-free diet for 3 days. This was to be followed by another glycogen-depleting exercise and then a high carbohydrate diet for 3 days. This process elevated muscle glycogen stores to above 200 μmol/g wet wt, which is 50–60% greater than the normal muscle glycogen stores of well-trained endurance athletes (Hultman, 1967). While this was a very effective means of increasing muscle glycogen, it was a very strenuous regimen and many athletes found it impractical as it interfered with normal training and resulted in muscle soreness and sometimes injury.

As a result of these concerns, Sherman et al. (1981) evaluated a glycogen supercompensation regimen using a normal training taper, with only a moderate initial restriction of dietary carbohydrate. They had subjects taper their training for 6 days after an exercise that substantially reduced muscle glycogen stores. Over the next 5 days, training duration was systematically decreased and on the sixth day only light exercise or rest was allowed. Subjects consumed a mixed diet consisting of 40–50% carbohydrate during the first 3 days of taper. During the

final 3 days, the carbohydrate content of the diet was increased to 70%. Sherman et al. (1981) found that this regimen raised muscle glycogen stores in excess of 200 μmol/g wet wt similar to the more stringent protocols previously used, but without the risk of muscle soreness or injury. Therefore, these results suggest that in preparation for competition, and when time permits, athletes can substantially raise their muscle glycogen stores with a normal training taper and only small modification to their diets.

Daily Replenishment of Muscle Glycogen Stores

During periods of intense training or consecutive days of competition, it can be difficult for the athlete to maintain muscle glycogen stores from one day to the next. Unless sufficient carbohydrate is ingested, muscle glycogen will not be normalized on a day-to-day basis when training bouts are repeated daily. This was first demonstrated by Costill et al. (1971). They had runners train by completing 16.1 km runs on a treadmill at 80% VO_2 max on three consecutive days while consuming a low carbohydrate diet. By the third day, glycogen stores were significantly below baseline levels and several of the runners were unable to complete their 16.1 km runs. Similar results were found by Sherman et al. (1993). They fed cyclists and distance runners either low or high carbohydrate diets composed of 5 or 10 g of carbohydrate/kg body mass, respectively, during 7 days of controlled training. The low carbohydrate diet resulted in a significant 30% decrease in muscle glycogen by the fifth day of training, but no decrease in muscle glycogen occurred in the athletes provided with high carbohydrate diet.

When attempting to replenish muscle glycogen stores during the 24-hour period after exercise, the amount of carbohydrate to consume is likely the most important consideration (Figure 8.2). Costill et al. (1981) found that muscle glycogen storage during a 24-hour recovery period increased as the amount of carbohydrate consumed increased. Consumption of 2.3, 4.6, 6.5, and 7.8 g carbohydrate/kg body mass/day resulted in a glycogen storage rate of 0, 25, 55, and 70 μmol/g wet wt/day, respectively. In agreement with Costill et al. (1981), Starling et al. (1997) reported a 24-hour glycogen storage rate of ~77 μmol/g wet wt/day when 10 g carbohydrate/kg body mass/day were ingested following exercise, but a rate of only 8 μmol/g wet wt/day when 2.0 g carbohydrate/kg body mass were provided. Burke et al. (1993) reported the highest rates of glycogen synthesis during a 24-hour period after exercise, with rates in excess of 100 μmol/g wet wt when providing 10 g of carbohydrate/kg body mass/day. To achieve this rate of synthesis, however, a high-glycemic carbohydrate had to be used. Nevertheless, these results indicate that the amount of muscle glycogen stored following exercise is directly related to the amount of carbohydrate consumed. Consumption of 10 g carbohydrate/kg body mass/day appears, however, to maximize storage rate at ~100 μmol/g wet wt/day (Figure 8.2).

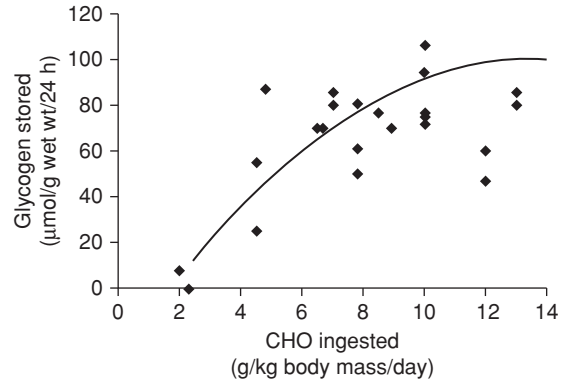

Figure 8.2 Muscle glycogen storage during the first 24 hours after exercise when different amounts of carbohydrate are ingested. (Studies analyzed are Blom et al., 1987a; Burke et al., 1995, 1996, 1993; Costill et al., 1981, 1990; Keizer et al., 1987; Parkin et al., 1997; Piehl, 1974; Sherman et al., 1983; and Starling et al., 1997).

As suggested by Burke et al. (1993), the type of carbohydrate ingested may be of significance. Costill et al. (1981) found that when runners received either 650 g/day of starch or simple carbohydrate, glycogen storage rates were similar over the first 24 hours of recovery. During the next 24 hours, muscle glycogen storage was 22.1 μmol/g wet wt for the starch diet and only 7.8 μmol/g wet wt for the simple carbohydrate diet. Burke et a1. (1993), however, found that during the first 24 hours after exercise, a diet incorporating high glycemic carbohydrates was

more effective than one incorporating low glycemic carbohydrates. Glycogen storage for the high and low glycemic carbohydrates was 106 and 71 μmol/g wet wt, respectively. More recently, Wee et al. (2005) studied the effects of a high or low glycemic breakfast consisting of 175 g of carbohydrate on muscle glycogen synthesis. During the postprandial period, muscle glycogen concentration increased by 15% after the high glycemic index meal but remained unchanged after the low glycemic index meal. Although glycogen synthesis was determined only 3 hours after breakfast, the results support the use of high glycemic carbohydrates to enhance muscle glycogen storage. Burke et al. (2004) suggested, however, that the greater rate of glycogen storage with high versus low glycemic carbohydrates cannot be completely explained in terms of a greater glucose and insulin response. They proposed that much of the difference in glycogen storage can be attributed to differences in the absorption rates of the carbohydrates.

The effect of frequency of food intake on rate of muscle glycogen storage has also been investigated. During recovery over a 24-hour period, frequency of meals does not appear to have an effect. Costill et al. (1981) reported that glycogen storage was essentially the same whether the carbohydrate was provided in two or seven meals over the course of a 24-hour recovery period. Burke et al. (1996) later confirmed these findings. They compared the effect of four large carbohydrate meals with a pattern of frequent, small, carbohydrate snacks during 24 hours of post-exercise recovery. Muscle glycogen storage following the four large meals averaged 74 ± 8 μmol/g wet wt and following the frequent snacks averaged 95 ± 15 μmol/g wet wt, but the difference in glycogen storage was not significant.

Timing of intake has also been viewed as important relative to the rate of muscle glycogen storage after exercise. Muscle glucose uptake and glycogen storage are accelerated when carbohydrate is ingested immediately after exercise as opposed to waiting several hours to supplement (Ivy et al., 1988a). However, Parkin et al. (1997) found that delaying supplementation by 2 hours after exercise had no effect on the total amount of glycogen storage during recovery periods of 8 and 24 hours. Therefore, while early post-exercise supplementation may be an appropriate strategy for short-term glycogen recovery, it does not appear to provide an advantage during long-term recovery.

In general, the most important consideration for replenishing muscle glycogen stores on a daily basis is the amount of carbohydrate consumed. Rates of muscle glycogen storage appear to increase in direct proportion to the amount of carbohydrate consumed up to about 10 g carbohydrate/kg body mass/day. The type of carbohydrate may also influence the rate of storage with high glycemic carbohydrates being more effective than low glycemic carbohydrates. Frequency and timing of supplementation appear less important for long-term glycogen recovery.

Short-Term Glycogen Storage

With the ever-increasing standard of competition in sport, athletes have to train harder, longer, and more frequently to remain competitive. Also, there are many sports in which the contest is divided into different periods providing brief intervals for recovery and other sports that require the athlete to compete in several different events on the same day. Because muscle glycogen synthesis is relatively slow, it cannot be completely replenished by nutritional intervention in such short periods of time, but that taking steps to maximize the rate of muscle glycogen replenishment during brief periods of recovery could provide a distinct competitive advantage. In this regard, timing, amount, frequency, and type of carbohydrate supplementation become significant. Also, adding protein and possibly caffeine to a carbohydrate supplement when recovery time is limited may increase the glycogen storage rate.

Timing of Carbohydrate Supplementation

Immediately following exercise that substantially lowers the muscle glycogen stores, skeletal muscle is highly sensitive to nutrient intervention. Muscle glycogen synthesis is more rapid if carbohydrate is consumed immediately after exercise as opposed to waiting several hours (Ivy et al., 1988a). When consuming carbohydrates immediately after exercise,

synthesis rates range between 5 and 7 μmol/g wet wt/h over 4 hours of recovery and these rates of synthesis can be maintained for up to 6 hours with frequent ingestion (Blom et al., 1987b; Ivy et al., 1988a, 1988b). Moreover, synthesis rates have been reported to be in excess of 12 μmol/g wet wt/h during the first 30–40 minutes after exercise (Price et al., 1994). Delaying supplementation for 2 hours reduces the rates of muscle glucose uptake and glycogen synthesis by 50% or more and occurs despite normal increases in blood glucose and insulin levels (Ivy et al., 1988a; Levenhagen et al., 2001). As previously discussed, glycogen synthesis can occur only if adequate substrate is available, and the rate of muscle glycogen synthesis can be extremely low after exercise if adequate carbohydrate is not made available to the muscle (Ivy et al., 1988a). Therefore, providing a carbohydrate supplement soon after exercise has the added benefit of starting the muscle glycogen recovery process immediately, thereby maximizing the effective recovery time.

Amount and Frequency of Carbohydrate Supplementation

The rate of muscle glycogen synthesis in the hours immediately following exercise is directly related to the amount of carbohydrate provided and frequency of supplementation. Blom et al. (1987b) found that when supplementing at 2-hour intervals, supplements containing a minimum of 0.7 g glucose/kg body mass raised the glycogen synthesis rate to ~6 μmol/g wet wt/h. Doubling the amount of glucose in the supplement did not increase the rate of synthesis, but by continuing to supplement at 2-hour intervals, it was possible to maintain a rapid rate of synthesis for up to 6 hours after exercise. Ivy et al. (1988b), however, reported that when supplementing at 2-hour intervals, 1.2–1.5 g glucose/kg body mass per supplement was required to reach rates of 5–7 μmol/g wet wt/h. When supplements exceeded 1.5 g glucose/kg body mass, the synthesis rate did not increase further. More recent research suggests that a synthesis rate of 8–10 μmol/g wet wt/h can be obtained during the immediate hours after exercise by increasing the amount of carbohydrate supplemented along with an increase in the frequency of supplementation (Doyle et al., 1993; Jentjens et al., 2001; van Hall et al., 2000; van Loon et al., 2000). Doyle et al. (1993) provided subjects 0.4 g glucose/kg body mass at 15-minute intervals for 4 hours starting immediately after a combination of endurance and resistance exercise. The total amount of carbohydrate provided averaged more than 500 g. Rates of glycogen synthesis, however, averaged ~10 μmol/g wet wt/h over the course of the 4-hour recovery. Jentjens et al. (2001) and van Loon et al. (2000) also reported synthesis rates of 8–10 μmol/g wet wt/h when subjects were provided 1.2 g glucose/kg body mass/h at 30-minute intervals during 5 and 3 hours of recovery, respectively. Increasing the amount of carbohydrate ingested to 1.6 g/kg body mass/h, however, did not have an additional benefit (Howarth et al., 2009). Therefore, the amount of carbohydrate required to maximize the rate of muscle glycogen synthesis appears to be ~1.2 g/kg body mass/h provided in 15- to 30-minute increments.

Type and Form of Carbohydrate Used for Supplementation

Many different types and combinations of carbohydrates have been evaluated for use in the rapid resynthesis of muscle glycogen. Blom et al. (1987b) found that the muscle glycogen synthesis rate was twice as fast when supplementing with glucose or sucrose compared with fructose during a 6-hour post-exercise recovery period. These results were later confirmed by van Den Bergh et al. (1996) using ^{13}C-NMR spectroscopy to follow muscle glycogen synthesis. Blom et al. (1987b) suggested that the difference in glycogen synthesis for glucose and fructose supplementation was due to differences in the way these sugars are metabolized. Fructose is preferentially metabolized in the liver whereas glucose is preferentially metabolized in the skeletal muscle. When infused, fructose has been found to cause a four times greater liver glycogen storage than glucose (Nilsson & Hultman, 1974). Accounting for the similar glycogen storage rates for sucrose and glucose, Blom et al. (1987b) suggested that the fructose fraction of sucrose, by virtue of its rapid metabolism in the liver, limited

hepatic uptake of the glucose fraction, thereby making a larger proportion of the glucose available for muscle glycogen synthesis.

Research has demonstrated that carbohydrate absorption is enhanced when free glucose and fructose are ingested simultaneously compared with glucose alone (Shi et al., 1995). Therefore, Wallis et al. (2008) compared the combination of glucose and fructose with glucose alone as a means of stimulating muscle glycogen synthesis. Starting immediately after exercise, subjects received either 60 g glucose and 30 g fructose or 90 g glucose/h at 30-minute increments for 4 hours. Muscle glycogen synthesis was not different between treatments and averaged between 8 and 9 μmol/g wet wt/h. The results suggest that supplementing with free glucose plus fructose has no advantage over supplementing with glucose alone during the hours immediately after exercise. However, prolonged endurance exercise can substantially reduce the liver glycogen stores and, therefore, there may be an advantage to using a combination of monosaccharides or added sucrose to enhance the rate of liver glycogen recovery. This approach appears feasible since substituting a portion of the glucose in a carbohydrate recovery supplement with fructose does not slow down the rate of muscle glycogen recovery (Wallis et al., 2008).

In an effort to investigate the effect of osmolality of a carbohydrate supplement on muscle glycogen synthesis, Piehl-Aulin et al. (2000) compared isoenergetic carbohydrate drinks containing a large polyglucoside with a mean molecular mass of 500,000–700,000 with a drink containing monomers and oligomers of glucose with a molecular mass of 500. The osmolality of the drinks was 84 and 350 mosmol/l, respectively. Each drink contained 75 g glucose in 500 ml water and was provided immediately, 30, 60, and 90 minutes after exercise. Glycogen synthesis during the first 2 hours of recovery was significantly higher for the low osmolality polyglucoside supplement (~10 μmol/g wet wt/h) than the high osmolality monomer–oligomer supplement (~6 μmol/g wet wt/h). The synthesis rate declined rapidly over the subsequent 2 hours of recovery and averaged ~4 μmol/g wet wt/h and ~5 μmol/g wet wt/h, respectively. The initial differences in glycogen synthesis between drinks were thought to be due to different rates of gastric emptying, as there were no differences in plasma glucose or insulin responses. These results suggest that a recovery drink containing a high molecular mass glucose polymer could be of benefit when limited time is available for restoration of muscle glycogen.

The finding that the osmolality of carbohydrate drinks may influence the rate of muscle glycogen storage suggests that the form in which the carbohydrate is presented may also have an effect. Little difference, however, seems to exist in the rates of glycogen synthesis elicited from solid and liquid carbohydrate supplements. For example, Keizer et al. (1987) evaluated the effectiveness of 300 g of carbohydrate in solid versus liquid form over a 5-hour recovery period. Despite higher plasma glucose and insulin levels during the solid feeding, the rate of muscle glycogen synthesis was not different between treatments. Similar results were obtained by Reed et al. (1989), when they provided 1.5 g glucose/kg body mass either as a liquid or solid supplement immediately and 2 hours after exercise, but the rates of glycogen synthesis for both the studies were rather slow, averaging ~5 μmol/g wet wt/h. Therefore, it may be of interest to re-evaluate this question with feeding protocols that promote higher rates of muscle glycogen synthesis.

The Addition of Protein

Zawadzki et al. (1992) were the first to evaluate the co-ingestion of protein and carbohydrate to stimulate post-exercise muscle glycogen synthesis. Subjects received a carbohydrate, protein, or carbohydrate plus protein supplement immediately after and 2 hours after exercise on three separate occasions. Carbohydrate plus protein significantly enhanced the insulin response produced by the carbohydrate and increased the rate of muscle glycogen synthesis by 38% during the first 4 hours of recovery. Protein supplementation alone had very little effect on glycogen synthesis. This study came under scrutiny because the carbohydrate plus protein supplement

and the carbohydrate supplement were not isoenergetic. Since this initial study, many studies using combinations of carbohydrate, protein, and amino acids have addressed this issue, with some studies supporting enhanced muscle glycogen synthesis with carbohydrate–protein supplements under isoenergetic conditions (Berardi et al., 2006; Ivy et al., 2002; Morifuji et al., 2010; Ruby et al., 2004) and others finding no difference (Carrithers et al., 2000; Howarth et al., 2009; Jentjens et al., 2001; van Hall et al., 2000; van Loon et al., 2000).

The differences in findings can most likely be attributed to differences in experimental design, including frequency of supplementation, and the amount and type of carbohydrate and protein provided. For example, in studies demonstrating a beneficial effect of co-ingestion of carbohydrate plus protein or amino acids, feeding intervals were 1–2 hours apart (Berardi et al., 2006; Ivy et al., 2002; Ruby et al., 2004; Zawadzki et al., 1992). Those studies that did not demonstrate a benefit from added protein used feeding intervals of 15–30 minutes (Carrithers et al., 2000; Jentjens et al., 2001; van Hall et al., 2000; van Loon et al., 2000). They also used substantially greater amounts of carbohydrate (Jentjens et al., 2001; van Hall et al., 2000; van Loon et al., 2000) and in some studies low levels of protein (Carrithers et al., 2000). The results from these studies imply that feeding high amounts of carbohydrate at frequent intervals can negate the benefits of added protein. However, the evidence is considerable that the addition of protein to a carbohydrate supplement will increase the efficiency of muscle glycogen storage when the amount of carbohydrate ingested is below the threshold of maximal glycogen synthesis or when feeding intervals are 1 hour or more apart (Berardi et al., 2006; Ivy et al., 2002; Morifuji et al., 2010; van Loon et al., 2000; Zawadzki et al., 1992). In fact, maximum rates of muscle glycogen synthesis can be achieved with substantially less carbohydrate and reduced frequency of supplementation when protein and carbohydrate are co-ingested. Moreover, addition of protein appears to have a very significant effect on muscle glycogen storage during the first hour of recovery as glycogen storage has been reported to be twice as fast after a carbohydrate–protein supplement than with an isoenergetic carbohydrate supplement, and four times faster than an iso-carbohydrate supplement (Ivy et al., 2002) (Figure 8.3). These results have implications for athletes who play sports in which they are regularly substituted during competition or participate in team sports in which competition is interspersed with periods of rest such as ice hockey, soccer, and basketball.

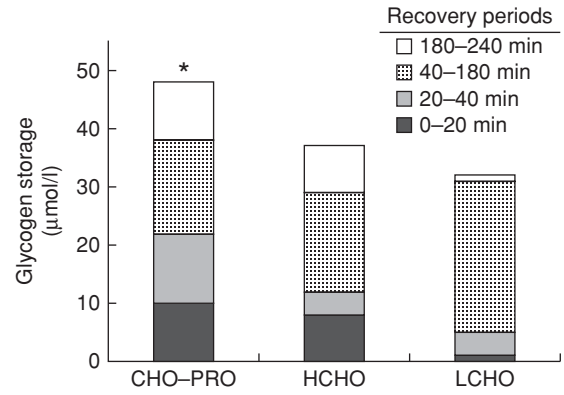

Figure 8.3 Patterns of muscle glycogen storage during recovery as determined by ^{13}C-NMR spectroscopy for carbohydrate–protein (CHO–PRO), isoenergetic carbohydrate (HCHO), and iso-carbohydrate (LCHO) supplements. Supplements were provided immediately and 2 hours after exercise. *Total glycogen storage for CHO–PRO was significantly different than HCHO and LCHO ($p < 0.05$). Based on data from Ivy et al. (2002).

The type of protein to use has received little attention, but the absorption rates and metabolism of proteins such as whey, casein, and soy are very different, and this could affect its efficacy with regard to glycogen storage. Recently, Morifuji et al. (2010) evaluated the effects of glucose or glucose combined with whey protein, whey hydrolysates, casein hydrolysates, or branched-chain amino acids (BCAA) on muscle glycogen synthesis in rats following glycogen-depleting exercise. During the initial 2 hours of recovery, glucose supplementation increased the glycogen stores onefold. With added whey protein or BCAA, glycogen stores increased twofold and were significantly greater than those seen with glucose alone. However, when whey hydrolysates were supplemented with glucose, muscle glycogen stores were increased threefold and were significantly greater than the glycogen

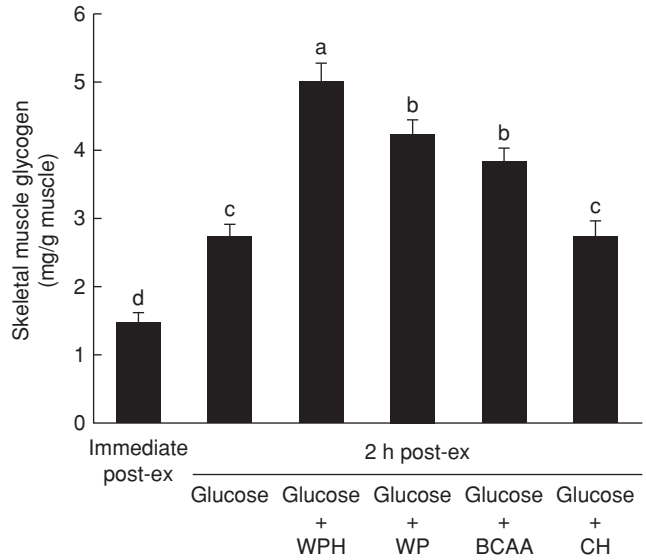

Figure 8.4 Muscle glycogen content immediately after exercise and after 2 hours of recovery grouped according to the different types of supplements ingested ($n = 8$ per group). Values are means ± SEM. Mean values within a column with unlike superscript letters are significantly different ($p < 0.05$). Supplement abbreviations are WPH, whey protein hydrolysates; WP, whey protein; BCAA, branched-chain amino acids; CH, casein hydrolysates. From Morifuji et al. (2010), with kind permission from Springer Science+Business Media.

stores following supplementation with whey or BCAA. Supplementing with casein hydrolysates plus glucose provided no additional benefit to glycogen storage beyond that of glucose alone (Figure 8.4).

The reason for the improved glycogen storage efficiency with carbohydrate–protein supplementation is not known. Although an increase in the plasma insulin response is typically observed when protein or amino acids are added to a carbohydrate supplement (van Loon et al., 2000; Zawadzki et al., 1992), this increase does not always correspond with an increase in muscle glycogen synthesis (van Loon et al., 2000). Furthermore, carbohydrate–protein supplementation has been observed to increase muscle glycogen synthesis without an enhanced insulin response (Ivy et al., 2002). Therefore, the improvement in glycogen storage with carbohydrate–protein supplementation is most likely unrelated to a greater insulin response. It has become evident that certain amino acids can regulate proteins in the insulin-signaling pathway and increase muscle glucose transport and glycogen synthesis in the absence of insulin (Doi et al., 2005; Kleinert et al., 2011). Also, they have been found to function additively with insulin to stimulate these processes (Kleinert et al., 2011). It, therefore, seems possible that by co-ingesting a carbohydrate–protein supplement, muscle glucose transport and glycogen synthesis can be activated by two separate signaling pathways resulting in a more efficient conversion of the carbohydrate portion of the supplement to glycogen.

The Addition of Caffeine

The co-ingestion of caffeine with carbohydrate was recently shown to enhance muscle glycogen storage after exercise (Pedersen et al., 2008). Immediately following exercise that substantially lowered muscle glycogen stores, subjects ingested ~1 g carbohydrate/kg body mass, and this was repeated at 60, 120, and 180 minutes after exercise, or they ingested 4 mg caffeine/kg body mass with the carbohydrate immediately after exercise and at 120 minutes after exercise. There was no difference

in the rate of muscle glycogen storage during the first hour of recovery, but by 4 hours of recovery glycogen storage had risen from ~20 to ~64 μmol/g wet wt with the carbohydrate plus caffeine treatment and from ~20 to ~46 μmol/g wet wt with the carbohydrate treatment ($p < 0.05$). However, this finding is not supported by a recent study by Beelen et al. (2012). Following exercise, their subjects received carbohydrate or carbohydrate plus caffeine supplementation every 30 minutes starting immediately after exercise. The supplements were designed to provide 1.2 g carbohydrate/kg body mass/h, or the carbohydrate plus 1.7 mg caffeine/kg body mass/h. During a 6-hour recovery period, subjects received a total of ~500 g of carbohydrate and ~725 mg of caffeine. Glycogen storage averaged ~6.2 μmol/g wet wt/h for both the treatments. Therefore, the benefit of adding caffeine to a post-exercise recovery supplement remains to be determined.

Recommendations

Unless an athlete is trying to gain or lose mass, the athlete should be encouraged to balance energy consumption with energy expenditure. If daily energy consumption is chronically low or high, it could have an adverse effect on the ability to train and compete. A recommendation for dietary carbohydrate should ensure the daily replenishment of the muscle glycogen stores and should be provided as an absolute amount of carbohydrate, not as a percentage of the macronutrient composition of the diet. Hence, the recommendation will depend on the type, duration, and intensity of exercise being performed by the athlete. During moderate duration, and low to moderate intensity training, 5–6 g/kg body mass of carbohydrate per day should suffice. If training duration is moderate, but the intensity is high, a carbohydrate intake of 7–8 g/kg body mass/day will be more appropriate. If the training program or competition is extreme and is carried out over multiple days, a carbohydrate intake of 9–10 g/kg body mass/day may be necessary. There appears to be little need to recommend more than ~10 g/kg body mass/day as this level of consumption appears to maximize the amount of carbohydrate stored naturally in a 24-hour period. Feeding frequency has not been found to significantly influence the amount of daily muscle glycogen storage. Therefore, athletes should be encouraged to develop a feeding schedule that best fits their individual situation. Research, however, suggests that carbohydrates with a moderate to high glycemic index should be a significant part of the athlete's diet.

When the time between training sessions or competitive events is only a few hours apart, athletes should begin carbohydrate consumption as soon after exercise as feasible, as the muscle is highly responsive to nutrient intervention at this time. Moreover, early intake maximizes the effective recovery time. Carbohydrate supplementation averaging 1.0–1.2 g/kg body mass/h provided at 30-minute intervals has been found to maximize glycogen storage during the early hours of recovery. However, this frequency of supplementation is impractical, and the amount of carbohydrate is excessive and constitutes an amount most athletes would reject. A more practical approach is to recommend a recovery supplement containing carbohydrate and protein. Carbohydrate supplements composed of 1.2–1.4 g/kg body mass with 0.3–0.4 g protein/kg body mass can maximize the muscle glycogen storage when provided at 2-hour intervals, thus reducing the carbohydrate requirement by 50% as well as the frequency of administration. In addition, proper post-exercise protein supplementation can limit muscle proteolysis and soreness, increase protein synthesis and enhance training adaptation.

Because muscle glycogen is such a vital fuel during exercise, athletes should be encouraged to raise their muscle glycogen stores to above normal levels in preparation for competition if there is adequate time. This can be done effectively by consuming a mixed diet consisting of about 50% carbohydrate during the first 3 days of a taper and then switching to a high carbohydrate diet consisting of ~70% carbohydrate during the final 3 days of the taper. This process has been found to elevate the muscle glycogen stores by between 50% and 70% in well-trained athletes.

References

Barre, L., Richardson, C., Hirshman, M.F., et al. (2007) Genetic model for the chronic activation of skeletal muscle AMP-activated protein kinase leads to glycogen accumulation. *American Journal of Physiology: Endocrinology and Metabolism* 292, E802–E811.

Beelen, M., Kranenburg, J.V., Senden, J.M., et al. (2012) Impact of caffeine and protein on postexercise muscle glycogen synthesis. *Medicine & Science in Sports & Exercise* 44, 692–700.

Berardi, J.M., Price, T.B. Noreen, E.E., et al. (2006) Postexercise muscle glycogen recovery enhanced with a carbohydrate–protein supplement. *Medicine & Science in Sports & Exercise* 38, 1106–1113.

Bergström, J., Hultman, E., & Roch-Norlund, A.E. (1972) Muscle glycogen synthetase in normal subjects. *Scandinavian Journal of Clinical and Laboratory Investigation* 29, 231–236.

Bloch, G., Chase, J.R., Meyer, D.B., et al. (1994) *In vivo* regulation of rat muscle glycogen resynthesis after intense exercise. *American Journal of Physiology, Endocrinology and Metabolism* 266, E85–E91.

Blom, P.C.S., Costill, D.L., & Vollestad, N.K. (1987a) Exhaustive running: inappropriate as a stimulus of muscle glycogen supercompensation. *Medicine & Science in Sports & Exercise*. 19, 398–403.

Blom, P.C.S., Høstmark, A.T., Vaage, O., et al. (1987b) Effect of different post-exercise sugar diets on the rate of muscle glycogen synthesis. *Medicine & Science in Sports & Exercise* 19, 491–496.

Burke, L.M., Collier G.R., & Hargreaves M. (1993) Muscle glycogen storage after prolonged exercise: effect of the glycemic index of carbohydrate feedings. *Journal of Applied Physiology* 75, 1019–1023.

Burke, L.M., Collier, G.R., Beasley, S.K., et al. (1995) Effect of coingestion of fat and protein with carbohydrate feedings on muscle glycogen storage. *Journal of Applied Physiology* 78, 2187–2192.

Burke, L.M., Collier, G.R., Davis, P.G., et al. (1996) Muscle glycogen storage after prolonged exercise: effect of the frequency of carbohydrate feedings. *American Journal of Clinical Nutrition* 64, 115–119.

Burke, L.M., Kiens, B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22, 15–30.

Carrithers, J.A., Williamson, D.L., Gallagher, P.M., et al. (2000) Effects of postexercise carbohydrate–protein feedings on muscle glycogen restoration. *Journal of Applied Physiology*, 88, 1976–1982.

Cartee, G.D., Young, D.A., Sleeper, M.D., et al. (1989) Prolonged increase in insulin-stimulated glucose transport in muscle after exercise. *American Journal of Physiology, Endocrinology and Metabolism* 256, E494–E499.

Costill, D.L., Bowers, R., Branam, G., et al. (1971) Muscle glycogen utilization during prolonged exercise on successive days. *Journal of Applied Physiology* 31, 834–838.

Costill, D.L., Sherman, W.M., Fink, W.J., et al. (1981) The role of dietary carbohydrate in muscle glycogen resynthesis after strenuous running. *American Journal of Clinical Nutrition* 34, 1831–1836.

Costill, D.L., Pascoe, W.J., Fink, W.J., et al. (1990) Impaired muscle glycogen resynthesis after eccentric exercise. *Journal of Applied Physiology* 69, 46–50.

Danforth, W.H. (1965) Glycogen synthetase activity in skeletal muscle. Interconversion of two forms and control of glycogen synthesis. *Journal of Biological Chemistry* 240, 588–593.

Doi, M., Yamaoka, I., Nakayama, M., et al. (2005) Isoleucine, a blood glucose-lowering amino acid, increases glucose uptake in rat skeletal muscle in the absence of increases in AMP-activated protein kinase activity. *Journal of Nutrition* 135, 2103–2108.

Doyle, J.A., Sherman, W.M., & Strauss, R.L. (1993) Effects of eccentric and concentric exercise on muscle glycogen replenishment. *Journal of Applied Physiology* 74, 1848–1855.

Goodyear, L. J., Hirshman, M.F., King, P.S., et al. (1990) Skeletal muscle plasma membrane glucose transport and glucose transporters after exercise. *Journal of Applied Physiology* 68, 193–198.

Graham, T.E., Yuan, Z., Hill, A.K., et al. (2010) The regulation of muscle glycogen: the granule and its proteins. *Acta Physiologica* 199, 489–498.

Howarth, K.R., Moreau, N.A., Phillips, S.M., et al. (2009) Coingestion of protein with carbohydrate during recovery from endurance exercise stimulates skeletal muscle proteins synthesis in humans. *Journal of Applied Physiology* 106, 1394–1402.

Hultman, E. (1967) Studies on muscle metabolism of glycogen and active phosphate in man with special reference to exercise and diet. *Scandinavian Journal of Clinical and Laboratory Investigation* 19(Suppl. 94), 1–63.

Ivy, J.L. & Holloszy, J.O. (1981) Persistent increase in glucose uptake by rat skeletal muscle following exercise. *American Journal of Physiology* 241, C200–C203.

Ivy, J.L., Katz, A.L., Cutler, C.L., et al. (1988a) Muscle glycogen synthesis after exercise: effect of time of carbohydrate ingestion. *Journal of Applied Physiology* 64, 1480–1485.

Ivy, J.L., Lee, M.C., Brozinick, J.T., et al. (1988b) Muscle glycogen storage after different amounts of carbohydrate ingestion. *Journal of Applied Physiology* 65, 2018–2023.

Ivy, J.L., Goforth, H.W., Damon, B.D., et al. (2002). Early post-exercise muscle glycogen recovery is enhanced with a carbohydrate–protein supplement. *Journal of Applied Physiology* 93, 1337–1344.

Jentjens, R.L., van Loon, L.J.C., Mann, CH., et al. (2001) Addition of protein and amino acids to carbohydrates does not enhance postexercise muscle glycogen synthesis. *Journal of Applied Physiology* 91, 839–846.

Keizer, H.A., Kuipers, H., van Kranenburg, G., et al. (1987) Influence of liquid and solid meals on muscle glycogen resynthesis, plasma fuel hormone response, and maximal physical work capacity. *International Journal of Sports Medicine* 8, 99–104.

Kleinert, M., Liao, Y.-H., Nelson, J.L., et al. (2011) An amino acid mixture enhances insulin-stimulated glucose uptake in rat epitrochlearis muscle. *Journal of Applied Physiology* 111, 163–169.

Kuo, C.-H., Browning, K.S., & Ivy, J.L. (1999) Regulation of GLUT4 protein expression and glycogen storage after prolonged exercise. *Acta Physiologica Scandinavica* 165, 193–202.

Levenhagen, D.K., Gresham, J.D., Carlson, M.G., et al. (2001) Postexercise nutrient intake timing in humans is critical to recovery of leg glucose and protein homeostasis. *American Journal of Physiology, Endocrinology and Metabolism* 280, E982–E993.

Meyer, F., Heilmeyer, L.M. Jr, Haschke, R.H., et al. (1970) Control of phosphorylase activity in a muscle glycogen particle. I. Isolation and

characterization of the protein-glycogen complex. *Journal of Biological Chemistry* 245, 6642–6648.

Morifuji, M., Kanda, A., Koga, J., et al. (2010) Post-exercise carbohydrate plus whey protein hydrolysates supplementation increases skeletal muscle glycogen level in rats. *Amino Acids* 38, 1109–1115.

Nilsson, L.H. & Hultman, E. (1974) Liver and muscle glycogen in man after glucose and fructose infusion. *Scandinavian Journal of Clinical and Laboratory Investigation* 33, 5–10.

Parkin, J.A.M., Carey, M.F., Martin, I.K., et al. (1997) Muscle glycogen storage following prolonged exercise: effect of timing of ingestion of high glycemic index food. *Medicine & Science in Sports & Exercise* 29, 220–224.

Pedersen, D.J., Lessard, S.J., Coffey, V.G., et al. (2008) High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coingested with caffeine. *Journal of Applied Physiology* 105, 7–13.

Piehl, K. (1974) Time course for refilling of glycogen stores in human muscle fibers following exercise-induced glycogen depletion. *Acta Physiologica Scandinavica* 90, 297–302.

Piehl-Aulin, K., Söderlund, K., & Hultman, E. (2000) Muscle glycogen resynthesis rate in humans after supplementation of drinks containing carbohydrates with low and high molecular masses. *European Journal of Applied Physiology* 81, 346–351.

Prats, C., Helge, J.W., Nordby, P., et al. (2009) Dual regulation of muscle glycogen synthase during exercise by activation of compartmentalization. *Journal of Biological Chemistry* 284, 15692–15700.

Prats, C., Gomez-Cabello, A., & Hansen, A.V. (2011) Intracellular compartmentalization of skeletal muscle glycogen metabolism and insulin signaling. *Experimental Physiology* 96, 385–390.

Price, T.B., Rothman, D.L., Taylor, R., et al. (1994) Human muscle glycogen resynthesis after exercise: insulin-dependent and -independent phases. *Journal of Applied Physiology* 76, 104–111.

Reed, M.J., Brozinick, J.T., Lee, M.C., et al. (1989) Muscle glycogen storage postexercise: effect of mode of carbohydrate administration. *Journal of Applied Physiology* 66, 720–726.

Richter, E.A., Garetto, L.P., Goodman, N.M., et al. (1984) Enhanced muscle glycogen metabolism after exercise: modulation by local factors. *American Journal of Physiology, Endocrinology and Metabolism* 246, E476–E482.

Ruby, B.C., Gaskill, S.E., Slivka, D., et al. (2004) The addition of fenugreek extract (*Trigonella foenum-graecum*) to glucose feeding increases muscle glycogen resynthesis after exercise. *Amino Acids* 28, 71–76.

Sherman, W.M., Costill, D.L., Fink, W.J., et al. (1981) The effect of exercise and diet manipulation on muscle glycogen and its subsequent utilization during performance. *International Journal of Sports Medicine* 2, 114–118.

Sherman, W.M., Costill, D.L., Fink, W.J., et al. (1983) Effect of a 42.2-km footrace and subsequent rest or exercise on muscle glycogen and enzymes. *Journal of Applied Physiology* 55, 1219–1224.

Sherman, W.M., Doyle, J.A., Lamb, D.R., et al. (1993) Dietary carbohydrate, muscle glycogen, and exercise performance during 7 d of training. *American Journal of Clinical Nutrition* 57, 27–31.

Shi, X., Summers, R.W., Schedl, H.P., et al. (1995) Effects of carbohydrate type and concentration and solution osmolality on water absorption. *Medicine & Science in Sports & Exercise* 27, 1607–1615.

Starling, R.D., Trappe, T.A., Parcell, A.C., et al. (1997) Effects of diet on muscle triglyceride and endurance performance. *Journal of Applied Physiology* 82, 1185–1189.

van Den Bergh, A.J., Houtman, S., Heerschap, A., et al. (1996) Muscle glycogen recovery after exercise during glucose and fructose intake monitored by ^{13}C-NMR. *Acta Physiologica Scandinavica* 70, 129–142.

van Hall, G., Shirreffs, S.M., & Calbert, J.A.L. (2000) Muscle glycogen resynthesis during recovery from cycle exercise: no effect of additional protein ingestion. *Journal of Applied Physiology* 88, 1631–1636.

van Loon, L.J.C., Saris, W.H.M., Kruijshoop, M., et al. (2000) Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *American Journal of Clinical Nutrition* 72, 106–111.

Wallis, G.A., Hulston, C.J., Mann, C.H., et al. (2008) Postexercise muscle glycogen synthesis with combined glucose and fructose ingestion. *Medicine & Science in Sports & Exercise* 40, 1789–1794.

Wee, S.L., Williams, C., Tsintzas, K., et al. (2005) Ingestion of a high-glycemic index meal increases muscle glycogen storage at rest but augments its utilization during subsequent exercise. *Journal of Applied Physiology* 99, 707–714.

Zawadzki, K.M., Yaspelkis, B.B., & Ivy, J.L. (1992) Carbohydrate-protein complex increases the rate of muscle glycogen storage after exercise. *Journal of Applied Physiology* 72, 1854–1859.

Chapter 9

Carbohydrate Ingestion During Exercise

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Introduction

Carbohydrate and fat are the two important fuels during exercise, and their relative contribution is dependent on a number of factors including the pre-exercise carbohydrate stores, the exercise intensity and duration, and the training status of the subject (Jeukendrup, 2003). However, during intense exercise (and thus most competitive situations), carbohydrate is the critical fuel and in particular, muscle glycogen has been linked to exercise performance. Originally, studies investigated the role of high muscle glycogen stores at the onset of exercise (carbo-loading) on exercise performance and recently, research has focused more on the potential role of carbohydrate ingested just before and during exercise. Although the exact mechanisms are still not entirely clear, it has been known for some time that carbohydrate ingestion during exercise can increase exercise capacity and improve exercise performance (for review, see Jeukendrup, 2004, 2008; Jeukendrup & Tipton, 2009). Since then, studies have investigated the effects of different feeding regimens, different types of carbohydrate, and different amounts of carbohydrate in order to improve the recommendations. This review will focus on these recent studies and will be limited to the role of carbohydrate ingested during exercise.

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High Intensity Exercise, 30–75 minute Duration

In an early study, we were surprised to demonstrate that carbohydrate intake during all-out exercise of 60 minutes duration resulted in improvements in performance (Jeukendrup et al., 1997). In this study, 17 out of 19 cyclists performed better in a time trial when they ingested a carbohydrate solution. This was surprising because of the short duration of the exercise and the expectation that only about 15 grams of carbohydrate would have been available to the working muscle. This was not the only study with such findings. Similar findings were reported by others using different designs but similar durations and intensities of exercise (Anantaraman et al., 1995; Below et al., 1995). So there appear to be several studies with similar findings to the study by Jeukendrup et al. (1997). However, it must be noted that there are also some studies that did not observe a difference in performance with carbohydrate feeding. There are several possible explanations for the differences between the studies that did and did not find a positive effect on performance. Although it is interesting that the majority of the studies that did not report an effect actually observed a positive effect on performance, which did not reach statistical significance. It could, therefore, be argued that the negative findings are a result of the lack of statistical power. However, there may be another reason why the conclusions of these studies are different as will be discussed a little later.

In summary, it seems that the majority of studies observed a performance improvement when

carbohydrate is ingested during high intensity exercise lasting ~1 hour and it is unlikely that the cause of this improvement is related to energy delivery to the working muscle.

Mechanisms

In order to study the potential role of carbohydrate as a fuel during this type of exercise, cyclists were asked to perform a 40-km time trial. On one occasion, they were infused with a glucose solution and on another occasion saline was infused (Carter et al., 2004b). It was observed that when glucose was infused, blood glucose concentrations were twice as high and glucose disappearance (Rd glucose), representing glucose uptake, has also doubled (Carter et al., 2004b). However, although most of the glucose was presumably taken up into the muscle and oxidized, there was no effect on performance (Carter et al., 2004b). This provides evidence that the effects of carbohydrate during this type of exercise are not metabolic and that there must be an alternative explanation for the ergogenic effect.

In a follow-up study, cyclists were asked to repeat the 40-km time trial, but only to rinse their mouth with a carbohydrate solution without swallowing it (Carter et al., 2004a). The carbohydrate used in this study was a nonsweet tasteless maltodextrin solution. The rinsing protocol was standardized; subjects rinsed their mouth for 5 seconds with the drink and then spat the drink out into a bowl. The results were remarkable: performance was improved with the carbohydrate mouth rinse compared with placebo and the magnitude of the effect was the same as we had seen in the early study with carbohydrate ingestion (Jeukendrup et al., 1997). Despite the fact that no carbohydrate had been absorbed, performance was improved by 2.8% (Carter et al., 2004a), very similar to the 2.3% improvement observed with carbohydrate feeding (Jeukendrup et al., 1997). These findings were confirmed in other studies. Rollo et al. (2008) demonstrated that mouth rinsing with a carbohydrate solution increased total distance covered during a self-selected 30-minute run in comparison with a color- and taste-matched placebo. Similar results were obtained during a 60-minute self-paced run (Rollo et al., 2009). Similar findings were reported by others (Chambers et al., 2009; Fares & Kayser, 2011; Pottier et al., 2008), although not all studies were positive (Beelen et al., 2009; Whitham & McKinney, 2007). It was suggested that an effect is more likely observed when a period of fasting preceded the performance measurement. However, a recent study, in which a direct comparison was made between fasted and fed conditions, found similar performance-enhancing effects of a carbohydrate mouth rinse (Fares & Kayser, 2011).

So overall, although not all studies confirm the findings, the effect of a carbohydrate mouth rinse is rather convincing and seems to be present in an exercise lasting 30–60 minutes. It is unlikely that these effects are present during shorter exercise periods (less than 30 minutes) (Jeukendrup et al., 2008) and it is unlikely that the mouth-rinse effect (central effect) can overrule some of the other factors that cause fatigue during more prolonged exercise (more than 2 hours).

Brain Responses to Oral Carbohydrate

In the follow-up studies conducted at the University of Birmingham, functional magnetic resonance imaging (fMRI) was used to investigate the responses of the human brain to a carbohydrate or placebo mouth rinse (Chambers et al., 2009). The study revealed that tasting both a sweet (glucose) and non-sweet (maltodextrin) carbohydrate solution activated the areas of brain, such as the anterior cingulate cortex and the ventral striatum, which were unresponsive to an artificial sweetener (saccharin). Other neuroimaging investigations have also reported that the presence of a carbohydrate solution in the mouth activates additional brain regions compared with an artificial sweetener, suggesting there may be "taste transduction" pathways that respond to carbohydrate independently of those for sweetness. The name "taste receptor" may, therefore, be a misnomer as it seems to be the carbohydrate that is detected and the effects are independent of taste.

Implications

These results suggest that it is not necessary to consume large amounts of carbohydrate during

exercise lasting ~30–75 minutes and a mouth rinse with carbohydrate may be sufficient to get a performance benefit. In most conditions, the performance effects with the mouth rinse were similar to those of ingesting the drink, so there does not seem to be a disadvantage of taking the drink, although occasionally, athletes may complain of gastrointestinal distress when consuming relatively large amounts of fluid. Of course when the exercise is more prolonged (2 hours or more), carbohydrate becomes a very important fuel and it is essential to ingest carbohydrate. Future research will lead to a better understanding of the role of carbohydrate receptors and the underlying mechanisms of observed performance improvements after oral carbohydrate sensing.

Intermittent and Skill Sports

A vast majority of studies have been performed with endurance athletes performing continuous exercise. Most team sports have a highly intermittent nature with bursts of very high intensity exercise followed by relatively low intensity recovery periods. Besides this, performance in these sports is often dependent on other factors than maintenance of speed or power, and factors like agility, timing, motor skill, decision making, jumping, and sprinting may all play a role. Nevertheless, carbohydrate ingestion during exercise has also been shown to enhance endurance capacity in intermittent activities. A large number of studies have demonstrated that if carbohydrate is ingested during intermittent running, fatigue can be delayed and time to exhaustion can be increased. For example, Nicholas et al. (1995) demonstrated that the time to exhaustion during a Loughborough Intermittent Shuttle Test (LIST) was increased compared with placebo when a carbohydrate–electrolyte solution was consumed. This exercise protocol was designed to mimic the intermittent nature of soccer (Nicholas et al., 2000). In this study, time to exhaustion was improved but there was no difference in sprint performance, probably, because sprinting is unlikely to be dependent on exogenous carbohydrate availability. Since this study, several studies have confirmed improvements in running to exhaustion, although only a few studies showed an effect on sprint performance as well.

The suggested mechanism for the improvements with intermittent exercise is a reduced net breakdown of muscle glycogen (Nicholas et al., 1995). Nicholas et al. (1995) showed a 22% reduction in net muscle glycogen utilization during a 90-minute LIST. It is possible that carbohydrate intake reduces glycogenolysis but it is equally likely that some glycogen synthesis does occur during the phases of low intensity exercise. Ultimately, because of a reduced net breakdown, glycogen concentrations are higher during the final phases of a game and as a result, endurance capacity is increased.

So there are plenty of evidences that time to exhaustion is increased with carbohydrate feeding during intermittent exercise. However, one could argue that increased time to exhaustion has little to do with improved performance in team sports where motor skills, coordination, and timing are perhaps more important determinants of performance than endurance capacity. Therefore, more recently, studies have incorporated measurements of skill into their performance measurements. Currell et al. (2009) developed a 90-minute soccer simulation protocol that included measurements of skill such as agility, dribbling, shooting, and heading. The soccer players performed 90 minutes of intermittent exercise that mimicked their movement patterns during a game. During the 90 minutes, skill performance measurements were performed at regular intervals. Agility, dribbling, and accuracy of shooting were all improved but heading was not affected with carbohydrate ingestion. Ali et al. (2007) performed a study in which trained game players performed an extended LIST (including skill tests) with or without carbohydrate intake, and in this study, shooting performance was better maintained toward the end but there was no effect of carbohydrate on passing skills. Winnick et al. (2005) used a modified LIST and found improved motor skills during the final 30 minutes. In a further study, Russell et al. (2012) reported that carbohydrate ingestion attenuated decrements in shooting performance during simulated soccer match play. Other studies did not find differences in agility, passing, or a motor skill test. Carbohydrate feeding during exercise has also been

shown to reduce fatigue and improve skill performance in a number of other sports. For example, one recent study showed that tennis players spent more time in moderate-intensity activity and less time in low intensity activity with carbohydrate ingestion compared to placebo (McRae & Galloway, 2012). Performance analysis also revealed that carbohydrate ingestion increased overall serve success and success of first serves as well as serves to the advantage side. Return success was greater during the second set of the match with carbohydrate feeding. An earlier study by Vergauwen et al. (1998) also concluded that carbohydrate supplementation improves stroke quality during the final stages of prolonged tennis play. In badminton, the long serve was better maintained when carbohydrate was ingested (Bottoms et al., 2012). The mechanisms behind the improvements in skill are unknown and have not been studied in any detail.

It appears that carbohydrate intake during team sports and other sports, with an element of skill, has the potential to improve not only fatigue resistance but also the skill components of a sport, especially toward the end of a game. The practical challenge is often to find ways to ingest carbohydrate during a game within the rules of the sport.

Ergogenic Effects of Carbohydrate Intake During Prolonged Exercise (>2 hours)

The beneficial effects of carbohydrate feeding on exercise performance have been well documented. In early studies, the positive effects of carbohydrate feeding were typically seen during exercise lasting at least 2 hours (Jeukendrup, 2008). Most of these studies investigated endurance capacity (measured as time to exhaustion at a fixed (constant) exercise intensity). A few studies, however, observed positive effects with time trial protocols in which the cyclists had to complete a fixed distance as fast as possible. The mechanism behind the ergogenic effect is most likely related to a greater contribution of exogenous carbohydrate (carbohydrate ingested in beverages or other foods), sparing of liver glycogen, prevention of hypoglycemia, and maintaining high rates of carbohydrate oxidation necessary to sustain exercise intensity. The effects of carbohydrate intake during prolonged exercise (>2 hours) have been described elsewhere in more detail and the reader is referred to these reviews (Jeukendrup, 2004, 2008).

Optimizing the Carbohydrate Delivery

Since the first studies showing that carbohydrate ingested during exercise can enhance performance, there has been a search for the optimum dose, amount, and type of carbohydrate to come up with better recommendations. The recommendations, however, remained very general until recently. For example, in 2007, the American College of Sports Medicine (ACSM) recommendations (Sawka et al., 2007) stated that athletes should aim for an intake of 30–60 g of carbohydrate per hour and did not differentiate between activities or duration of events. Another set of guidelines was produced in 2009 with similar conclusions (Rodriguez et al., 2009). These recommendations came without clear guidelines on the type of carbohydrate or the form in which it should be ingested. Recent studies have given insights into the type of carbohydrate that should be ingested and recommendations can be much more specific (Jeukendrup, 2011). These new recommendations were discussed in Lausanne in 2010 at an IOC Nutrition Consensus Conference and the guidelines were generally accepted and adopted (Jeukendrup, 2011). In the following sections, the background to these new recommendations will be discussed in more detail.

Different Types of Carbohydrate

Isotopic labeling of carbohydrates has been used to study the efficacy of various carbohydrates. This enabled the investigators to describe the time course of oxidation of a carbohydrate and also compare the oxidation of different carbohydrates. When carbohydrates are ingested from the onset of exercise and at regular intervals thereafter, oxidation of the ingested carbohydrate increases and typically reaches a plateau after 60–90 minutes. Originally, carbohydrates like glucose, fructose, galactose, sucrose, maltose, and glucose polymers

were studied, and it was found that fructose and galactose were oxidized at slightly lower rates than glucose, sucrose, maltose, maltodextrins, or amylopectin-type starches (for review see Jeukendrup, 2008). This was explained by differences in the rates of intestinal absorption as well as the fact that fructose and galactose have to be converted to glucose in the liver before they can be oxidized in the muscle. Until recently, it was believed that carbohydrate ingested during exercise could be oxidized at a rate no higher than 1 g/min (60 g/h) (for detailed review see Jeukendrup, 2004, 2008; Jeukendrup & Tipton, 2009). This is reflected in guidelines by the ACSM in 2007 which state that athletes should take between 30 and 60 grams of carbohydrate (Sawka et al., 2007). However, in recent years, it has repeatedly been demonstrated that the ingestion of multiple transportable carbohydrates can result in exogenous carbohydrate oxidation rates well in excess of 1 g/min. Intakes greater than 90 g/h (1.5 g/min) are less practical and might cause gastrointestinal discomfort in some athletes. Therefore, recommendations are usually in the 1.5 g/min range and not the 2.4 g/min that was used in some of the laboratory studies.

Multiple Transportable Carbohydrates

Although the observation of a maximum exogenous carbohydrate oxidation rate of 1 g/min seemed uniform and generally accepted, the reasons for this apparent ceiling were unclear. By way of elimination, it was concluded that the most likely reason for the limitation might be intestinal absorption (Jeukendrup, 2008). Glucose is absorbed through a sodium-dependent glucose transporter protein called SGLT1. This transport protein in the brush border membrane has a high affinity for glucose and galactose, but not for fructose. We hypothesized that the limitation for exogenous carbohydrate oxidation was a saturation of the SGLT1 transporters in brush border membrane of the intestine, which may occur at high rates of glucose ingestion. Fructose absorption is not affected by this because it is absorbed independently by a sodium-independent transporter: GLUT5. The combined ingestion should, therefore, result in an increased total delivery of carbohydrates into the circulation and increased oxidation by the muscle. In a study by Jentjens et al. (2004), evidence was obtained that exogenous carbohydrate oxidation was also enhanced. In this study, subjects exercised for 3 hours at a moderate intensity in a randomized crossover design and ingested isoenergetic amounts of either glucose or glucose:fructose. The oxidation rates in the glucose trials peaked around 0.8 g/min whereas the oxidation rates with glucose:fructose peaked at 1.26 g/min. The studies were extended by studying different carbohydrate mixes such as glucose:sucrose: fructose, glucose:sucrose, and maltodextrin (MD):fructose, and different ingestion rates. In summary, these studies show that when multiple transportable carbohydrates are ingested at high rates (90–144 g/h), oxidation rates can be increased up to 1.75 g/min. There does not seem to be an optimal ratio of carbohydrates, but it is essential to saturate the SGLT1 transporter by providing glucose, maltose, maltodextrins, or starch at a rate of 60–70 g/h and any additional fructose which uses a different transporter will result in even higher exogenous carbohydrate oxidation rates.

Effects of Multiple Transportable Carbohydrates on Exercise Performance

In subsequent studies, more practical but still quite large amounts of carbohydrate were ingested by the subjects (1.5 g/min) and it was observed that the subjects' ratings of perceived exertion (RPE) tended to be lower with the mixture of glucose and fructose compared with glucose alone, and cyclists were able to maintain their cadence better toward the end of 5 hours cycling (Jeukendrup et al., 2006). Rowlands et al. (2008) also reported reduced fatigue when ingesting a maltodextrin:fructose mix. It was also demonstrated that a glucose:fructose drink could improve exercise performance (Currell & Jeukendrup, 2008). Cyclists exercised for 2 hours on a cycle ergometer at 54% VO_2 max during which they ingested either a carbohydrate drink or placebo and were then asked to perform a time trial that lasted approximately another 60 minutes. The results were astounding. When the subjects ingested a glucose drink (at 1.8 g/min), they improved their power

output by 10% (254W vs. 231W). However, when they ingested glucose:fructose, there was another 8% improvement of the power output over and above the improvement by glucose ingestion (275W vs. 254W). Although numerous studies had demonstrated the beneficial metabolic effects, this was the first study to show that exogenous carbohydrate oxidation rates may be linked to performance and the first study to demonstrate a clear performance benefit with glucose:fructose compared with glucose (Currell & Jeukendrup, 2008).

A more recent work focused on the effects of high rates of mixed carbohydrate ingestion on gastric emptying and fluid delivery. Again the results were remarkable. Gastric emptying measured by either a gastric tube or by using a ^{13}C-acetate tracer was found to be improved with a glucose:fructose mixture compared with glucose. Also, fluid delivery has been shown to be improved with glucose:fructose compared with glucose in a number of studies.

Training the Gut

It is important to note that in order to benefit from a glucose:fructose mixture, it may be necessary to saturate glucose transporters in the intestine by ingesting sufficient quantities. When carbohydrate is ingested at rates of 0.8 g/min and saturation may not occur, ingesting part of this carbohydrate as fructose may not result in higher exogenous carbohydrate oxidation rates (Hulston et al., 2009). However, although glucose was ingested at very low rates in one of the trials (0.54 g/min plus 0.26 g/min of fructose), the oxidation rates for both carbohydrate drinks were the same! This indicates that in some cases glucose:fructose may be an advantage (at high ingestion rates) but there is no disadvantage when it is ingested at lower rates.

If the recommendation is to ingest carbohydrate at relatively high rates, the practical implication is that the athlete will have to be able to tolerate such intakes. Although high intakes have sometimes been linked with gastrointestinal discomfort, anecdotal evidence would suggest that the gut is trainable and that individuals who regularly consume carbohydrate or have a high daily carbohydrate intake have an increased capacity to empty carbohydrate from the stomach and absorb it (Jeukendrup & McLaughlin, 2011). From animal studies, it would appear that this is indeed the case. It has been demonstrated that the intestinal carbohydrate transporters can be upregulated by exposing an animal to a high carbohydrate diet.

A recent study by Cox et al. (2010) investigated whether altering daily carbohydrate intake affects substrate oxidation and in particular exogenous carbohydrate oxidation. It was hypothesized that when exposed to a high carbohydrate diet for a prolonged period of time (28 days), carbohydrate transporters in the intestine would be upregulated and this would result in an increase in exogenous carbohydrate oxidation during exercise. So, essentially, the adaptation to a high carbohydrate diet would allow an athlete to use ingested carbohydrate more rapidly in the working muscle. In order to study this, the investigators recruited 16 subjects and divided them into a high carbohydrate and a low carbohydrate group. Both groups were fed a diet containing 5 g/kg carbohydrate per day but the high carbohydrate group received supplements providing an additional 1.5 g/kg carbohydrate per day. Before and after the 28-day period, exogenous carbohydrate oxidation was measured during a 100-minute steady state trial around 70% VO_2 peak. During the exercise bout, the subjects received a 10% glucose solution at 20-minute intervals providing almost 2 g of carbohydrate per minute. Exogenous carbohydrate oxidation rates were higher after the high carbohydrate diet, providing evidence that the gut is indeed adaptable and this can be used as a practical method to increase exogenous carbohydrate oxidation. We recently suggested that this may be highly relevant to the endurance athlete and may be a prerequisite for the first person to break the 2-hour marathon barrier (Stellingwerff & Jeukendrup, 2011). Here I introduce the term "nutritional training" to indicate that within the training program of an endurance athlete, specific workouts need to be scheduled that aim to "train the gut." The suggestion is to include one workout per week with the goal to match or exceed the planned carbohydrate intake for race day in order to practice the strategy and give the gut a chance to adapt. Future studies will have to investigate the number and

frequency of nutritional training workouts that are needed to obtain such adaptations.

Better Oxidation Efficiency and Less Gastrointestinal Discomfort

The term *oxidation efficiency* was introduced to describe the percentage of the ingested carbohydrate that is oxidized (Jeukendrup & Jentjens, 2000). High oxidation efficiency means that smaller amounts of carbohydrate remain in the gastrointestinal tract, reducing the risk of causing gastrointestinal discomfort that is frequently reported during prolonged exercise. Therefore, compared to a single source of carbohydrate, ingesting multiple carbohydrate sources results in a smaller amount of carbohydrate remaining in the intestine, and osmotic shifts and malabsorption may be reduced. This probably means that drinks with multiple transportable carbohydrates are less likely to cause gastrointestinal discomfort. Interestingly, this has been a consistent finding in studies that have attempted to evaluate gastrointestinal discomfort during exercise (Jeukendrup, 2008). Subjects tended to feel less bloated with the glucose plus fructose drinks compared to drinking glucose-only solutions. It should be noted that fructose on its own is known to cause gastrointestinal problems. However, in the presence of glucose, these effects seem to disappear, most likely because glucose aids the fructose intestinal transport process and vice versa.

Individual Differences

There appears to be no correlation between body mass and exogenous carbohydrate oxidation. Although the absolute numbers are difficult to compare between studies because of differences in the carbohydrate composition, the duration of exercise, the amount of carbohydrate ingested, and the environmental conditions, it is safe to conclude that studies showed no correlation between body mass and exogenous carbohydrate oxidation (measured during the final hour of exercise). The reason is probably that the limiting factor is carbohydrate absorption, and absorption is largely independent of body mass. It is likely that the absorptive capacity of the intestine is determined, or at least strongly influenced, by carbohydrate content of the diet as it has been shown in animal studies that intestinal transporters are upregulated with increased carbohydrate intake. Since exogenous carbohydrate is independent of body mass or muscle mass, but dependent on absorption and to some degree power output, the advice on carbohydrate intake given to athletes should be in absolute amounts. These results clearly show that there is no rationale for expressing carbohydrate recommendations for athletes per kilogram body mass, at least for consumption during exercise.

Individual differences in exogenous carbohydrate oxidation exist, although they are generally small. These differences are not related to body mass but more likely to a capacity to absorb carbohydrates. This in turn could be diet-related.

Practical Implications

Based on the recent findings, the guidelines by ACSM (Rodriguez et al., 2009) may have to be adjusted in certain situations, in particular, during very prolonged exercise. A preferred carbohydrate intake for such events, provided that the energy expenditure is high enough, may be 90 g/h (Jeukendrup, 2004, 2008; Jeukendrup & Tipton, 2009) (Figure 9.1). Interestingly, studies in the 1980s with Tour de France cyclists already showed a high carbohydrate intake of >90 g/h (Saris et al., 1989). Whether these findings translate to other sports remains to be seen but many athletes in long distance triathlon races seem to adopt a similar strategy (Pfeiffer et al., 2011). We tested whether the recommended high carbohydrate intake (in the form of a carbohydrate gel) could be tolerated during a 16-km running race and concluded that high carbohydrate gel intake, regardless of the blend (glucose vs. glucose:fructose), was surprisingly well tolerated (Pfeiffer et al., 2009). However, it must also be noted that certain individuals could not cope with the high intake and this stresses the need for an individualized feeding strategy that takes into account personal preferences as well as tolerance.

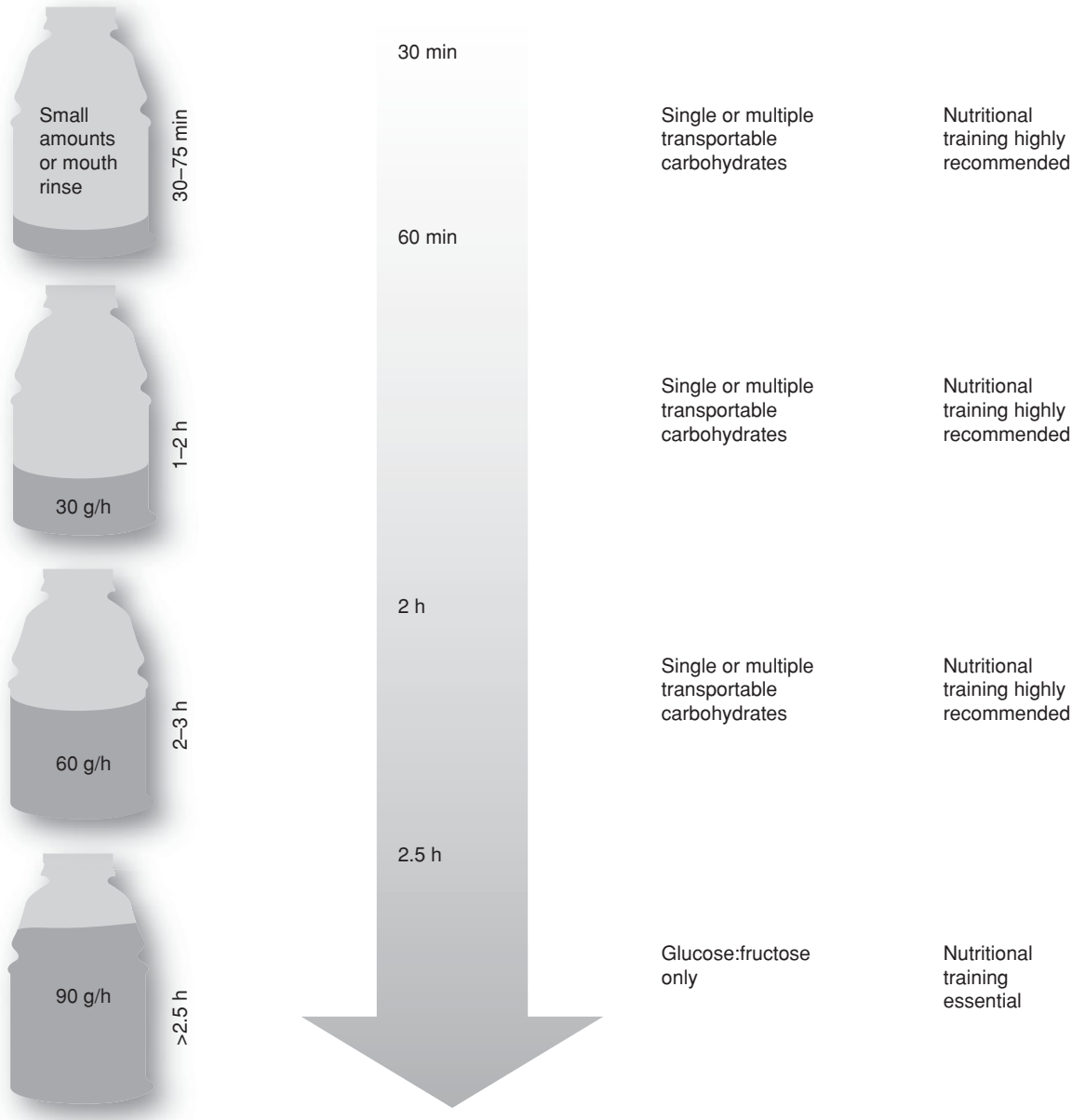

Figure 9.1 Carbohydrate intake recommendations during exercise for exercise of different durations. These values are for high level athletes and should be adjusted downward for aspiring athletes.

Conclusion

Carbohydrate intake during exercise has been studied extensively since the 1980s, but more recent advances have resulted in more sophisticated guidelines and recommendations. It is clear that carbohydrate intake during exercise has the potential to influence performance in events lasting as little as 30–75 minutes and the effects can be very substantial during more prolonged exercise. It is also clear that carbohydrate intake can affect the performance

in intermittent sports and sports with skill components. Recommendations are dependent on the level of the athlete and the duration of the event. In general, a higher carbohydrate intake is recommended for high level athletes and longer durations. For short duration events, low intakes up to 30 g/h may be sufficient, whereas during very prolonged events, recommended intakes may be up to 90 g/h. With such high intakes, multiple transportable carbohydrates are recommended and the athletes will have to accustom themselves to such intakes.

Summary

Carbohydrate feeding has been shown to be ergogenic, but recently, substantial advances have been made in optimizing the guidelines for carbohydrate intake during prolonged exercise. The guidelines have become more specific to the athlete and the type and duration of activity (summarized in Figure 9.1). Although carbohydrate ingestion during exercise seems to be ergogenic during a number of events that last 30 minutes or longer, the mechanisms may be quite different during various activities. During all-out, high intensity exercise of 30–75 minutes duration, the effects may be through carbohydrate receptors in the oral cavity that modify the central command and result in increased power output. Carbohydrate ingestion has also been shown to increase fatigue resistance in intermittent (team) sports and improve motor skills and/or decision making. During more prolonged exercise, carbohydrate provides an additional fuel which becomes especially important when endogenous carbohydrate stores become depleted. Recent studies suggest a dose–response relationship with higher carbohydrate intakes resulting in better performance, especially during events that are 2.5 hours or longer. Therefore, attempts have been made to optimize the delivery of carbohydrate to the working muscle. It has become clear that limitations to exogenous carbohydrate oxidation were in the absorptive process, most likely because of a saturation of carbohydrate transporters. By using a combination of carbohydrates that uses different intestinal transporters for absorption, it was shown that carbohydrate delivery and oxidation could be increased. Studies demonstrated increases in exogenous carbohydrate oxidation rates up to 65% of glucose:fructose compared with glucose only. Exogenous carbohydrate oxidation rates reach values of 1.75 g/min whereas previously 1 g/min was considered as the absolute maximum. The increased carbohydrate oxidation with multiple transportable carbohydrates was accompanied by increased fluid delivery and improved oxidation efficiency and thus the likelihood of gastrointestinal distress may be diminished. Studies also demonstrated reduced fatigue and improved exercise performance with multiple transportable carbohydrates compared with a single carbohydrate. Multiple transportable carbohydrates, ingested at high rates (90 g/h recommended), can be beneficial during endurance sports where the duration of exercise is 2.5 hours or more.

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References

Ali, A., Williams, C., Nicholas, C.W., et al. (2007) The influence of carbohydrate-electrolyte ingestion on soccer skill performance. *Medicine & Science in Sports & Exercise* 39, 1969–1976.

Anantaraman, R., Carmines, A.A., Gaesser, G.A., et al. (1995) Effects of carbohydrate supplementation on performance during 1 hour of high-intensity exercise. *International Journal of Sports Medicine* 16, 461–465.

Beelen, M., Berghuis, J., Bonaparte, B., et al. (2009) Carbohydrate mouth rinsing in the fed state: lack of enhancement of time-trial performance. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 400–409.

Below, P.R., Mora-Rodríguez, R., Gonzáles-Alonso, J., et al. (1995) Fluid and carbohydrate ingestion independently improve performance during 1 h of intense exercise. *Medicine & Science in Sports & Exercise* 27, 200–210.

Bottoms, L., Sinclair, J., Taylor, K., et al. (2012) The effects of carbohydrate ingestion on the badminton serve after fatiguing exercise. *Journal of Sports Sciences* 30, 285–293.

Carter, J.M., Jeukendrup, A.E., & Jones, D.A. (2004a) The effect of carbohydrate mouth rinse on 1-h cycle time trial

performance. *Medicine & Science in Sports & Exercise* 36, 2107–2111.
Carter, J.M., Jeukendrup, A.E., Mann, C. H. et al. (2004b) The effect of glucose infusion on glucose kinetics during a 1-h time trial. *Medicine & Science in Sports & Exercise* 36, 1543–1550.
Chambers, E.S., Bridge, M.W., & Jones, D.A. (2009) Carbohydrate sensing in the human mouth: effects on exercise performance and brain activity. *Journal of Physiology* 587, 1779–1794.
Cox, G.R., Clark, S.A., Cox, A.J., et al. (2010) Daily training with high carbohydrate availability increases exogenous carbohydrate oxidation during endurance cycling. *Journal of Applied Physiology* 109, 126–134.
Currell, K. & Jeukendrup, A.E. (2008) Superior endurance performance with ingestion of multiple transportable carbohydrates. *Medicine & Science in Sports & Exercise* 40, 275–281.
Currell, K., Conway, S., & Jeukendrup, A.E. (2009) Carbohydrate ingestion improves performance of a new reliable test of soccer performance. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 34–46.
Fares, E.J. & Kayser, B. (2011). Carbohydrate mouth rinse effects on exercise capacity in pre- and postprandial States. *Journal of Nutrition and Metabolism* 2011, 385962.
Hulston, C.J., Wallis, G.A., & Jeukendrup, A.E. (2009). Exogenous CHO oxidation with glucose plus fructose intake during exercise. *Medicine & Science in Sports & Exercise* 41, 357–363.
Jentjens, R.L., Moseley, L., Waring, R.H., et al. (2004) Oxidation of combined ingestion of glucose and fructose during exercise. *Journal of Applied Physiology* 96, 1277–1284.
Jeukendrup, A.E. (2003) Modulation of carbohydrate and fat utilization by diet, exercise and environment. *Biochemical Society Transactions* 31, 1270–1273.
Jeukendrup, A.E. (2004) Carbohydrate intake during exercise and performance. *Nutrition* 20, 669–677.
Jeukendrup, A.E. (2008) Carbohydrate feeding during exercise. *European Journal of Sport Science* 8, 77–86.
Jeukendrup, A.E. (2011) Nutrition for endurance sports: marathon, triathlon, and road cycling. *Journal of Sports Sciences* 29(Suppl. 1), S91–S99.
Jeukendrup, A.E. & Jentjens, R. (2000) Oxidation of carbohydrate feedings during prolonged exercise: current thoughts, guidelines and directions for future research. *Sports Medicine* 29, 407–424.
Jeukendrup, A.E. & McLaughlin, J. (2011) Carbohydrate ingestion during exercise: effects on performance, training adaptations and trainability of the gut. *Nestle Nutrition Institute Workshop Series* 69, 1–17.
Jeukendrup, A. & Tipton, K.D. (2009) Legal nutritional boosting for cycling. *Current Sports Medicine Reports* 8, 186–191.
Jeukendrup, A., Brouns, F., Wagenmakers, A.J., et al. (1997) Carbohydrate-electrolyte feedings improve 1 h time trial cycling performance. *International Journal of Sports Medicine* 18, 125–129.
Jeukendrup, A.E., Moseley, L., Mainwaring, G.I., et al. (2006) Exogenous carbohydrate oxidation during ultraendurance exercise. *Journal of Applied Physiology* 100, 1134–41.
Jeukendrup, A.E., Hopkins, S., Aragon-Vargas, L.F., et al. (2008) No effect of carbohydrate feeding on 16 km cycling time trial performance. *European Journal of Applied Physiology* 104, 831–837.
McRae, K.A. & Galloway, S.D. (2012) Carbohydrate-electrolyte drink ingestion and skill performance during and after 2 hr of indoor tennis match play. *International Journal of Sport Nutrition and Exercise Metabolism* 22, 38–46.
Nicholas, C.W., Williams, C., Lakomy, H.K., et al. (1995) Influence of ingesting a carbohydrate-electrolyte solution on endurance capacity during intermittent, high-intensity shuttle running. *Journal of Sports Sciences* 13, 283–90.
Nicholas, C.W., Nuttall, F.E., & Williams, C. (2000) The Loughborough Intermittent Shuttle Test: a field test that simulates the activity pattern of soccer. *Journal of Sports Sciences* 18, 97–104.
Pfeiffer, B., Cotterill, A., Grathwohl, D., et al. (2009) The effect of carbohydrate gels on gastrointestinal tolerance during a 16-km run. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 485–503.
Pfeiffer, B., Stellingwerff, T., Hodgson, A. B., et al. (2012) Nutritional intake and gastrointestinal problems during competitive endurance events. *Medicine & Science in Sports & Exercise* 44, 344–351.
Pottier, A., Bouckaert, J., Gilis, W., et al. (2010) Mouth rinse but not ingestion of a carbohydrate solution improves 1-h cycle time trial performance. *Scandinavian Journal of Medicine and Science in Sports* 20, 105–111.
Rodriguez, N.R., Di Marco, N.M., & Langley, S. (2009) American College of Sports Medicine position stand. Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41, 709–731.
Rollo, I., Williams, C., Gant, N., et al. (2008) The influence of carbohydrate mouth rinse on self-selected speeds during a 30-min treadmill run. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 585–600.
Rollo, I., Cole, M., Miller, R., et al. (2010) Influence of mouth rinsing a carbohydrate solution on 1-h running performance. *Medicine & Science in Sports & Exercise* 42, 798–804.
Rowlands, D.S., Thorburn, M.S., Thorp, R.M., et al. (2008) Effect of graded fructose coingestion with maltodextrin on exogenous 14C-fructose and 13C-glucose oxidation efficiency and high-intensity cycling performance. *Journal of Applied Physiology* 104, 1709–1719.
Russell, M., Benton, D., & Kingsley, M. (2012) Influence of carbohydrate supplementation on skill performance during a soccer match simulation. *Journal of Science and Medicine in Sport/Sports Medicine Australia* 15, 348–354.
Saris, W.H., van Erp-Baart, M.A., Brouns, F., et al. (1989) Study on food intake and energy expenditure during extreme sustained exercise: the Tour de France. *International Journal of Sports Medicine* 10, S26–S31.
Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39(2), 377–390.
Stellingwerff, T. & Jeukendrup, A.E. (2011) Authors reply to viewpoint by Joyner et al. entitled "The Two-Hour Marathon: Who and When?" *Journal of Applied Physiology* 110, 278–293.
Vergauwen, L., Brouns, F., & Hespel, P. (1998) Carbohydrate supplementation improves stroke performance in tennis. *Medicine & Science in Sports & Exercise* 30, 1289–1295.
Whitham, M. & McKinney, J. (2007) Effect of a carbohydrate mouthwash on running time-trial performance. *Journal of Sports Sciences* 25, 1385–1392.
Winnick, J.J., Davis, J.M., Welsh, R.S., et al. (2005) Carbohydrate feedings during team sport exercise preserve physical and CNS function. *Medicine & Science in Sports & Exercise* 37, 306–315.

Chapter 10

Defining Optimum Protein Intakes for Athletes

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Protein was once believed to be the macronutrient of primary importance in fueling activity but we now know that is not the case. Nonetheless, the importance of protein is still paramount. Currently, a large difference of opinion exists between those who establish dietary protein requirements for the general population (Institute of Medicine, 2005) and those issuing guidelines for athletes (Rodriguez et al., 2009). A relatively consistent consensus is that healthy but relatively sedentary adults need no more than 0.8–0.9 g protein/kg/day to satisfy their protein needs (Institute of Medicine, 2005; WHO Technical Report Series 935, 2011). In contrast, recent reviews are available in which recommendations for dietary protein in athletes are assessed and scrutinized (Rodriguez et al., 2009) and the general consensus from these reviews is that the protein needs of athletes are higher than those of sedentary persons. The recommended intake from these reviews varies but is generally between 1.2 and 1.7 g protein/kg/day (Rodriguez et al., 2009).

It is unclear whether there is a compromise between these points of view. The goal of this chapter is to try to reconcile the apparent disparity of opinion over whether or not athletes have elevated needs for dietary protein. A relevant point relates not to an absolute "*need*" or "*requirement*" for dietary protein, as it is defined by body nitrogen balance, but whether athletes could derive some benefit from additional dietary protein over and above the RDA. Such a protein intake might be considered an "optimum protein intake" based more on maintenance/gain of the functional capacity of tissues such as muscle and maintenance of tissues such as bone, ligaments, and tendons. A central tenet of this argument is that tissues such as muscle, bone, tendon, and ligaments may not be fully accounted for in short-term measures of nitrogen balance owing to their much slower rates of protein turnover (in some cases 50-fold lower) by comparison to gut (Nakshabendi et al., 1995) or plasma proteins. The result is that short-term nitrogen balance, and even indicator amino acid experiments, will be dominated by rapidly turning-over tissues. Finally, an important point for the athlete and the coach is what intakes of protein might become "excessive" and a potential risk for compromising health or athletic performance. In this chapter, these issues are viewed in the light of macronutrient displacement as well as chronic health problems.

Current Recommendations

The requirement for protein is set for normal populations by the method of nitrogen balance. Nitrogen, since it is the essential nuclide of amino acids, is used and the assumption of balanced intake with excretion means that all protein-requiring processes are sufficiently supplied with amino acids and operating at their full capacity (Institute of Medicine, 2005; WHO Technical Report Series 935, 2011). There are, however, other caveats of the current nitrogen balance method that are summarized in the following

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quotation from the WHO report (WHO Technical Report Series 935, 2011): "…the protein requirement of adults can be defined as the minimum intake that will allow nitrogen equilibrium (zero nitrogen balance), at an appropriate body composition during energy balance and at moderate physical activity. [underline added] In practice, the nitrogen balance studies that formed the basis of the previous (1985) and present reports involve studies on healthy adults assumed to be in energy balance, usually on the basis of weight maintenance and of an appropriate body composition, but without specific measurement to ensure that this was the case. [underline added]" The main point of relevance for athletes and their coaches is that not all of these assumptions have been tested in sedentary person, let alone athletes, and are also often clearly not valid in athletic populations. For example, athletes often purposely alter their body weight and composition into and out of ranges that may be considered "appropriate" by WHO standards. Moreover, athletes are certainly not always engaging in moderate physical activity. Also of relevance to athletes is the admission by the WHO that body composition was not even measured in the studies they used to derive the estimate for protein requirements.

The apparent incongruence between those deriving dietary guidelines versus athletes and sports practitioners for protein requirements may well be the focus on the recommended dietary allowance or RDA (in the United States and Canada), the recommended dietary intakes or RDI (in Australia), and the reference nutrient intake (RNI) in the United Kingdom, which while held as the protein intake "target" by some was actually never intended as such. These estimates of requirement establish the level of protein that will replace losses and thus prevent deficiency. Such a protein intake would not necessarily be optimal but instead would be the minimum consistent with health. The methodology used in establishing the protein RDA is nitrogen balance (Institute of Medicine, 2005; WHO Technical Report Series 935, 2011). Use of nitrogen balance is an adequate method for establishing nitrogen or amino acid requirements necessary to prevent deficiency and achieve a balance of nitrogen in weight-stable individuals who have relatively low levels of physical activity, as per the definition outlined above. It is, however, likely that the same method is inadequate to establish intakes of dietary that are optimal for maximizing gains in muscle mass and strength induced by resistance training or adaptations in metabolic function induced by resistance or endurance training, or for preserving lean mass during periods of rapid weight loss. An interesting concept in this regard is one of "anabolic drive," in which deposition of protein during growth determines need. However, for athletes, at least in adulthood, none is truly growing so instead the requirement for protein would be to not only optimize the rate of replacement of proteins being broken down but also optimize adaptive processes. In short, defining requirements in terms of preventing deficiency would, from an athlete's perspective, hardly be considered a position from which to frame their "requirement" for dietary protein.

The flaws of using nitrogen balance to determine protein requirements have been recognized for some time. As a method for determining protein needs, nitrogen balance is a "blind" process since it does not provide any insight into the kinetics of body proteins but instead merely provides information on the net balance of intake versus excretion. Could such a method provide adequate enough information for athletes to determine how much protein they not only require, but also need to optimize adaptation? The methodological limitations of nitrogen balance include (i) implausibly high nitrogen balances typically observed at high protein intakes (this implies, of course, that higher protein intakes are associated with greater nitrogen retention); (ii) an increase in the economy of nitrogen use at low protein intakes; and (iii) often estimated rather than measured dermal and miscellaneous obligatory losses of nitrogen (Institute of Medicine, 2005; WHO Technical Report Series 935, 2011). From an athletic perspective it is also important to realize that regardless of whether or not nitrogen balance is achieved at a particular protein intake, it is possible that the level of protein consumed may be less than that required to optimize all aspects of the protein-requiring processes. In other words, adaptive changes in protein metabolism can occur to reduce protein utilization at the expense of a less than opti-

mal rate of protein synthesis of one particular protein or another. This point is made with the recognition that in short-term nitrogen balance studies (note that in the studies analyzed, which were used to set the requirements for protein in the current RDA, the mean study duration was only 10–15 days) it seems unlikely that adaptations in muscle, bone, and connective tissues will be captured. This is because the rates of turnover of proteins in bone (Babraj et al., 2002), tendon (Heinemeier et al., 2003), and skeletal muscle tissues vary between 0.6% and 1.2% per day (Rennie et al., 2004). In contrast, the rates of protein turnover for more labile tissues are 48% per day for ileal protein (Nakshabendi et al., 1995); even plasma proteins such as albumin, fibrinogen, and fibronectin have turnover rates of between 10% and 30% per day. Thus, within the context of a 10-day-long nitrogen balance study the chance of detecting changes in tissue turnover in musculoskeletal tissues or a decline in tissue mass, let alone any change in the function of that tissue, is highly unlikely. Importantly, at marginal protein intakes nitrogen equilibrium can be attained by adaptive and potentially accommodative downregulation of amino acid-requiring processes (Young, 1986), which may not be maladaptive in sedentary persons, but may not be optimal for an athlete. In addition, it needs to be appreciated that as individuals adapt to less than adequate protein intakes they do so by lowering nitrogen excretion (Young, 1986) such that there is no apparent relationship between nitrogen balance and musculoskeletal tissue protein synthetic rates, and eventually mass, for reasons outlined above. An important point for athletes is that there is no relation between nitrogen balance and muscle function be it strength, power, or aerobically based, which is a critical measure for athletes but one that has never been measured in the context of studies of protein adequacy. Long-term studies would, of course, be needed to address the previous two points.

More importantly from an athlete's perspective is the idea of whether protein intakes higher than the RDA translate into improved performance or potentially prevent declines in performance. This is an important consideration if we are to make arguments directed at optimizing physiological function based on protein intakes that would likely exceed the RDA, namely, is there benefit to consumption of protein at levels higher than the RDA and, if so, how much higher? Also, are there considerations beyond simply daily protein intake with respect to general health or athletic performance? These are difficult questions to answer.

Optimizing Protein Intake

A better model for setting protein intake recommendations for athletes may well come from examination of acute protein turnover studies in which a framework to understand protein deposition has been described (Phillips, 2004; Phillips et al., 2005). In this model, acute periods of positive muscle protein balance have been theorized to accumulate, over a period of time, to explain how muscle hypertrophy occurs (Phillips, 2004; Phillips et al., 2005). Accepting that not all athletes wish to achieve muscle hypertrophy, it is still a practical approach to trying to understand the optimization of protein intake as opposed to simply balancing nitrogen in a whole-body sense. This is because all athletes need to remodel proteins and so need to engage in the maximum possible rate of protein renewal to maintain optimum function of all proteins. In the model proposed (Phillips, 2004; Phillips et al., 2005), two variables dictate whether muscle protein is lost or gained, and they are muscle protein synthesis (MPS) and muscle protein breakdown (MPB). The algebraic difference between MPS and MPB yields net protein balance (NPB). A variety of studies, too numerous to list but reviewed in detail in Chapter 11, show that resistance exercise stimulates not only MPS, but also MPB, whereas consumption of protein after resistance exercise causes a further stimulation of MPS and prevents the normal rise in MPB resulting in a positive NPB and protein accumulation (for reviews see Phillips, 2004; Phillips et al., 2005). While the evidence is not as extensive for endurance-type exercise as it is for resistance exercise, there are still studies that show this form of exercise also stimulates protein synthesis (Breen et al., 2011; Wilkinson et al., 2008), but more so for mitochondrial as opposed to myofibrillar proteins (Breen et al., 2011; Wilkinson

et al., 2008). The situation of endurance exercise is difficult to assess because in this case the stimulus is not anabolic and does not ultimately result in a net accumulation of muscle contractile protein mass (as is the case with resistance exercise). Instead, the argument often given is that extra protein for endurance athletes is required because endurance exercise increases amino acid oxidation (Lamont et al., 2001). However, the only amino acid oxidized to a substantial extent during exercise would appear to be leucine (Lamont et al., 2001). The argument has, however, been based on an average human body-tissue leucine content of 590 μmol/g protein, that is if x amount of leucine is oxidized during an exercise bout then $x/590$ is equivalent to the number of grams of tissue protein broken down to provide that leucine. Such a calculation relies, however, on a number of very tenuous assumptions that are not tested in most experimental paradigms and have never shown to be true. It is possible that the increased leucine oxidation during endurance exercise may mean an increased need for dietary leucine and not necessarily an increased need for dietary protein. In a practical sense, however, unless leucine supplements are ingested, an increased dietary leucine requirement would represent an increased need to ingest protein (especially those containing leucine, such as lean meats and dairy proteins like whey) in endurance athletes.

Figure 10.1a shows a schematic representation of our current understanding of how muscle NPB is increased throughout a given day by protein ingestion. Figure 10.1b shows how fed-state gains are increased and fasted-state losses are lessened with the performance of resistance exercise. Finally, Figure 10.1c shows how further protein meals could be added throughout a day to increase the periods of positive protein balance leading to an even greater increase in muscle mass. It is important to realize that the same model could easily be applied to tissues such as bone (Babraj et al., 2002) and tendon (Miller et al., 2005), the protein synthetic rates of which are also amplified by feeding and exercise. Thus even if accretion of tissue is not the goal, as is shown in Figure 10.1 for muscle, then at least a case could be made for feeding an optimum amount of protein at each meal to maximize the synthetic response to repair and remodel proteins within any protein-containing structure. If we accept this position then one can begin, from a muscle protein perspective (i.e., the tissue for which we have data), to estimate an optimum protein intake to maximize MPS. We have defined protein intakes that maximally stimulate protein synthesis in younger men at ~20–25 g of protein per meal following resistance exercise (Moore et al., 2009), an estimate that is not incongruent with what young men at rest require (Cuthbertson et al., 2005). We estimate that one can likely consume such a protein intake five to six times daily or 125–150 g at most. Why is this? Why not simply continually feed and eat higher doses? The first point is that there needs to be some time in between meals to allow a "resetting" of the protein synthetic machinery in between meals. This assumption is based on the findings of a study by Bohe et al. (2001), which showed that a sustained aminoacidemia actually results in the process of MPS being turned off. Thus, discrete meals are going to be more effective in periodically stimulating MPS rather than "continuous" consumption of protein. At the same time, larger protein meals would not contribute to the synthetic response and instead serve merely to increase amino acid oxidation and urea production (Moore et al., 2009). Thus if the suppositions of 125–150 g protein/day are correct, a maximum daily protein requirement for a 90 kg, resistance-trained male athlete would be about 1.4–1.7 g protein/kg/day, which is not far from what is recommended (Rodriguez et al., 2009).

One could envision that such an intake might be required to support a number of processes in an athlete, such as to support an athlete's ability to repair and replace any damaged proteins (resulting potentially from oxidative stress, protein nitrosylation, or mechanical disruption); to efficiently remodel proteins in structures such as muscle, bone, tendon, and ligaments to better withstand the stress and strain imposed by training and competition; to maintain optimum function of all metabolic pathways in which amino acids are participatory intermediates (which includes being oxidative fuels); to support increments in lean mass, if desired; to support an optimally functioning immune system; and to support the optimum rate of production of all plasma

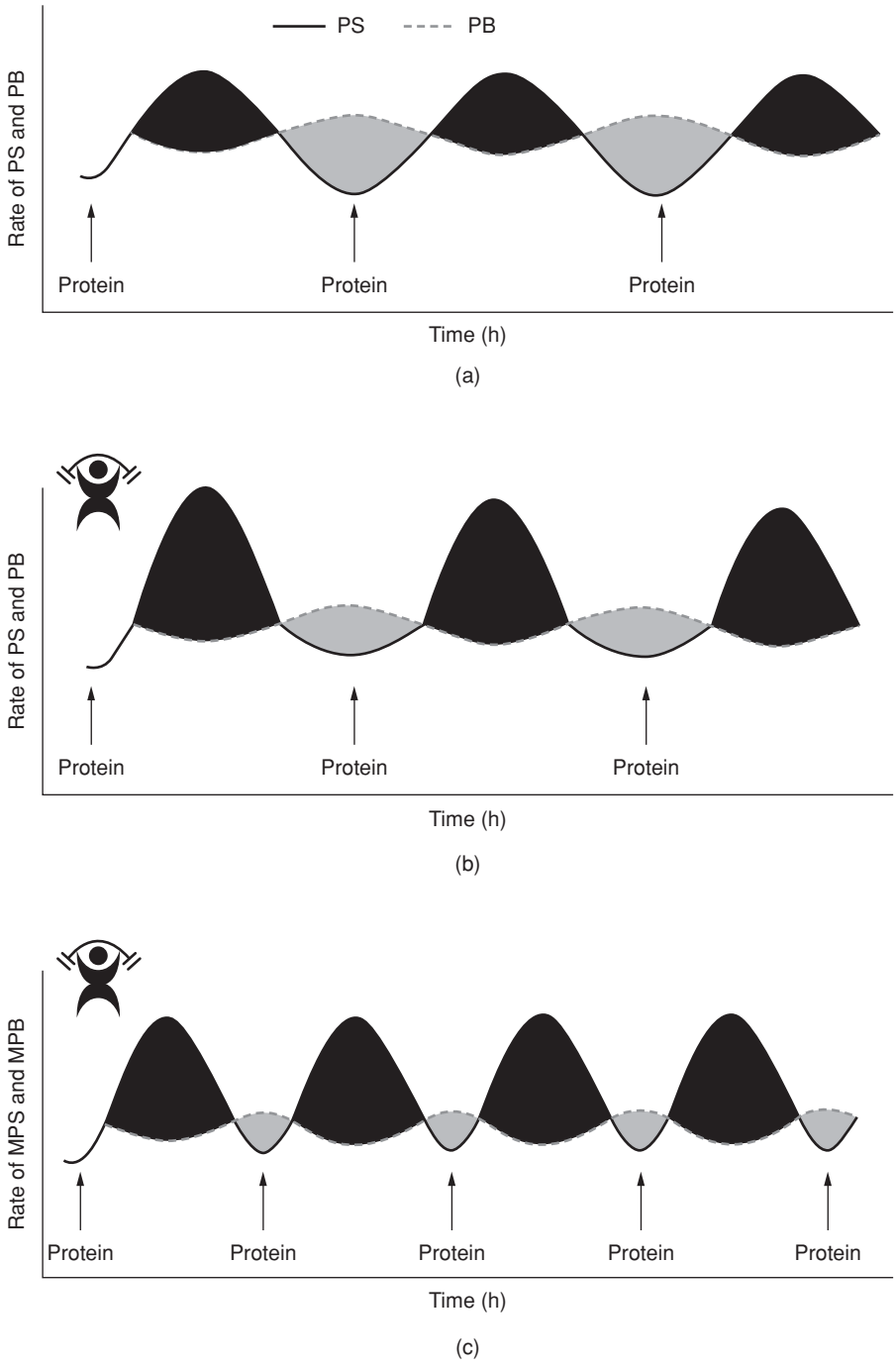

Figure 10.1 Schematic representation of changes in muscle protein synthesis (PS) and muscle protein breakdown (PB) induced by diurnal feeding. A positive net protein balance is shown in black and a net loss in grey (a). (b) shows the effect of adding a bout of resistance exercise on the same three protein-containing meals as in panel (a). (c) shows the effect of adding an additional protein-containing meal in the same time period as the situation depicted in (b).

proteins required for physiological function. This last point is emphasized since we have shown that the same protein requirement kinetics for maximizing MPS (i.e., 20–25 g) were also true of albumin protein synthesis (Moore et al., 2009). Ultimately, the answer to the question of how much protein is required to, for example, support optimum immune system function or to optimize flux through intermediary amino acid-requiring metabolic pathways is difficult to answer directly. Thus, the position of many athletes is to consume large quantities of protein so as to maximize the rates of protein synthesis and "cover all their bases." An important question is whether such a practice is beneficial or whether it leads to athletes getting too much protein? From the standpoint of what constitutes "too much" protein this could be from a health perspective or from a performance perspective.

Excessive Protein Intake

A relevant question is whether protein intakes can become "excessive" or sufficiently high to cause harm to an athlete or to compromise their performance. It is hard to define from a health standpoint what would constitute an excessive protein intake. One could assume that this is any intake greater than the RDA, but there is limited evidence that habitual consumption of protein in amounts greater than the RDA would lead to health problems. One main argument as to why protein intakes could become excessive is based on a well-recognized clinical argument that patients at a variety of stages of chronic kidney disease are placed on low-protein diets to alleviate the progression of their disease (De Santo et al., 2011). Through the corollary argument the notion then is that a high-protein diet contributes to a decline in kidney function; however, reviews in this area have been unable to establish a link, causal or otherwise, between higher protein intakes and a progression of chronic kidney disease (Calvez et al., 2012; Robertson et al., 2007). For example, many strength athletes habitually consume protein intakes in excess of 2–2.5 g protein/kg/day without any adverse consequences and certainly without developing kidney disease. Northern Canadian and Alaskan Inuit also have lifetime protein intakes of close to 3 g protein/kg/day but are also not a population in whom kidney disease is common. Certainly, preexisting kidney disease is a cause for concern and high protein intakes would not be recommended for this group; but, for most athletes who are in good health a higher than normal protein intake would not be a concern insofar as kidney disease is concerned. In short-term clinical trials, egg white, dairy, and soya protein consumption in high quantities did not affect renal function. Bernstein et al. (2007) noted that "From these studies, it is difficult to conclude whether or not there is a long-term association between amount of animal or vegetable protein intake and change in normal renal function." A review by Friedman (2004) reached a similar conclusion that people with healthy, functioning kidneys are not at risk with a high-protein diet, but does provide some guidelines that could be used in determining a person's risk for progression of kidney disease that may be useful for athletes and coaches who are concerned with this issue.

Another argument as to why protein intakes should not be too high is their potential impact on bone. Some reports have shown increased calciuria with higher protein intakes and a subsequent increased risk for bone fracture or osteoporosis (Feskanich et al., 1996); however, several studies now exist supporting a contrary position (Wengreen et al., 2004). In fact, the relationship between protein and bone health has recently been highlighted to be a positive one; that is, the more dietary protein consumed, the greater the peak bone mass achieved so long as dietary calcium is adequate (reviewed in Bonjour, 2005). The mechanism underpinning the greater bone mass with higher intakes of dietary protein appears to be mediated through a protein-induced increase in circulating levels of IGF-1 (Bonjour, 2005). Increased protein intake may also interact with the high forces generated during resistive-type activities, which are potent stimuli for increasing IGF-I (both systemically and locally) (Nindl et al., 2001), to further increase peak bone mass. Thus high dietary protein intakes do not appear to threaten peak bone mass in athletes or in the general population.

One potential downside to a higher protein diet is the diet-induced upregulation of the catabolic path-

ways for amino nitrogen disposal and amino acid oxidation that occurs on adjustment to higher protein intakes (Quevedo et al., 1994). This is because there is no storage "reserve" for protein consumed in excess of that which can immediately be used for protein synthesis and other amino acid-requiring processes. The nitrogen from amino acids is still fundamentally toxic in mammalian systems and so requires rapid and efficient excretion (Quevedo et al., 1994). With limited experimental evidence, it has been argued that athletes with a habitually high protein intake risk, should they ever suddenly reduce their habitually high protein intake, would succumb to excessive catabolism of ingested protein (Quevedo et al., 1994) and so risk loss of lean mass. Thus, while fed-state gains might be higher on a high-protein diet, fasted-state losses would appear to be equally high (Quevedo et al., 1994). In an acute experimental paradigm, such a thesis may well be true; however, what we lack is the direct experimental evidence that shows what happens to athletic performance or lean mass in the transition from a high- to a low-protein diet.

Perhaps the strongest argument against higher protein diets would be that of displacement of other macronutrients and specifically carbohydrate. Since carbohydrate is the preferred fuel for all athletic events, even short, high-intensity events, then practices that compromise stored muscle glycogen would be counterintuitive for optimizing performance (for examples, see Chapter 7). To restore glycogen, estimated carbohydrate requirements for athletes have ranged from as little as 3–5 g carbohydrate/kg/day up to as high as 10–12 g carbohydrate/kg/day (Burke et al., 2004). It is unlikely, at least at the very high end of the suggested carbohydrate intakes, that any athlete other than highly competitive triathletes, runners, or cyclists would require such intakes to maintain the ability to train and perform (Burke et al., 2004). Thus, an important question to consider is when lower carbohydrate intakes would begin to compromise performance, and at what specific level. The answer is likely to be sport and training specific, but it needs to be stressed that even high-intensity, short-duration muscular efforts (i.e., sprinting and lifting) rely heavily on carbohydrate (see Chapter 7). Given that resistance training/power lifting athletes tend to consume high protein intakes, such individuals may be at greater risk for lower than optimum carbohydrate intakes to support the most intense training effort possible. Data from MacDougall et al. (1999) showed that with three sets of biceps curls (8–10 reps per set) performed at a weight providing 80% of the subjects' single repetition maximum load (1 RM), muscle glycogen concentration is reduced by almost 35% from starting levels. Similar results have been obtained by others (Tesch et al., 1986). These results provide some support for the idea that carbohydrate is an important and potentially limiting substrate even during resistance exercise workouts (Tesch et al., 1986). Nevertheless, from a practical standpoint athletes need to consider, in a sport-specific manner, their postexercise carbohydrate intake, in addition to their protein intake, to optimize performance.

If taken to extremes higher dietary protein intakes would, unless weight gain were a desired goal, have to displace another dietary macronutrient. If it is dietary lipid that is displaced, the outcomes are not likely to be of great concern. If, however, the increased consumption of dietary protein results in a lower dietary carbohydrate intake, performance could be compromised. This may be a situation of greater concern if the athlete has voluntarily assumed an energy deficit to change his or her body weight and composition.

Energy Intake

Assuming energy balance is a desired goal, an increased energy intake is needed to balance exercising energy expenditure; nevertheless, additional protein intake need not be overly high to achieve nitrogen balance. An interesting concept in this regard is the one of available dietary energy highlighted by the interesting work of Loucks et al. (2011). This is particularly true if the increased energy comes from carbohydrate (Richardson et al., 1979), which owing to the ability of this substrate to stimulate insulin release can markedly suppress proteolysis, consequently improving nitrogen balance (Borsheim et al., 2004). However, as previously stated, most athletes are not seeking nitrogen balance (i.e., simply getting enough protein to offset

nitrogen loss) but instead are looking for an optimum protein intake. It is worth noting that, even in the complete absence of protein intake following exercise, leg muscle protein balance can be brought to levels not different from zero (i.e., no net loss or gain of proteins) simply with the ingestion of carbohydrates alone (Borsheim et al., 2004). A recent study involved people confined to a metabolic ward who were overfed 40% more than their estimated energy requirement or 950 kcal/day (range 884–1022 kcal/day) (Bray et al., 2012). The composition of the diets these subjects overconsumed was either 5% (low protein), 15% (normal protein), or 25% (high protein) protein. Overeating produced significantly less weight gain in the low-protein diet group (3.16 kg) than in the normal protein diet group (6.05 kg) or the high-protein diet group (6.51 kg). However, body fat content increased similarly in all three protein diet groups and represented 50% (high protein) to more than 90% (low protein) of the excess stored energy (Figure 10.2). Concurrently, resting energy expenditure, total energy expenditure, and body protein did not increase during overfeeding with the low-protein diet. By contrast, resting energy expenditure increased by 160 kcal/day in the normal protein diet and by 227 kcal/day in the high-protein diet. Lean mass increased only in the normal protein diet (2.87 kg) and the high-protein diet (3.18 kg) highlighting the capacity for protein ingestion-induced gains in lean mass that one can only theorize would have been greater with concurrent performance of resistive exercise

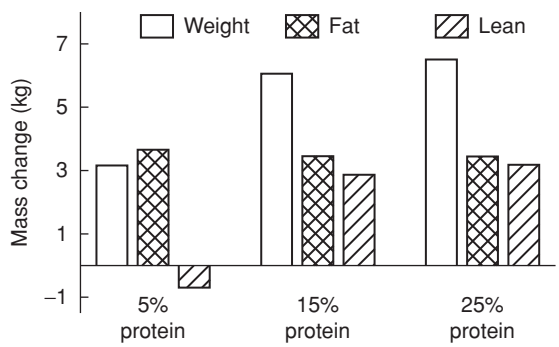

Figure 10.2 Changes in body weight, fat, and leans mass in people fed excess energy (950 kcal/day) for 8 weeks. Data are from Bray et al. (2012).

(Hartman et al., 2007). These data also provide good evidence to suggest that excess energy contributes to weight gain, but that the composition of the gain is affected by the composition of what is overeaten; specifically, overconsumption of fat and carbohydrates promotes only fat gain, whereas protein promotes lean mass gain.

There are studies in which a marked fat loss have occurred concomitant with a "sparing" and even gains in muscle mass through induction of an energy deficit with varying macronutrient ratios (Josse et al., 2011a). The reader may wish to refer a meta-analysis showing that, during hypoenergetic periods, lower carbohydrate (less than 40% of total energy) and higher protein (>1.05 g/kg/day) intakes result in increased fat mass loss and lean mass preservation, compared to diets higher in carbohydrate and lower in protein (Krieger et al., 2006). In addition, Layman et al. (2005) showed that a hypoenergetic diet containing lower carbohydrate and higher protein (carbohydrate-to-protein ratio of 1.6) combined with the addition of primarily endurance, but also some resistive, exercise appeared to be the most effective strategy for promoting fat loss and preserving lean mass. This finding may not be surprising when one considers that endurance exercise, to a greater degree (Sheffield-Moore et al., 2004), and resistance exercise, to a lesser degree (Phillips et al., 1999), are anabolic in that they stimulate MPS even in the fasted state, forcing an increased net "conservation" of amino acids arising from proteolysis. While simultaneous lean mass gain with fat mass loss is a promise from all personal trainers, the existence of such a phenomenon in published literature reports is sparse. For example, in a review of protein sparing by both diet and exercise (Weinheimer et al., 2010) it was reported that only a handful (<5%) of studies have reported simultaneous fat mass loss and lean mass gain both with exercise and higher protein energy-restricted diets (Weinheimer et al., 2010).

Recently, we examined how a higher protein, higher dairy diet during weight loss affected the change not in body weight but in body composition as well as bone health (Josse et al., 2011b). We found that a diet containing 30% of energy from protein (1.3 g protein/kg/day) with six servings of dairy

per day was able to promote fat mass loss and lean mass gains that were greater than two other diets containing only 15% energy from protein (or about 0.8 g protein/kg/day) and either 0–1 or 3 servings of dairy per day in which subjects lost fat mass but it comprised 80% and 93% of their weight loss as opposed to lean mass gain in the higher protein, higher dairy group (Josse et al., 2011b). We also found that markers of bone health were improved only in the higher protein plus higher dairy group (Josse et al., 2012). Of course, our data do not permit a conclusion regarding the impact of quantity of protein versus quality of protein since the higher protein was exclusively from dairy sources.

In many sports it is recognized that a higher lean:fat body composition can translate into a competitive advantage. Thus, we concluded previously (Phillips, 2006) that a lower carbohydrate, higher protein, hypoenergetic diet, particularly when combined with exercise, is likely of substantial benefit for athletes if they wish to attain the associated performance advantage of modifying their body composition by losing stored body fat as opposed to muscle mass (Mettler et al., 2010). Of course, such a strategy is not without the obvious limitation that a lower carbohydrate intake in athletes will result in lower muscle glycogen stores (see Chapters 7 and 8). Thus, athletes who adhere to a lower carbohydrate and higher protein diet may be depriving themselves of the fuel that is by far the preferred substrate to power muscular contraction. Clearly, body composition change needs to occur in the lead-up to an athlete's competitive season so as to not adversely affect performance.

Timing of Protein Consumption

When it comes to the stimulation of new muscle protein accretion via resistance exercise it appears that immediate postexercise protein supplementation is beneficial. A review of studies in which protein was given as a supplement to subjects after exercise appears to agree with a general statement that the timing of protein consumption after exercise may be a determinant of muscle mass and strength gains. Although acute studies suggest that muscle is sensitive to the provision of nutrients (especially amino acids) for up to 3 hours after resistance exercise (Rasmussen et al., 2000), longitudinal training studies suggest that increases in strength and muscle mass are greatest when protein is consumed immediately after exercise (Esmarck et al., 2001; Holm et al., 2006). In addition, strength and muscle mass gains in patients who had just undergone knee surgery were promoted to a greater degree by protein and carbohydrate consumption than simply carbohydrate or a placebo (Holm et al., 2006). Gains in muscle fiber size were seen with young men training for 14 weeks only if they consumed protein after exercise versus isoenergetic carbohydrate (Andersen et al., 2005). Cribb and Hayes (2006) reported that ingestion of a supplement containing creatine and protein immediately before and after exercise resulted in greater lean mass gains, strength, and type II muscle fiber area than seen in a group who got the same supplement but at different times of the day. We reported that, in groups of young men (Hartman et al., 2007) and women (Josse et al., 2010), immediate postexercise consumption of either skim milk or isoenergetic carbohydrate resulted in greater gains in lean mass than the equivalent amount of protein as soya. Hence, it is proposed that our data (Hartman et al., 2007; Josse et al., 2010), taken together with previous data from chronic studies manipulating postexercise protein consumption (Esmarck et al., 2001; Holm et al., 2006), support the general thesis that immediate consumption of protein, particularly milk protein (Hartman et al., 2007), after resistance exercise serves to maximize exercise-induced increases in muscle mass. Furthermore, consumption of energy in the form of carbohydrate after a resistance exercise workout, when ingested without protein, results in lower gains in muscle mass when compared to protein (Hartman et al., 2007).

Practical Recommendations

To achieve peak performance, athletes need to monitor all aspects of their diet including protein and be acutely aware of the sport-specific requirements for energy. As presented here, protein intakes of 1.2–1.7 g protein/kg/day would appear to be advantageous in aiding athletes achieve optimum adaptation, repair, remodeling, and protein

turnover. This level is greater than the RDA/RDI/RNI, with the general proviso that these levels of protein were not designed to be targets for population consumption of protein but instead minimum estimates designed simply to alleviate deficiency. What is clear is that, as with the recommendations for carbohydrate, the timing of ingestion is very important. Put simply, protein should be consumed early during the postexercise recovery phase (i.e., immediately to 2 hour after exercise). Protein quality also appears to be important in maximizing the accretion of muscle proteins, so athletes would do well to focus on high-quality protein sources such as dairy protein, eggs, and lean meat. When athletes find it inconvenient to consume such protein sources then portable protein sources, particularly protein supplements, offer a practical alternative. The content of these protein supplements should be closely scrutinized by athletes for quality, however, since protein bars and drinks are highly heterogeneous in terms of their composition. The high-quality protein dose that appears to maximally stimulate MPS is close to 20–25 g, above which protein synthesis is not further stimulated, but increases in amino acid oxidation and urea synthesis may result. Attention to these guidelines should allow an athlete to achieve peak performance in a variety of sports without compromising any aspect of their health.

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References

Andersen, L.L., Tufekovic, G., Zebis, M.K., et al. (2005) The effect of resistance training combined with timed ingestion of protein on muscle fiber size and muscle strength. *Metabolism* 54, 151–156.

Babraj, J., Cuthbertson, D.J., Rickhuss, P., et al. (2002) Sequential extracts of human bone show differing collagen synthetic rates. *Biochemical Society Transactions* 30, 61–65.

Bernstein, A.M., Treyzon, L., & Li, Z. (2007) Are high-protein, vegetable-based diets safe for kidney function? A review of the literature. *Journal of the American Dietetic Association* 107, 644–650.

Bohe, J., Low, J.F., Wolfe R.R., et al. (2001) Latency and duration of stimulation of human muscle protein synthesis during continuous infusion of amino acids. *Journal of Physiology* 532, 575–579.

Bonjour, J.P. (2005) Dietary protein: an essential nutrient for bone health. *Journal of the American College of Nutrition* 24, 526S–536S.

Borsheim E., Cree, M.G., Tipton, K.D., et al. (2004) Effect of carbohydrate intake on net muscle protein synthesis during recovery from resistance exercise. *Journal of Applied Physiology* 96, 674–678.

Bray, G.A., Smith, S.R., De, J.L., et al. (2012) Effect of dietary protein content on weight gain, energy expenditure, and body composition during overeating: a randomized controlled trial. *The Journal of the American Medical Association* 307, 47–55.

Breen, L., Philp, A., Witard, O.C., et al. (2011) The influence of carbohydrate–protein co-ingestion following endurance exercise on myofibrillar and mitochondrial protein synthesis. *Journal of Physiology* 589, 4011–4025.

Burke, L.M., Kiens B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22, 15–30.

Calvez, J., Poupin, N., Chesneau, C., et al. (2012) Protein intake, calcium balance and health consequences. *European Journal of Clinical Nutrition* 66, 281–295.

Cribb, P.J. & Hayes, A. (2006) Effects of supplement timing and resistance exercise on skeletal muscle hypertrophy. *Medicine & Science in Sports & Exercise* 38, 1918–1925.

Cuthbertson, D., Smith, K., Babraj, J., et al. (2005) Anabolic signaling deficits underlie amino acid resistance of wasting, aging muscle. *FASEB Journal* 19, 422–424.

De Santo, N.G., Perna, A., & Cirillo, M. (2011) Low protein diets are mainstay for management of chronic kidney disease. *Frontiers in Bioscience (Scholar Edition)* 3, 1432–1442.

Esmarck, B., Andersen, J.L., Olsen, S., et al. (2001) Timing of postexercise protein intake is important for muscle hypertrophy with resistance training in elderly humans. *Journal of Physiology* 535, 301–311.

Feskanich, D., Willett, W.C., Stampfer M.J., et al. (1996) Protein consumption and bone fractures in women. *American Journal of Epidemiology* 143, 472–479.

Friedman, A.N. (2004) High-protein diets: potential effects on the kidney in renal health and disease. *American Journal of Kidney Diseases* 44, 950–962.

Hartman, J.W., Tang, J.E., Wilkinson, S.B., et al. (2007) Consumption of fat-free fluid milk after resistance exercise promotes greater lean mass accretion than does consumption of soy or carbohydrate in young, novice, male weightlifters. *American Journal of Clinical Nutrition* 86, 373–381.

Heinemeier, K., Langberg, H., Olesen, J.L., et al. (2003) Role of TGF-beta1 in relation to exercise-induced type I collagen synthesis in human tendinous tissue. *Journal of Applied Physiology* 95, 2390–2397.

Holm, L., Esmarck, B., Mizuno, M., et al. (2006) The effect of protein and carbohydrate supplementation on strength training outcome of rehabilitation in ACL patients. *Journal of Orthopaedic Research* 24, 2114–2123.

Institute of Medicine (2005) *Dietary Reference Intakes for Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein,*

and Amino Acids. National Academies Press, Washington, DC.

Josse, A.R., Tang, J.E., Tarnopolsky, M.A., et al. (2010) Body composition and strength changes in women with milk and resistance exercise. *Medicine & Science in Sports & Exercise* 42, 1122–1130.

Josse, A.R., Atkinson, S.A., Tarnopolsky, M.A., et al. (2011a) Increased consumption of dairy foods and protein during diet- and exercise-induced weight loss promotes fat mass loss and lean mass gain in overweight and obese premenopausal women. *Journal of Nutrition* 141, 1626–1634.

Josse, A.R., Azizian, H., French, S.B., et al. (2011b) Body fat content determination in premenopausal, overweight, and obese young women using DXA and FT-NIR. *Obesity (Silver Spring)* 19, 1497–1502.

Josse, A.R., Atkinson, S.A., Tarnopolsky, M.A., et al. (2012) Diets higher in dairy foods and dietary protein support bone health during diet- and exercise-induced weight loss in overweight and obese premenopausal women. *Journal of Clinical Endocrinology and Metabolism* 97, 251–260.

Krieger, J.W., Sitren, H.S., Daniels, M.J., et al. (2006) Effects of variation in protein and carbohydrate intake on body mass and composition during energy restriction: a meta-regression. *American Journal of Clinical Nutrition* 83, 260–274.

Lamont, L.S., McCullough, A.J., & Kalhan, S.C. (2001) Relationship between leucine oxidation and oxygen consumption during steady-state exercise. *Medicine & Science in Sports & Exercise* 33, 237–241.

Layman, D.K., Evans, E., Baum, J.I., et al. (2005) Dietary protein and exercise have additive effects on body composition during weight loss in adult women. *Journal of Nutrition* 135, 1903–1910.

Loucks, A.B., Kiens, B., & Wright, H.H. (2011) Energy availability in athletes. *Journal of Sports Sciences* 29(Suppl. 1), S7–S15.

MacDougall, J.D., Ray, S., Sale, D.G., et al. (1999) Muscle substrate utilization and lactate production. *Canadian Journal of Applied Physiology* 24, 209–215.

Mettler, S., Mitchell, N., & Tipton, K.D. (2010) Increased protein intake reduces lean body mass loss during weight loss in athletes. *Medicine & Science in Sports & Exercise* 42, 326–337.

Miller, B.F., Olesen, J.L., Hansen, M., et al. (2005) Coordinated collagen and muscle protein synthesis in human patella tendon and quadriceps muscle after exercise. *Journal of Physiology* 567, 1021–1033.

Moore, D.R., Robinson, M.J., Fry, J.L., et al. (2009) Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. *American Journal of Clinical Nutrition* 89, 161–168.

Nakshabendi, I.M., Obeidat, W., Russell, R.I., et al. (1995) Gut mucosal protein synthesis measured using intravenous and intragastric delivery of stable tracer amino acids. *American Journal of Physiology* 269, E996–E999.

Nindl, B.C., Kraemer, W.J., Marx, J.O., et al. (2001) Overnight responses of the circulating IGF-I system after acute, heavy-resistance exercise. *Journal of Applied Physiology* 90, 1319–1326.

Phillips, S.M. (2004) Protein requirements and supplementation in strength sports. *Nutrition* 20, 689–695.

Phillips, S.M. (2006) Dietary protein for athletes: from requirements to metabolic advantage. *Applied Physiology, Nutrition, and Metabolism* 31, 647–654.

Phillips, S.M., Tipton, K.D., Ferrando, A.A., et al. (1999) Resistance training reduces the acute exercise-induced increase in muscle protein turnover. *American Journal of Physiology* 276, E118–E124.

Phillips, S.M., Hartman, J.W., & Wilkinson S.B. (2005) Dietary protein to support anabolism with resistance exercise in young men. *Journal of the American College of Nutrition* 24, 134S–139S.

Quevedo, M.R., Price, G.M., Halliday, D., et al. (1994) Nitrogen homoeostasis in man: diurnal changes in nitrogen excretion, leucine oxidation and whole body leucine kinetics during a reduction from a high to a moderate protein intake. *Clinical Science (London)* 86, 185–193.

Rasmussen, B.B., Tipton, K.D., Miller, S.L., et al. (2000) An oral essential amino acid-carbohydrate supplement enhances muscle protein anabolism after resistance exercise. *Journal of Applied Physiology* 88, 386–392.

Rennie, M.J., Wackerhage, H., Spangenburg, E.E., et al. (2004) Control of the size of the human muscle mass. *Annual Review of Physiology* 66, 799–828.

Richardson, D.P., Wayler, A.H., Scrimshaw, N.S., et al. (1979) Quantitative effect of an isoenergetic exchange of fat for carbohydrate on dietary protein utilization in healthy young men. *American Journal of Clinical Nutrition* 32, 2217–2226.

Robertson, L., Waugh, N., & Robertson, A. (2007) Protein restriction for diabetic renal disease. *Cochrane Database of Systematic Reviews* (Art. No.: CD002181).

Rodriguez, N.R., Di Marco, N.M., & Langley, S. (2009) American College of Sports Medicine position stand. Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41, 709–731.

Sheffield-Moore, M., Yeckel, C.W., Volpi, E., et al. (2004) Postexercise protein metabolism in older and younger men following moderate-intensity aerobic exercise. *American Journal of Physiology. Endocrinology and Metabolism* 287, E513–E522.

Tesch, P.A., Colliander, E.B., & Kaiser, P. (1986) Muscle metabolism during intense, heavy-resistance exercise. *European Journal of Applied Physiology and Occupational Physiology* 55, 362–366.

Weinheimer, E.M., Sands, L.P., & Campbell, W.W. (2010) A systematic review of the separate and combined effects of energy restriction and exercise on fat-free mass in middle-aged and older adults: implications for sarcopenic obesity. *Nutrition Reviews* 68, 375–388.

Wengreen, H.J., Munger, R.G., West, N.A., et al. (2004) Dietary protein intake and risk of osteoporotic hip fracture in elderly residents of Utah. *Journal of Bone and Mineral Research* 19, 537–545.

WHO Technical Report Series 935 (2011) Protein and Amino Acid Requirements in Human Nutrition. Report of a Joint WHO/FAO/UNU Expert Consultation.

Wilkinson, S.B., Phillips, S.M., Atherton, P.J., et al. (2008) Differential effects of resistance and endurance exercise in the fed state on signalling molecule phosphorylation and protein synthesis in human muscle. *Journal of Physiology* 586, 3701–3717.

Young, V.R. (1986) Nutritional balance studies: indicators of human requirements or of adaptive mechanisms? *Journal of Nutrition* 116, 700–703.

Chapter 11

Dietary Protein as a Trigger for Metabolic Adaptation

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Introduction

Besides a certain level of genetic predisposition and subsequent adherence and compliance to a well-designed training regimen, nutrition plays a key factor in determining exercise performance capacity. Proper dietary practice becomes even more important, as athletes approach their individual limits with respect to training volume and intensity. This has renewed the interest among athletes, coaches, and scientists in the role of diet and nutritional modulation to optimize training efficiency and subsequently maximize the physical performance capacity. Nutritional interventions, including the use of specifically designed sports nutrition products, are widely used by athletes in an effort to compensate for the metabolic demands imposed by intense exercise training and/or competition. To improve endurance exercise performance capacity, dietary interventions generally aim to manipulate endogenous carbohydrate availability before, during, and/or after exercise. For this purpose, numerous carbohydrate-based sports drinks, energy bars, and gels have been developed. In contrast, to enhance performance capacity in resistance-type exercise tasks, nutritional interventions generally aim to increase postexercise muscle protein synthesis rates and/or to reduce postexercise muscle protein breakdown to stimulate skeletal muscle hypertrophy. This chapter will provide an overview on the impact of dietary protein administration during and/or immediately after exercise on subsequent postexercise recovery and its possible impact on the skeletal muscle adaptive response to exercise training. A few basic guidelines regarding the preferred type and amount of dietary protein and the appropriate timing of protein administration to maximize postexercise muscle protein accretion will be formulated for practical purposes.

Postexercise Muscle Protein Reconditioning

Skeletal muscle tissue has an incredible capacity to adapt to changes in its use and/or disuse. This concept of muscle tissue plasticity is most evident when we compare the phenotypic response to prolonged endurance versus resistance-type exercise training, with the physique of an endurance-trained athlete (Figure 11.1a) being quite different from that of an athlete trained for high power development (Figure 11.1b). The capacity of skeletal muscle to structurally adapt to changes in its use is facilitated by the fact that skeletal muscle tissue is quite dynamic in nature. Skeletal muscle protein turns over at a rate of about 1–2% per day. The latter implies that skeletal muscle tissue is broken down and rebuilt approximately every 3 months. Throughout the day, muscle protein synthesis rates vary considerably and range between 0.04% and 0.14% per hour. Muscle protein synthesis responds to two main anabolic stimuli, these being food intake and physical activity. Food intake directly stimulates

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(a)

(b)

Figure 11.1 Muscle mass differences between (a) an Olympic Marathon runner and (b) an Olympic weightlifter. © 2012 IOC/Christopher Furlong and Tsutomu Kishimoto.

muscle protein synthesis rates, which is attributed to the presence of dietary protein in the meal. After protein digestion and absorption, amino acids will appear in the circulation. The essential amino acids (EAA), and leucine in particular, directly stimulate muscle protein synthesis, increasing muscle protein synthesis rates for 2–3 hours following ingestion of a single meal. Clearly, amino acids are much more than simply precursors for *de novo* muscle protein synthesis, and act as strong nutritional signaling molecules regulating multiple cellular processes. In addition to food intake, physical activity represents another potent anabolic stimulus. A single bout of exercise strongly increases muscle protein synthesis rates. The stimulating properties of food intake and physical activity on muscle protein synthesis throughout the day compensate for daily protein breakdown, thereby allowing skeletal muscle maintenance and, as such, ensuring proper functional capacity.

For the athlete in training, it is evident that the main goal is to adapt to the exercise training load thereby inducing structural changes that will allow greater performance capacity. For such muscle reconditioning to occur, muscle protein synthesis and breakdown rates need to be increased over a given period of time. In agreement, a single bout of exercise has been reported to stimulate skeletal muscle protein synthesis: muscle protein breakdown is also stimulated, but to a lesser extent, thereby improving muscle protein balance (Chesley et al., 1992; Phillips et al., 1997). Of course, differences will exist in the specific sets of muscle proteins that are expressed to a greater extent following different types of exercise—this is referred to as the specificity of the training response. Whereas resistance-type exercise mainly stimulates the expression of myofibrillar proteins, endurance-type exercise more effectively stimulates the expression of mitochondrial proteins, thereby allowing sport-specific muscle reconditioning (Wilkinson et al., 2008). Though exercise will improve muscle protein balance, net balance will remain negative in the absence of nutrient intake. Nutrition is required to allow proper muscle reconditioning and muscle hypertrophy to occur. It is here that the synergy between physical activity and nutrition becomes clear. When food is ingested following a single bout of exercise, muscle protein synthesis rates are increased to a much higher level and subsequently remain elevated for an extended period of time (Moore et al., 2009b). In fact, recent work actually shows that exercise performed prior to food intake will allow more of the ingested protein to be used for *de novo* muscle protein synthesis (Pennings et al., 2010). The

stimulating properties of exercise on the postprandial muscle protein synthetic response seem to be long-lived, with the muscle protein synthetic response to protein ingestion still being elevated 24 hours after performing a single bout of resistance-type exercise (Burd et al., 2011).

From the previous section, it has become clear that nutrition forms a key factor in determining the impact of exercise training on muscle reconditioning. Carbohydrate ingestion during postexercise recovery is an effective intervention to inhibit exercise-stimulated muscle protein breakdown, but does not seem to affect muscle protein synthesis (Borsheim et al., 2004). Though postexercise protein balance will improve following the ingestion of carbohydrate, net protein balance will remain negative (Borsheim et al., 2004). The inhibitory effect of carbohydrate ingestion on postexercise muscle protein breakdown has largely been attributed to the concomitant rise in circulating plasma insulin concentrations. However, even though elevated plasma insulin levels have been reported to stimulate net muscle protein anabolism, these properties are evident only in the presence of increased amino acid availability (Biolo et al., 1995). Recent studies support the contention that insulin is not a major regulatory factor determining muscle protein balance and identify amino acid availability as being the main stimulus for muscle protein synthesis under normal, resting conditions (Fujita et al., 2006). Though there are many reasons to ingest carbohydrate following exercise, it does not increase muscle protein synthesis rates. Protein/amino acid administration is required to effectively stimulate postexercise muscle protein synthesis rates. Biolo et al. (1997) demonstrated that hyperaminoacidemia, following intravenous amino acid infusion, increased the postexercise muscle protein synthesis rates and suppressed the exercise-induced increase in protein breakdown. Thereafter, Tipton et al. (1999) showed that only the postexercise ingestion of 40 g of either mixed amino acids (MAA) or EAA also effectively stimulated muscle protein synthesis rates. Follow-up studies assessed the impact of smaller amounts of EAA with and without additional carbohydrate and showed that these were also effective in stimulating postexercise muscle protein synthesis, resulting in a positive net protein balance during acute postexercise recovery (Borsheim et al., 2002; Rasmussen et al., 2000). Since then, numerous other studies have shown that amino acid and/or protein administration increases muscle protein synthesis rates following resistance-type exercise (Borsheim et al., 2002, 2004; Koopman et al., 2005, 2007; Miller et al., 2003; Rasmussen et al., 2000; Tipton et al., 1999, 2001). Though most studies have applied resistance-type exercise, there are several studies showing protein administration to increase muscle protein synthesis rates following endurance-type exercise activities (Gibala, 2007; Howarth et al., 2009; Levenhagen et al., 2002). In short, to facilitate muscle reconditioning following exercise training, dietary protein should be provided. In the following section, we will define the optimum nutritional strategies that will allow maximal muscle protein synthesis rates following exercise.

Dietary Protein Ingestion Following Exercise

Although it is well established that dietary protein ingestion effectively stimulates muscle protein synthesis both at rest and following exercise, there is still considerable debate regarding the exact amount and type of dietary protein and the desired timing of protein ingestion to maximize postexercise muscle protein synthesis rates.

Amount of Dietary Protein

Previously, Tipton et al. (1999) showed that only the ingestion of 40-g MAAs or EAAs effectively stimulated postexercise muscle protein synthesis. Since the ingestion of 40-g MAA or 40-g EAA resulted in a similar net protein balance, it was suggested that it might not be necessary to ingest nonessential amino acids during immediate postexercise recovery. Follow-up studies assessed the impact of ingesting merely 6-g EAA (which would translate to ~12-g protein) with and without carbohydrate and showed that this amount was also effective in stimulating postexercise muscle protein synthesis. However, ingestion of such a small amount of EAA after exercise resulted in a positive net protein balance

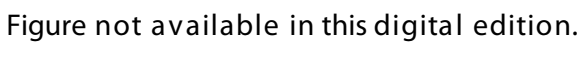

Figure 11.2 Dose-response relationship between the amount of protein ingested and postexercise muscle protein synthesis rates. Values represent means ± SEM. Means with different letters are significantly different from each other. From Moore et al. (2009a), with permission from American Society for Nutrition.

for up to 2 hours only, after which net protein balance became negative again (Borsheim et al., 2002). The latter implies that ingestion of such a small amount of amino acids is insufficient to remain in an anabolic state. Recently, Moore et al. (2009a) conducted a dose-response study to investigate the relationship between the amount of dietary protein ingested and subsequent postexercise muscle protein synthesis rates (Figure 11.2). The fractional mixed muscle protein synthetic rate increased with the ingestion of greater amounts of protein, reaching maximum synthesis rates following ingestion of 20 g intact (egg) protein, which provides ~8.6-g EAA. The authors speculated that athletes should ingest this amount of dietary protein 5–6 times daily to allow maximal stimulation of skeletal muscle protein synthesis throughout the day.

Source of Dietary Protein

Studies have reported improved postexercise protein balance and/or greater muscle protein synthesis rates following the ingestion of whey protein (Borsheim et al., 2004), casein protein (Borsheim et al., 2004), soy protein (Wilkinson et al., 2007), casein protein hydrolysate (Koopman et al., 2005, 2006b), egg protein (Moore et al., 2009a), and whole-milk and/or fat-free milk (Elliot et al., 2006; Wilkinson et al., 2007). It seems obvious to question which source of dietary protein is most effective to promote postexercise muscle protein synthesis. There is only limited research comparing the efficacy of the ingestion of different proteins sources on the postexercise protein synthetic response. It is, therefore, not possible to identify a specific protein source that is most potentiating. This is further complicated by the fact that numerous parameters modulate the postexercise muscle protein synthetic response to protein ingestion. The type, intensity, and duration of the exercise performed prior to protein ingestion, the duration of the recovery period that is assessed, the amount and timing of protein administration, the amino acid composition of the protein, and the digestion and absorption kinetics of the protein source (or mixed meal), may all modulate the muscle protein synthetic response. This makes it impossible to compare the muscle protein synthetic response to the ingestion of different proteins between different studies. Only few studies have tried to directly assess differences in the postexercise muscle protein synthetic response to the ingestion of various types of dietary protein within a single study.

Milk protein and its main isolated constituents, whey and casein, seem to offer an anabolic advantage over soy protein for promoting muscle hypertrophy (Fouillet et al., 2002; Tang et al., 2009; Wilkinson et al., 2007). Casein and whey protein seem to have distinct anabolic properties, which are attributed to differences in digestion and absorption kinetics (Boirie et al., 1997; Borsheim et al., 2004; Dangin et al., 2001, 2003; Pennings et al., 2011; Tipton et al., 2004). Whereas whey protein is a soluble protein that leads to fast intestinal absorption, intact casein clots in the stomach delaying its digestion and absorption and the subsequent release of amino acids in the circulation (Koopman et al., 2009). The fast, but transient rise, in plasma amino acid concentration after whey protein ingestion can lead to higher protein synthesis and oxidation rates (Boirie et al., 1997; Dangin et al., 2001, 2003). In addition to intrinsic differences in digestion and absorption rate, it has been suggested that whey protein can more effectively stimulate protein synthesis due to its greater leucine content when compared to casein

(Pennings et al., 2011; Tang et al., 2009). However, the latter does not mean that simply supplementing crystalline leucine can further augment the postexercise muscle protein synthetic response to protein ingestion. In a series of studies, Koopman et al. showed that co-ingestion of free leucine does not further increase muscle protein synthesis rate when an ample amount of casein protein hydrolysate is ingested during postexercise recovery (Koopman et al., 2005, 2006b, 2008). Additional studies are warranted to assess the impact of the digestion and absorption kinetics of a protein source and its amino acid composition on stimulating muscle protein synthesis rates following resistance- and endurance-type exercise.

Carbohydrate Ingestion

Postexercise nutritional interventions are generally targeted to replenish endogenous substrate stores, repair skeletal muscle damage, and/or facilitate the adaptive response to exercise training. In the trained athlete involved in endurance or team sports activities, rapid restoration of depleted glycogen stores is generally a priority to accelerate postexercise recovery and maintain performance capacity (Beelen et al., 2011a). The latter is of particular relevance when a second bout of exercise is scheduled within 24 hours. Consequently, these athletes mainly focus on carbohydrate ingestion for acute postexercise recovery. Recently, co-ingestion of relative small amounts of protein and/or amino acids has become popular among these athletes, mainly because this can further accelerate muscle glycogen repletion (van Loon et al., 2000). Several studies have shown that co-ingestion of protein with or without additional free amino acids stimulates endogenous insulin release during postexercise recovery, thereby stimulating glucose uptake and subsequent storage as glycogen in skeletal muscle tissue. Though various studies have shown accelerated postexercise glycogen repletion rates following co-ingestion of protein and/or amino acid with moderate amounts of carbohydrate (<1.0 g/kg bodyweight/h), such benefits were no longer apparent when large(r) amounts of carbohydrate (>1.2 g/kg/h) were ingested (Koopman et al., 2006a). Therefore, protein amino acid co-ingestion can be used as an effective strategy to accelerate glycogen repletion during the first few hours of postexercise recovery, when food intake (i.e., carbohydrate ingestion) is generally restricted (Beelen et al., 2011a).

Of course, protein and/or amino acids are generally ingested following exercise as a means to stimulate muscle hypertrophy. Since protein and/or amino acid ingestion has been proven essential to allow net muscle protein accretion following exercise (Borsheim et al., 2004; Koopman et al., 2005, 2006b; Miller et al., 2003), athletes involved in resistance-type exercise training often ingest large quantities of protein and carbohydrate following cessation of exercise (i.e., traditional "weight gainers"). Though the combined ingestion of protein with large amounts of carbohydrate is generally advocated among these athletes, there is little evidence that co-ingestion of large amounts of carbohydrate will augment net muscle protein accretion following exercise. Co-ingestion of carbohydrate during postexercise recovery has been shown to improve net leg amino acid balance (Borsheim et al., 2004), which has been attributed to the concomitant increase in circulating plasma insulin concentrations. In an attempt to assess whether carbohydrate co-ingestion is required to maximize postexercise muscle protein synthesis, we observed no additional benefit of the co-ingestion of either a small or large amount of carbohydrate on postexercise muscle protein synthesis rates under conditions where ample protein is ingested (Koopman et al., 2007). Though carbohydrate co-ingestion does not seem to be required to maximize postexercise muscle protein synthesis rates, it is likely that some carbohydrate can attenuate the postexercise rise in muscle protein breakdown rate, thereby improving net protein balance (Borsheim et al., 2004). Furthermore, as muscle glycogen content can be reduced by 30–40% following a single session of resistance-type exercise (Koopman et al., 2006a), some carbohydrate co-ingestion may be preferred when these athletes wish to allow full muscle glycogen repletion to maintain exercise performance capacity. However, the large doses of carbohydrate that are often advocated are not required to maximize postexercise muscle protein accretion.

Timing of Dietary Protein Ingestion

As both the amount and type of protein ingested seem to modulate the postexercise muscle protein synthetic response, it is hardly surprising that the timing of protein ingestion forms another key factor in stimulating postexercise muscle anabolism. Levenhagen et al. (2001) reported an improved postexercise net protein balance after consuming a protein-containing supplement immediately after cessation of exercise as opposed to 3 hours later. Furthermore, recent studies suggest that carbohydrate and protein co-ingestion prior to and/or during exercise may further augment postexercise muscle protein accretion (Beelen et al., 2008a; Tipton et al., 2001). Tipton et al. (2001) showed that amino acid ingestion prior to, as opposed to after, exercise further augments net muscle protein accretion during subsequent recovery. The stimulating effect of protein or amino acid supplementation prior to exercise on muscle protein synthesis after exercise has been attributed to a more rapid supply of amino acids to the muscle during the acute stages of postexercise recovery. However, it could also be speculated that protein ingestion prior to and/or during resistance-type exercise already stimulates muscle protein synthesis during exercise, thereby creating a larger timeframe for muscle protein synthesis to be elevated (Figure 11.3). In a recent study, we confirmed that co-ingestion of protein with carbohydrate before and during 2 hours of intermittent, resistance-type exercise stimulates muscle protein synthesis during exercise (Beelen et al., 2008a). It was speculated that the observed impact of protein co-ingestion on mixed muscle protein synthesis during exercise is restricted to intermittent, resistance-type exercise activities (Beelen et al., 2008a). It remains to be determined if protein ingestion before and/or during exercise also increases muscle protein synthesis during more continuous, endurance-type, exercise activities. Preliminary findings in our laboratory seem to indicate that even during moderate intensity endurance-type exercise muscle protein synthesis rates are stimulated in the working muscle by protein co-ingestion prior to and during exercise (Beelen et al., 2011b). More work is needed to address the relevance of the potential to stimulate muscle protein synthesis during exercise, thereby creating a larger timeframe for muscle protein synthesis rates to be increased when compared with merely focusing on acute postexercise recovery (Figure 11.4).

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Figure 11.3 Dietary protein ingestion prior to and during resistance-type exercise stimulates muscle protein synthesis during exercise. Fractional synthesis rate (FSR) of mixed muscle protein during exercise following carbohydrate (CHO) or carbohydrate plus protein (CHO + PRO) ingestion. Values represent means ± SEM. *Significantly different from CHO. From Beelen et al. (2008a), with permission from American Physiological Society.

Acute Versus Long-Term Adaptive Response to Exercise

Research on the impact of dietary intervention on sports performance has traditionally focused on the acute ergogenic properties of nutritional

- Provide sufficient protein (20–25 g) with each main meal
- Ingest 20–25 g dietary protein during or immediately after an exercise session
- Whey forms an excellent source of dietary protein to promote post-exercise recovery
- Co-ingest carbohydrate based on the need to replete liver and muscle glycogen stores

Figure 11.4 Practical recommendations for the athlete regarding dietary protein consumption during and/or immediately after an exercise session.

compounds and/or supplements. At present, the relevance of more structural nutritional modulation to increase the efficiency of the adaptive response to (regular) exercise training is receiving more interest from both science and practice perspective. The efficacy of specific nutritional interventions to improve postexercise recovery constitutes a key factor driving the skeletal muscle adaptive response to exercise training and, in turn, determines performance capacity. More studies are warranted to assess the adaptive response following a single or several successive bouts of exercise in a setting more representative of real-life conditions. Most recovery studies have assessed the impact of nutrition on muscle protein synthesis following a single bout of exercise performed in an overnight fasted state. The latter is not representative of habitual exercise training or competition in which athletes generally practice standard pre-competition dietary guidelines. Furthermore, most (recreational) athletes exercise in the evening and have dinner before or after a workout. In this respect, the benefits of postexercise recovery nutrition remain largely uninvestigated.

One question that is presently under investigation is whether protein administration following exercise performed in the evening has an impact on subsequent overnight recovery. For obvious methodological issues, postexercise muscle reconditioning has hardly been studied during overnight sleep. Recently, we evaluated the impact of exercise performed in the evening on muscle protein synthesis during subsequent overnight recovery (Beelen et al., 2008b). We observed an increase in muscle protein synthesis during the first few hours of postexercise recovery when protein was ingested after cessation of exercise. However, muscle protein synthesis rates during subsequent overnight sleep were unexpectedly low, with values being close to basal, postabsorptive values. Clearly, many people misinterpret the outcome of classic studies like Phillips et al. (1997) when suggesting that the postexercise increase in muscle protein synthesis rate persists for up to 24–48 hours. It has been well established that greater postexercise muscle protein synthesis rates reach peak values somewhere between 2 and 3 hours after cessation of exercise in the fed state after which levels decline to pre-exercise levels by 5–6 hours of postexercise recovery (Moore et al., 2009b). Clearly, though postexercise protein ingestion stimulates muscle protein synthesis during the acute stages of postexercise recovery, these muscle protein synthesis rates are not maintained during subsequent overnight recovery and/or other conditions. However, recent work shows that the skeletal muscle protein synthetic response to protein intake remains elevated for up to 24 hours after cessation of exercise (Burd et al., 2011). More research is required to investigate to what extent this exercise-induced increase in skeletal muscle sensitivity to protein administration can be applied to maximize the adaptive response to a single bout of exercise. It will be of interest to further explore the impact of protein provided prior to or during overnight sleep (Groen et al., 2011), and the impact of changes in dietary protein distribution throughout the day on muscle tissue reconditioning.

So far most work in the field aims to establish the impact of nutritional intervention on muscle protein accretion during the acute stages of postexercise recovery. However, it should be noted that dietary interventions that optimize postexercise muscle protein accretion do not necessarily translate into a more successful skeletal muscle adaptive response following more prolonged exercise training. Though a discussion on this topic is beyond the scope of this review, it is evident that numerous intrinsic and extrinsic factors are responsible for orchestrating the long-term skeletal muscle adaptive response to exercise training and we need to establish the effect of nutrition on all of these processes. However, a healthy balanced diet and the use of specifically designed sports nutrition will allow a more efficient adaptive response to regular exercise training. With regard to the application of protein supplements it seems evident that differences in amino acid composition and specific differences in digestion and absorption kinetics would be of great relevance here. As a consequence, more specific designer proteins or protein mixtures will be defined and applied in more individualized sports recovery nutrition, with specificity regarding the type, intensity, duration, and frequency of exercise and optimized for the concomitant priorities set for postexercise recovery and subsequent muscle tissue reconditioning in the individual athlete.

Conclusions

Dietary protein intake during and/or immediately after cessation of resistance- or endurance-type exercise stimulates postexercise muscle protein synthesis, inhibits protein breakdown and, as such, allows net muscle protein accretion. Such dietary practice will allow a more efficient adaptive response to each successive exercise bout, resulting in improved muscle tissue reconditioning. Whey protein seems most effective to increase muscle protein synthesis rates during acute postexercise recovery. This is likely attributed to its rapid digestion and absorption kinetics as well as its amino acid composition. About 20 g of a high quality dietary protein should be provided during and/or immediately after cessation of exercise to allow postexercise muscle protein synthesis rates to reach maximal levels. Co-ingestion of (large) amounts of carbohydrate or additional crystalline leucine does not further augment postexercise muscle protein synthesis rates when ample dietary protein is already provided. A healthy diet with smart timing of the ingestion of dietary protein after exercise will further improve the skeletal muscle adaptive response to prolonged exercise training.

References

Beelen, M., Koopman, R., Gijsen, A. P., et al. (2008a) Protein coingestion stimulates muscle protein synthesis during resistance-type exercise. *American Journal of Physiology. Endocrinology and Metabolism* 295, E70–E77.

Beelen, M., Tieland, M., Gijsen, A.P., et al. (2008b) Coingestion of carbohydrate and protein hydrolysate stimulates muscle protein synthesis during exercise in young men, with no further increase during subsequent overnight recovery. *Journal of Nutrition* 138, 2198–2204.

Beelen, M., Burke, L.M., Gibala, M.J., et al. (2011a) Nutritional strategies to promote postexercise recovery. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 515–532.

Beelen, M., Zorenc, A., Pennings, B., et al. (2011b) Impact of protein coingestion on muscle protein synthesis during continuous endurance type exercise. *American Journal of Physiology. Endocrinology and Metabolism* 300, E945–E954.

Biolo, G., Maggi, S.P., Williams, B.D., et al. (1995) Increased rates of muscle protein turnover and amino acid transport after resistance exercise in humans. *American Journal of Physiology* 268, E514–E520.

Biolo, G., Tipton, K.D., Klein, S., et al. (1997) An abundant supply of amino acids enhances the metabolic effect of exercise on muscle protein. *American Journal of Physiology* 273, E122–E129.

Boirie, Y., Dangin, M., Gachon, P., et al. (1997) Slow and fast dietary proteins differently modulate postprandial protein accretion. *Proceedings of the National Academy of Sciences of USA* 94, 14930–14935.

Borsheim, E., Tipton, K.D., Wolf, S. E., et al. (2002) Essential amino acids and muscle protein recovery from resistance exercise. *American Journal of Physiology. Endocrinology and Metabolism* 283, E648–E657.

Borsheim, E., Cree, M.G., Tipton, K. D., et al. (2004) Effect of carbohydrate intake on net muscle protein synthesis during recovery from resistance exercise. *Journal of Applied Physiology* 96, 674–678.

Burd, N.A., West, D.W., Moore, D.R., et al. (2011) Enhanced amino acid sensitivity of myofibrillar protein synthesis persists for up to 24 h after resistance exercise in young men. *Journal of Nutrition* 141, 568–573.

Chesley, A., Macdougall, J.D., Tarnopolsky, M.A., et al. (1992) Changes in human muscle protein synthesis after resistance exercise. *Journal of Applied Physiology* 73, 1383–1388.

Dangin, M., Boirie, Y., Garcia-Rodenas, C., et al. (2001) The digestion rate of protein is an independent regulating factor of postprandial protein retention. *American Journal of Physiology. Endocrinology and Metabolism* 280, E340–E348.

Dangin, M., Guillet, C., Garcia-Rodenas, C., et al. (2003) The rate of protein digestion affects protein gain differently during aging in humans. *Journal of Physiology* 549, 635–644.

Elliot, T.A., Cree, M.G., Sanford, A.P., et al. (2006) Milk ingestion stimulates net muscle protein synthesis following resistance exercise. *Medicine & Science in Sports & Exercise* 38, 667–674.

Fouillet, H., Mariotti, F., Gaudichon, C., et al. (2002) Peripheral and splanchnic metabolism of dietary nitrogen are differently affected by the protein source in humans as assessed by compartmental modeling. *Journal of Nutrition* 132, 125–133.

Fujita, S., Rasmussen, B.B., Cadenas, J.G., et al. (2006) Effect of insulin on human skeletal muscle protein synthesis is modulated by insulin-induced changes in muscle blood flow and amino acid availability. *American Journal of Physiology. Endocrinology and Metabolism* 291, E745–E754.

Gibala, M.J. (2007) Protein metabolism and endurance exercise. *Journal of Sports Medicine* 37, 337–340.

Groen, B.B., Res, P.T., Pennings, B., et al. (2011) Intragastric protein administration stimulates overnight muscle protein synthesis in elderly men. *American Journal of Physiology. Endocrinology and Metabolism* 302, E52–E60.

Howarth, K.R., Moreau, N.A., Phillips, S.M., et al. (2009) Coingestion of protein with carbohydrate during recovery from endurance exercise stimulates skeletal muscle protein synthesis in humans. *Journal of Applied Physiology* 106, 1394–1402.

Koopman, R., Wagenmakers, A.J., Manders, R.J., et al. (2005) Combined ingestion of protein and free leucine with carbohydrate increases postexercise muscle protein synthesis in vivo in male subjects. *American Journal of Physiology. Endocrinology and Metabolism* 288, E645–E653.

Koopman, R., Manders, R.J., Jonkers, R.A., et al. (2006a) Intramyocellular lipid and glycogen content are reduced following resistance exercise in untrained healthy males. *European Journal of Applied Physiology* 96, 525–534.

Koopman, R., Verdijk, L., Manders, R.J., et al. (2006b) Co-ingestion of protein and leucine stimulates muscle protein synthesis rates to the same extent in young and elderly lean men. *American Journal of Clinical Nutrition* 84, 623–632.

Koopman, R., Beelen, M., Stellingwerff, T., et al. (2007) Coingestion of carbohydrate with protein does not further augment postexercise muscle protein synthesis. *American Journal of Physiology. Endocrinology and Metabolism* 293, E833–E842.

Koopman, R., Verdijk, L.B., Beelen, M., et al. (2008) Co-ingestion of leucine with protein does not further augment post-exercise muscle protein synthesis rates in elderly men. *British Journal of Nutrition* 99, 571–580.

Koopman, R., Crombach, N., Gijsen, A.P., et al. (2009) Ingestion of a protein hydrolysate is accompanied by an accelerated in vivo digestion and absorption rate when compared with its intact protein. *American Journal of Clinical Nutrition* 90, 106–115.

Levenhagen, D.K., Gresham, J. D., Carlson, M.G., et al. (2001) Postexercise nutrient intake timing in humans is critical to recovery of leg glucose and protein homeostasis. *American Journal of Physiology. Endocrinology and Metabolism* 280, E982–E993.

Levenhagen, D.K., Carr, C., Carlson, M.G., et al. (2002) Postexercise protein intake enhances whole-body and leg protein accretion in humans. *Medicine & Science in Sports & Exercise* 34, 828–837.

Miller, S.L., Tipton, K.D., Chinkes, D.L., et al. (2003) Independent and combined effects of amino acids and glucose after resistance exercise. *Medicine & Science in Sports & Exercise* 35, 449–455.

Moore, D.R., Robinson, M.J., Fry, J. L., et al. (2009a) Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. *American Journal of Clinical Nutrition* 89, 161–168.

Moore, D.R., Tang, J.E., Burd, N.A., et al. (2009b) Differential stimulation of myofibrillar and sarcoplasmic protein synthesis with protein ingestion at rest and after resistance exercise. *Journal of Physiology* 587, 897–904.

Pennings, B., Koopman, R., Beelen, M., et al. (2010) Exercising before protein intake allows for greater use of dietary protein-derived amino acids for de novo muscle protein synthesis in both young and elderly men. *American Journal of Clinical Nutrition* 93, 322–331.

Pennings, B., Boirie, Y., Senden, J. M., et al. (2011) Whey protein stimulates postprandial muscle protein accretion more effectively than do casein and casein hydrolysate in older men. *American Journal of Clinical Nutrition* 93, 997–1005.

Phillips, S.M., Tipton, K.D., Aarsland, A., et al. (1997) Mixed muscle protein synthesis and breakdown after resistance exercise in humans. *American Journal of Physiology* 273, E99–E107.

Rasmussen, B.B., Tipton, K.D., Miller, S.L., et al. (2000) An oral essential amino acid-carbohydrate supplement enhances muscle protein anabolism after resistance exercise. *Journal of Applied Physiology* 88, 386–392.

Tang, J.E., Moore, D.R., Kujbida, G.W., et al. (2009) Ingestion of whey hydrolysate, casein, or soy protein isolate: effects on mixed muscle protein synthesis at rest and following resistance exercise in young men. *Journal of Applied Physiology* 107, 987–992.

Tipton, K.D., Ferrando, A.A., Phillips, S.M., et al. (1999) Postexercise net protein synthesis in human muscle from orally administered amino acids. *American Journal of Physiology* 276, E628–E634.

Tipton, K.D., Rasmussen, B.B., Miller, S.L., et al. (2001) Timing of amino acid-carbohydrate ingestion alters anabolic response of muscle to resistance exercise. *American Journal of Physiology. Endocrinology and Metabolism* 281, E197–E206.

Tipton, K.D., Elliott, T.A., Cree, M.G., et al. (2004) Ingestion of casein and whey proteins result in muscle anabolism after resistance exercise. *Medicine & Science in Sports & Exercise* 36, 2073–2081.

Van Loon, L.J., Saris, W.H., Kruijshoop, M., et al. (2000) Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *American Journal of Clinical Nutrition* 72, 106–111.

Wilkinson, S.B., Tarnopolsky, M.A., Macdonald, M.J., et al. (2007) Consumption of fluid skim milk promotes greater muscle protein accretion after resistance exercise than does consumption of an isonitrogenous and isoenergetic soy-protein beverage. *American Journal of Clinical Nutrition* 85, 1031–1040.

Wilkinson, S.B., Phillips, S.M., Atherton, P.J., et al. (2008) Differential effects of resistance and endurance exercise in the fed state on signalling molecule phosphorylation and protein synthesis in human muscle. *Journal of Physiology* 586, 3701–3717.

Chapter 12

Fat Metabolism During and After Exercise

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Introduction

Fatty acids (FA) are major fuels for muscle contractions, especially during light-to-moderate exercise intensities and during exercise of long duration. With increasing exercise intensities, however, there is a progressive shift from FA to carbohydrate (CHO) oxidation. During the postexercise recovery period, muscle glycogen resynthesis has a high metabolic priority resulting in elevated FA oxidation lasting several hours after termination of exercise. The aim of this chapter is to provide the reader with general insight into the many steps involved from FA delivery to their final destination in mitochondria for oxidation, how the different FA sources contribute to energy turnover during exercise, how FA oxidation during exercise is regulated and which factors might be limiting in FA oxidation with increasing exercise intensity. Furthermore, the role of FA during postexercise recovery is discussed.

Fatty Acids as Energy Sources

Lipids constitute a broad group of molecules, ranging from FA and triacylglycerols (TGs) to phospholipids, cholesterols, and sphingolipids, and serve several important functions in the organism, like structural components of cell membranes, in cellular signaling, and represent the largest storage pool for energy turnover.

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Endogenous TG represents the largest readily available chemical energy source that can be used for biological work (e.g., muscle contraction). TG consists of three FA esterified to glycerol. FA provide the highest content of energy of all nutrients (37.5 kJ/g for stearic acid), which is much higher than that of carbohydrates (CHO) (16.9 kJ/g for glucose). FA, when oxidized, also provide more adenosine triphosphate (ATP) per molecule than glucose (147 vs. 38 ATP), although the complete oxidation of FA requires more oxygen than the oxidation of CHO (26 vs. 6 mole of oxygen per mole of substrate for stearic acid and glucose oxidation, respectively).

Most of the TGs are stored in the adipose tissue. The size of the adipose tissue lipid pool is difficult to estimate and depends on the fat mass of each individual. In order for TG, located in adipose tissue, to be used as a substrate for oxidative metabolism, it requires hydrolysis of TG (lipolysis) to release FA. When released from the adipocyte the liberated FA are transported bound to albumin by the blood to the active tissues for oxidation.

Circulating Very Low Density Lipoprotein Triacylglycerol

Albumin-bound FA entering the liver can result in the formation of TG, which then is secreted into the circulation bound to very low density lipoproteins (VLDL-TG). VLDL is the main carrier of TG in the postabsorptive state. Hydrolysis of TG bound to VLDL is mediated by the enzyme lipoprotein lipase (LPL) (Figure 12.1), which in its active form is located at the luminal surface of the endothelial cells

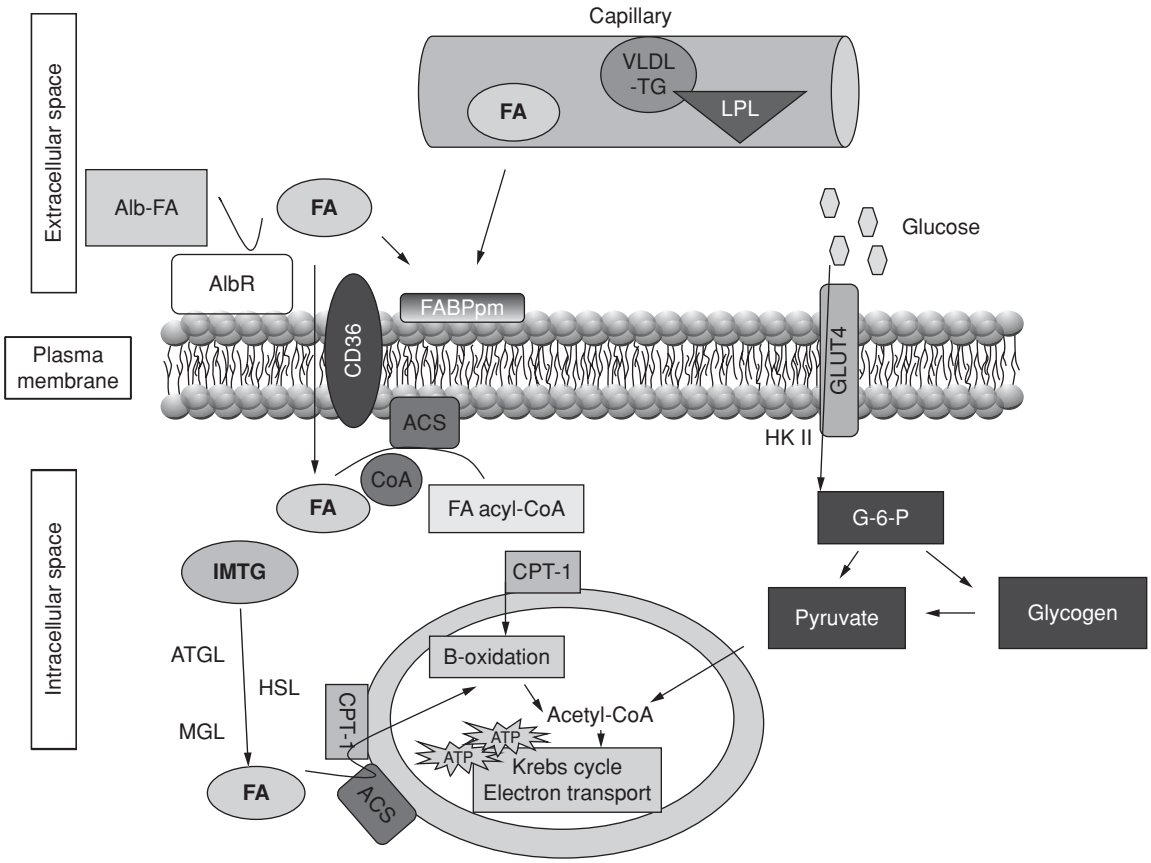

Figure 12.1 Schematic representation of the likely routes taken by fatty acids (FA) and glucose from the capillaries to the mitochondria for oxidation or storage as intramyocellular triacylglycerol (IMTG) and glycogen, respectively, in skeletal muscle. Alb-FA, albumin-bound fatty acids; AlbR, albumin receptor; ACS, acyl-CoA synthetase; TCA, tricarboxylic acid cycle; HK II, hexokinase II. Other abbreviations are given in the text.

in the capillary bed in several tissues including both skeletal muscle and adipose tissue. Upon hydrolysis of VLDL-TG, FA are liberated and are then available for uptake into the surrounding tissue, including skeletal muscle. TG is a very energy-dense fuel and FA liberated from VLDL-TG hydrolysis contribute significantly to the total energy turnover.

Transport of Plasma FA into Muscle

For energy turnover, FA derived from albumin-bound FA and FA liberated from hydrolysis of VLDL-TG enter the muscle cell and are oxidized in the mitochondria through a process involving several steps: the FA have to pass (1) the endothelium, (2) the interstitial space, (3) the sarcolemma, (4) the cytosol, and (5) the mitochondrial membranes toward final oxidation in the mitochondria (Figure 12.1). Regarding transport out of the capillaries, detailed knowledge is not available, but the FA likely leave the capillaries by passive diffusion or aided by proteins, as described in more detail for the transport across sarcolemma. As albumin is present in the interstitial space, but at a markedly lower concentration than in plasma, it is also likely that some FA are simply transferred across the endothelium bound to albumin in an exocytotic process. Uptake of FA across the sarcolemma is mediated both by passive diffusion and lipid-binding proteins. Within recent years,

several membrane-bound lipid-binding proteins have been identified in human skeletal muscle and increasing evidence is emerging that these proteins, either individually or in complexes, act as regulators of FA transmembrane transport. However, the mechanism by which this occurs is unknown. The 43 kDa fatty acid-binding protein (FABPpm), a peripheral membrane-bound protein, and the 88 kDa fatty acid translocase CD36 (FAT/CD36) protein are currently the best-described lipid-binding proteins in human skeletal muscle (for detailed review see Glatz et al., 2010). The fatty acid transport proteins (FATPs) are also suggested to be involved in transmembrane FA transport. When entering the cytosol, FA is activated by the enzyme acyl-CoA synthase (ACS) to form fatty acyl-CoA (Figure 12.1). The intracellular transport of FA is mediated by two proteins: the cytosolic fatty acid-binding protein (FABPc) and acyl-CoA-binding protein (ACBP) (Glatz et al., 2010).

TG Located within the Skeletal Muscle Cell

Another physiologically important store of lipid can be found within the skeletal muscle (intramuscular triacylglycerol, IMTG). IMTGs are stored in confined organelles known as lipid droplets, most of which are located adjacent to the mitochondria. The total muscle mass may contain up to 300 g of TG within the myocytes located in lipid droplets within the myocytes, although this amount can vary substantially due to individual differences in fiber-type content, diet, and sex. In the 1970s, Essen et al. (1975) showed that type I fibers contain a greater concentration of IMTG than type II fibers. A fat-rich diet increases IMTG content (Kiens et al., 1987) and it has also been shown that IMTG is more abundant in females than in males when females and males were matched according to their maximal oxygen uptake (VO_2 peak) expressed relative to lean body mass, their physical activity level, and training history, and females were in the follicular phase of the menstrual cycle, which might be ascribed to more type I fibers in females than in males (Steffensen et al., 2002). An additional explanation for the higher IMTG content in females than in males could be the percent body fat, which usually is higher in females than in males. Support for this are the findings of a significant correlation between percent body fat and basal IMTG content (Roepstorff et al., 2006a; White et al., 2006).

Lipolysis of IMTG to FA is mediated by lipases, which sequentially hydrolyze FA from the glycerol backbone. Three enzymes have been implicated in the hydrolysis of cellular TG: adipose tissue triglyceride (ATGL), hormone-sensitive lipase (HSL), and monoglyceride lipase (MGL) (Figure 12.1). Most of the current knowledge is generated in studies performed on adipose tissue, where it is believed that ATGL and HSL are responsible for the first two steps of lipolysis of TG, and MGL is hydrolyzing the last ester bond. The importance of ATGL for skeletal muscle lipolysis is highlighted by the findings of ATGL knockout mice displaying a massive accumulation of TG in virtually all tissues, including skeletal muscle (Haemmerle et al., 2006). Recently, it was shown that ATGL protein content was increased after a period of endurance training in male volunteers (Alsted et al., 2009). Although these observations suggest that ATGL plays a role for lipolysis of IMTG, it has not been demonstrated under which physiological conditions ATGL is activated and the role of ATGL for lipolysis of IMTG during exercise (muscle contractions) is therefore unknown.

Fat Metabolism during Prolonged Exercise

The contribution of lipids to the total energy utilized during exercise is dependent on a variety of factors including exercise duration and intensity. The classical studies by Christensen and Hansen (1939) clearly demonstrated that with increasing exercise intensity there is a shift toward a higher reliance on carbohydrate oxidation and less FA oxidation for energy production. The maximum contribution of lipids as energy substrate is reached at approximately 60–65% of maximal oxygen uptake (Christensen & Hansen, 1939; Romijn et al., 1993; van Loon et al., 2001). Above this exercise intensity, FA oxidation progressively decreases while the energy is increasingly covered by carbohydrates via glycogen breakdown and glucose oxidation

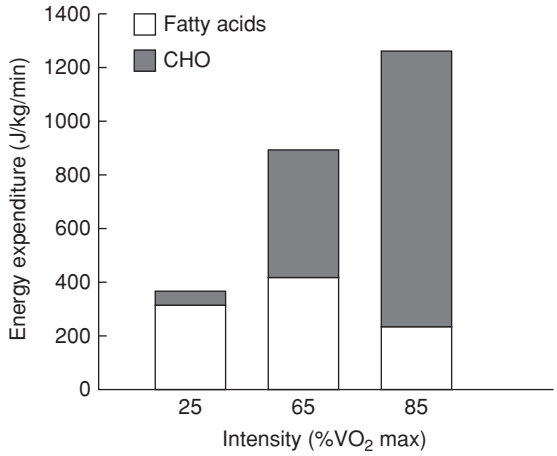

Figure 12.2 Maximum contribution to energy expenditure from fatty acids and carbohydrates (CHO) in relation to exercise intensity. Modified from Romijn et al. (1993).

(Figure 12.2). It is, however, important to remember that even though maximal lipid contribution occurs at moderate exercise intensities, still around half the energy is covered by carbohydrates. Already in the 1930s, Christensen and Hansen (1939) showed that FA oxidation was gradually enhanced during prolonged exercise when exercise lasted for more than 1–1.5 hours (Figure 12.3). It is noteworthy that in this and in several other studies the ratio between carbohydrate and FA oxidation is quite stable when exercise duration is below 1–1.5hours both in untrained and trained individuals (Helge et al., 1996; Mendenhall et al., 1994; Roepstorff et al., 2002). Thus a shift from carbohydrate to FA oxidation is only significant when exercise duration is beyond 1–1.5 hours, though it can be greatly affected by the preceding diet (Maughan et al., 1978).

It is also worth noticing that a higher FA oxidation is often observed in endurance-trained females during prolonged exercise at moderate exercise

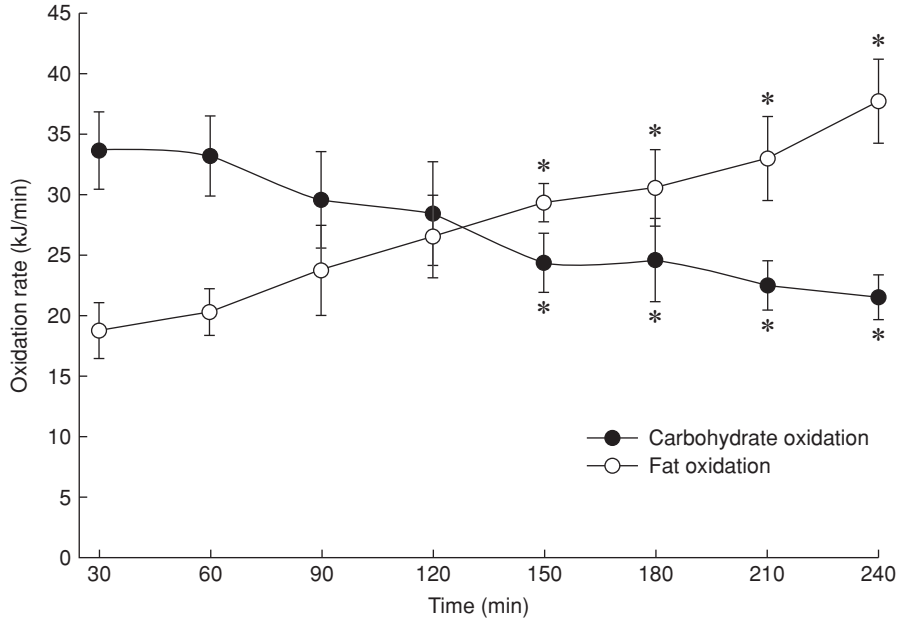

Figure 12.3 Whole body fatty acid and carbohydrate oxidation estimated from respiratory exchange ratio (RER) during prolonged (240 minute) exercise at a moderate intensity (55% VO_2 max). Values are means ± SE. *$p < 0.05$. From Watt et al. (2002).

intensities (60–65% VO_2 max) than in well-matched male subjects (Roepstorff et al., 2006b). This is likely due to the on average higher percentage of type I muscle fibers and higher capillarization in females than males (Roepstorff et al., 2006b).

The Different FA Sources Utilized during Exercise

The contribution from either of the different lipid sources to energy supply during exercise depends on several factors as exercise duration, intensity, training status, diet, and gender.

Both albumin-bound FA and VLDL-TG contribute to total energy turnover during prolonged exercise. At moderate exercise intensity (~50% of VO_2 peak), plasma FA concentration is increased several folds in a time-dependent manner as compared with resting levels and this is associated with an increase in FA oxidation (Ahlborg et al., 1974; Watt et al., 2002). Within recent years, data have emerged indicating that FA released from hydrolysis of VLDL-TG contributes to the total FA oxidation during exercise. When moderate exercise for 2 hours was restricted to a single muscle group (the knee extensors), VLDL-TG uptake, measured as femoral arteriovenous differences multiplied by plasma flow, amounted to approximately 67 μmol/min (Kiens et al., 1993). Also, when whole body exercise was performed for 1 hour at 50% of VO_2 max, VLDL-TG uptake in the leg amounted to 47 μmol/min (Enevoldsen et al., 2005). Taken together with recent data, where a tracer technique has been used to measure VLDL-TG oxidation in muscle, this suggests a small but consistent contribution of FA from VLDL-TG of 5–10% of total energy expenditure during prolonged exercise of moderate exercise intensities. Data also indicate that extracellular FA sources cover gradually more and more of the combusted energy during prolonged exercise at moderate intensities.

The importance of IMTG as an energy substrate for muscle during exercise has been discussed for years because of inconsistent findings in different studies. The different findings can be attributed to different study designs such as intensity, duration and mode of exercise, training status, dietary status, and sex of the subjects—each situation causing use of IMTG to a different extent. Besides, different methods for IMTG measurements have been used (for review see Kiens, 2006). In contrast, in the resting muscle the IMTG pool may be a central component of skeletal muscle lipid metabolism. Thus, earlier findings suggested that plasma FA taken up by the muscle at rest are esterified to IMTG prior to oxidation in the mitochondria (Dagenais et al., 1976). In support of this, a recent study revealed that upon uptake by the muscle FA originating from the plasma were not directly converted to long-chain acyl carnitine (LCAC) and oxidized but traversed the IMTG pool prior to oxidation in resting female and male subjects (Kanaley et al., 2009). Whether this is also the case during different exercise intensities remains to be elucidated. When exercise at a moderate intensity (57% VO_2 max) was performed for 4 hours, IMTG was measured after 2 and 4 hours of exercise. After 2 hours of exercise a decrease in IMTG of 13% was observed, but interestingly, during the following 2 hours of exercise no further decrease in IMTG content was seen (Watt et al., 2002). The attenuation in IMTG breakdown was associated with an increase in plasma FA concentration indicating that FA for oxidation increasingly originates from extracellular sources as exercise progresses (Watt et al., 2002). It is not clear why an attenuation of IMTG hydrolysis takes place during prolonged exercise. One suggestion could be that the available FA first transverse the IMTG pool prior to oxidation, as seen at rest, resulting in no net IMTG breakdown or that the increased FA availability in muscle inhibits lipolysis. These speculations need to be elucidated.

Effect of Exercise Intensity on FA Metabolism

When whole body exercise was studied in endurance-trained men by the use of isotopic tracer techniques, the results confirmed the classical findings of Christensen and Hansen (1939) that total FA oxidation decreased when exercise intensity increased above about 65% of VO_2 max (Romijn et al., 1993; van Loon et al., 2001) (Figure 12.2). Thus, the progressive shift toward CHO oxidation at increasing intensities is an indication of a limitation in FA utilization during high-intensity exercise.

The limitations in FA utilization during high-intensity exercise could be due to either a decrease in circulating FA concentrations for oxidation and/or a limitation in the ability of skeletal muscle to oxidize FA.

Delivery of plasma FA to the working muscle during exercise is a combination of perfusion of working muscle and plasma FA concentration which is a function of adipose tissue lipolysis. However, muscle blood flow repeatedly has been shown to increase concomitantly with increasing exercise intensity (Andersen & Saltin, 1985; Calbet et al., 2007; Helge et al., 2007), so this does not seem to be limiting for FA oxidation during exercise, at least not at exercise intensities up to 85% of VO_2 peak. Therefore, a decrease in FA delivery to the working muscle could be due to either a decreased lipolysis in adipose tissue or an inadequate perfusion of adipose tissue leading to accumulation of FA in adipose tissue and subsequent re-esterification into TG (Frayn, 2010). Lipolysis in adipose tissue was, however, similar when male subjects exercised at 65% and 85% of VO_2 peak (Romijn et al., 1993). On the other hand, findings by Bülow and Madsen (1981) demonstrated that the high sympathoadrenal response during whole body high-intensity exercise induced a reduction in adipose tissue blood flow resulting in a reduced release of FA from adipose tissue to the circulation (Bulow & Madsen, 1981). Therefore, the observation that arterial FA concentration does not increase much during high-intensity exercise is likely due to an inadequate perfusion of adipose tissue and thereby less albumin for circulating FA transport, rather than decreased adipose tissue lipolysis.

The question is then whether delivery of systemic albumin-bound FA to the working muscle is limiting for FA oxidation during exercise at high intensities. Romijn et al. (1995) artificially increased plasma FA concentrations to 2 mmol/l in men during exercise at 85% of VO_2 peak, by intravenous infusion of a lipid emulsion and heparin (the latter activates LPL in plasma) and showed that FA oxidation was increased only by 27% compared to exercise at the same intensity without infusion of lipids. However, FA oxidation did not reach the same level as seen at an exercise intensity of 65% of VO_2 peak, despite the increased FA availability (Romijn et al., 1995). These findings were further extended by van Loon et al. (2001), who reported a decrease in total FA oxidation during high-intensity exercise (72% VO_2 peak) compared to moderate intensities at 44% and 55% of VO_2 peak, despite no change in plasma FA availability. These findings suggest that the reduced FA oxidation observed with increasing intensities during exercise with large muscle groups cannot fully be explained by a decreased delivery of FA to the working muscle and therefore points to a limitation within the working muscle. Interestingly, when exercise was restricted to a limited muscle mass (the knee extensors) *plasma FA oxidation* increased with increasing exercise intensity from 25% up to 85% of maximum leg work capacity (Helge et al., 2007). These findings show that skeletal muscle does in fact have the capacity to oxidize FA at high intensities, at least up to 85% of maximal leg work capacity in the presence of a high blood flow and oxygen supply. In the study by Helge et al. (2007), the *total FA oxidation* remained unchanged during increasing intensities, demonstrating that the increase in plasma FA oxidation during high-intensity exercise displaced other sources of FA for oxidation. This implies that with increasing exercise intensities plasma FA was preferentially oxidized at the expense of other FA sources. These findings are supported in studies where whole body exercise was performed for 4 hours at a moderate intensity (57% VO_2 peak) where IMTG breakdown was attenuated in association with increased FA availability (Watt et al., 2002).

Taken together, these findings show that FA delivery to the working muscle is important for FA oxidation during exercise, but the reduced FA oxidation and shift toward CHO oxidation during exercise at high intensities cannot fully be explained by a decreased delivery of FA to the working muscle and therefore points to a limitation within the working muscle. However, the study by Helge et al. (2007) engaging a small muscle mass in exercise suggests that the muscle does have the capacity to oxidize FA even at high workloads when perfusion is unrestricted and whole body sympathetic drive is low. The reason for the decrease in total fat oxidation during high-intensity exercise when engaging a

large muscle mass therefore points to other external systemic factors limiting fat oxidation.

A limiting step in skeletal muscle FA oxidation could be the transport of circulating FA across the plasma membrane. Recent studies have suggested a role for FAT/CD36 in the acute increase in FA uptake in skeletal muscle seen in the transition from rest to exercise (Bonen et al., 2000; Jeppesen et al., 2011). Still, a number of observations indicate that FA transport across the sarcolemma is not limiting for FA oxidation. For instance, when exercise intensity on a cycle ergometer was increased from 65% to 90% of VO_2 peak, an accumulation of intramuscular FA was observed in healthy males, despite a decrease in plasma FA concentration, suggesting a barrier for FA oxidation within the muscle cell (Kiens et al., 1999). Furthermore, when subjects were studied during exercise in two different trials with either low or high pre-exercise muscle glycogen content, a twofold higher FA oxidation was shown during exercise in the low glycogen trial compared with the high glycogen trial, without changes in FA uptake measured across the working limb with stable isotopes (Roepstorff et al., 2004). These findings support the notion that regulation of FA oxidation lies downstream of FA uptake, as FA uptake capacity exceeds the capacity for FA oxidation in skeletal muscle.

Taken together, these findings indicate that FA transport across sarcolemma is not limiting for FA oxidation at high exercise intensities and point more toward a regulatory step in or around the mitochondria being limiting for FA oxidation during exercise at high intensities.

Mitochondrial Metabolism

Regulation of long-chain FA entry into mitochondria is a highly regulated process, as acyl-CoA derivatives cannot pass the mitochondrial inner membrane directly. First they have to be converted to their acylcarnitine form, a reaction catalyzed by carnitine palmitoyltransferase 1 (CPT-1) located at the outer mitochondrial membrane. Earlier findings demonstrated that CPT-1 was potently regulated by malonyl-CoA (Bird & Saggerson, 1984). Furthermore, a close relationship between malonyl-CoA concentration in muscle and decreased FA oxidation was observed in both humans and rats under resting conditions (Bavenholm et al., 2000; Chien et al., 2000). However, the relationship between malonyl-CoA and FA oxidation, observed at rest, is less clear during exercise. Thus, in humans muscle malonyl-CoA concentrations remained unchanged when exercise intensity was increased from 65% to 90% of VO_2 peak (Dean et al., 2000; Odland et al., 1996, 1998; Roepstorff et al., 2005). Furthermore, when male volunteers performed exercise at 65% VO_2 peak with either low or high pre-exercise muscle glycogen levels, marked differences in FA oxidation were seen without differences in muscle malonyl-CoA content. Taken together, this suggests that malonyl-CoA content is not the major regulator of FA oxidation in working muscle, at least in humans.

The activity of CPT-1 is dependent on the presence of the muscle metabolite carnitine (Figure 12.4). Besides the role of carnitine in mediating FA entry into mitochondria another important role in muscle is to protect against excess acetylation of the cellular CoA pool, by buffering the acetyl group and forming acetylcarnitine. Thus, carnitine serves as a sink for acetyl moieties and secures the necessary CoA concentration for metabolism (Stephens et al., 2007). During high-intensity exercise acetyl-CoA content in muscle is increased compared with rest (Constantin-Teodosiu et al., 1991; Odland et al., 1998), likely due to the inability of the tricarboxylic acid cycle (TCA) to handle the high amount of acetyl-CoA formed by the increased glycolysis at high-intensity exercise. Furthermore, increasing exercise intensities resulted in increased muscle concentrations of acetylcarnitine and a decrease in free carnitine content (Constantin-Teodosiu et al., 1991; Sahlin, 1990; van Loon et al., 2001). Consequently, less free carnitine in muscle will provide less substrate for CPT-1 and thereby limit the transport of FA into the mitochondria. This could be an important site of regulation in limiting FA oxidation at high exercise intensities. Recently it was shown that when pre-exercise muscle glycogen stores were low, providing few acetyl groups to carnitine, the content of muscle free carnitine and the rate of FA oxidation were significantly higher during moderate exercise than when the pre-exercise muscle glycogen content

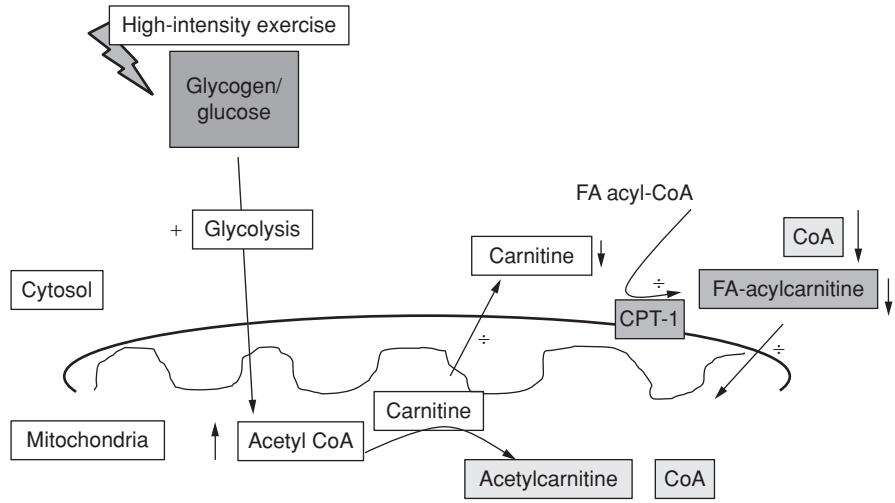

Figure 12.4 Schematic representation of the potential role of carnitine in the regulation of FA oxidation at high exercise intensities and high glycolytic rate.

was high and muscle free carnitine concentrations were low (Roepstorff et al., 2005). These findings give further support to the notion that availability of free carnitine is important for oxidation of FA and provides a potential mechanism in the regulation of FA oxidation.

The possible role of carnitine has led to the question if carnitine supplementation would increase muscle free carnitine and thereby increase the potential for muscle to oxidize FA which on the other hand would spare muscle glycogen utilization. For several years, attempts to increase skeletal muscle total carnitine content in humans by carnitine feeding have been unsuccessful (Vukovich et al., 1994; Wachter et al., 2002). But recently Wall et al. (2011) were the first to show that 24 weeks of supplement with L-carnitine together with the consumption of 80 g of carbohydrates twice a day resulted in a significant increase of 21% in muscle total carnitine whereas no changes were seen in untreated controls. In addition, supplementation resulted in muscle glycogen sparing during low-intensity exercise and a better matching of glycolytic and mitochondrial flux during high-intensity exercise. Unfortunately, lipid oxidation was not measured in this study, but the findings could suggest that an enhanced carnitine concentration in skeletal muscle would favor an increased lipid oxidation, at least during low-intensity exercise.

Postexercise Recovery

It is well established that muscle glycogen stores are depleted after prolonged moderate exercise (1.5–2 hours) when exercise is performed both continuously and intermittently. During the postexercise recovery period after glycogen-depleted exercise, it appears that oxidation of lipids covers more than 50% of total energy turnover as demonstrated by a low RER lasting several hours after exercise is discontinued. This high FA oxidation is observed despite consumption of a high CHO diet after exercise in order to replenish the glycogen stores (Bahr et al., 1990; Kiens & Richter, 1998; Kimber et al., 2003). The findings suggest that lipids are an important fuel in the postexercise period during which restoration of muscle glycogen has a high metabolic priority (Kiens & Richter, 1998). Thus, the glucose taken up by skeletal muscle is directed toward the depleted muscle glycogen stores rather than toward oxidation. The mechanisms behind this are not fully understood. The low glycogen stimulates glycogen synthase activity—the rate-limiting enzyme in glycogen synthesis—thereby increasing the

incorporation of glucose-6-phosphate into glycogen. Moreover, when a CHO-rich diet is consumed during the postexercise period, plasma insulin concentrations will be enhanced which at the same time will stimulate an increased translocation of the glucose transport protein GLUT4 from an intracellular location to the sarcolemma and thereby increase glucose uptake in skeletal muscle increasing the availability of glucose to glycogen synthesis. Meanwhile basal energy turnover is to a large extent covered by the different lipid energy sources. Both lipids from the circulation (plasma FA and VLDL-TG) and from IMTG seem to contribute to postexercise lipid oxidation. Thus, a net uptake of VLDL-TG was observed 4 hours after exercise, amounting to 37 μmol/min (Enevoldsen et al., 2005) and IMTG has also been shown in some studies to contribute to the total postexercise energy turnover (Kiens & Richter, 1998).

References

Ahlborg, G., Felig, P., Hagenfeldt, L., et al. (1974) Substrate turnover during prolonged exercise in man. Splanchnic and leg metabolism of glucose, free fatty acids, and amino acids. *Journal of Clinical Investigation* 53, 1080–1090.

Alsted, T.J., Nybo, L., Schweiger, M., et al. (2009) Adipose triglyceride lipase in human skeletal muscle is upregulated by exercise training. *American Journal of Physiology. Endocrinology and Metabolism* 296, E445–E453.

Andersen, P. & Saltin, B. (1985) Maximal perfusion of skeletal muscle in man. *Journal of Physiology* 366, 233–249.

Bahr, R., Hansson, P., & Sejersted, O.M. (1990) Triglyceride/fatty acid cycling is increased after exercise. *Metabolism* 39, 993–999.

Bavenholm, P.N., Pigon, J., Saha, A.K., et al. (2000) Fatty acid oxidation and the regulation of malonyl-CoA in human muscle. *Diabetes* 49, 1078–1083.

Bird, M.I. & Saggerson, E.D. (1984) Binding of malonyl-CoA to isolated mitochondria. Evidence for high- and low-affinity sites in liver and heart and relationship to inhibition of carnitine palmitoyltransferase activity. *Biochemical Journal* 222, 639–647.

Bonen, A., Luiken, J.J., Arumugam, Y., et al. (2000) Acute regulation of fatty acid uptake involves the cellular redistribution of fatty acid translocase. *Journal of Biological Chemistry* 275, 14501–14508.

Bulow, J. & Madsen, J. (1981) Influence of blood flow on fatty acid mobilization form lipolytically active adipose tissue. *Pflugers Archiv: European Journal of Physiology* 390, 169–174.

Calbet, J.A., Gonzalez-Alonso, J., Helge, J.W., et al. (2007) Cardiac output and leg and arm blood flow during incremental exercise to exhaustion on the cycle ergometer. *Journal of Applied Physiology* 103, 969–978.

Chien, D., Dean, D., Saha, A.K., et al. (2000) Malonyl-CoA content and fatty acid oxidation in rat muscle and liver in vivo. *American Journal of Physiology. Endocrinology and Metabolism* 279, E259–E265.

Christensen, E.H. & Hansen, O. (1939) Respiratorischer Quotient und O_2-Aufname. *Skandinavisches Archiv für Physiologie* 81, 160–171.

Constantin-Teodosiu, D., Carlin, J.I., Cederblad, G., et al. (1991) Acetyl group accumulation and pyruvate dehydrogenase activity in human muscle during incremental exercise. *Acta Physiologica Scandinavica* 143, 367–372.

Dagenais, G.R., Tancredi, R.G., & Zierler, K.L. (1976) Free fatty acid oxidation by forearm muscle at rest, and evidence for an intramuscular lipid pool in the human forearm. *Journal of Clinical Investigation* 58, 421–431.

Dean, D., Daugaard, J.R., Young, M.E., et al. (2000) Exercise diminishes the activity of acetyl-CoA carboxylase in human muscle. *Diabetes* 49, 1295–1300.

Enevoldsen, L.H., Simonsen, L., & Bulow, J. (2005) Postprandial triacylglycerol uptake in the legs is increased during exercise and post-exercise recovery. *Journal of Physiology* 568, 941–950.

Essen, B., Jansson, E., Henriksson, J., et al. (1975) Metabolic characteristics of fibre types in human skeletal muscle. *Acta Physiologica Scandinavica* 95, 153–165.

Frayn, K.N. (2010) Fat as a fuel: emerging understanding of the adipose tissue-skeletal muscle axis. *Acta Physiologica (Oxford, England)* 199, 509–518.

Glatz, J.F., Luiken, J.J., & Bonen, A. (2010) Membrane fatty acid transporters as regulators of lipid metabolism: implications for metabolic disease. *Physiological Reviews* 90, 367–417.

Haemmerle, G., Lass, A., Zimmermann, R., et al. (2006) Defective lipolysis and altered energy metabolism in mice lacking adipose triglyceride lipase. *Science* 312, 734–737.

Helge, J.W., Richter, E.A., & Kiens, B. (1996) Interaction of training and diet on metabolism and endurance during exercise in man. *Journal of Physiology* 492(Pt 1), 293–306.

Helge, J.W., Stallknecht, B., Richter, E.A., et al. (2007) Muscle metabolism during graded quadriceps exercise in man. *Journal of Physiology* 581, 1247–1258.

Jeppesen, J., Albers, P.H., Rose, A.J., et al. (2011) Contraction induced skeletal muscle FAT/CD36 trafficking and fatty acid uptake is AMPK independent. *Journal of Lipid Research* 52, 699–711

Kanaley, J.A., Shadid, S., Sheehan, M.T., et al. (2009) Relationship between plasma free fatty acid, intramyocellular triglycerides and long-chain acylcarnitines in resting humans. *Journal of Physiology* 587, 5939–5950.

Kiens, B. (2006) Skeletal muscle lipid metabolism in exercise and insulin resistance. *Physiological Reviews* 86, 205–243.

Kiens, B. & Richter, E.A. (1998) Utilization of skeletal muscle triacylglycerol during postexercise recovery in humans. *American Journal of Physiology* 275, E332–E337.

Kiens, B., Essen-Gustavsson, B., Gad, P., et al. (1987) Lipoprotein lipase activity and intramuscular triglyceride stores after long-term high-fat and high-carbohydrate diets in physically trained men. *Clinical Physiology* 7, 1–9.

Kiens, B., Essen-Gustavsson, B., Christensen, N.J., et al. (1993) Skeletal muscle substrate utilization during submaximal exercise in man: effect of endurance training. *Journal of Physiology* 469, 459–478.

Kiens, B., Roemen, T.H. & van der Vusse, G.J. (1999) Muscular long-chain

fatty acid content during graded exercise in humans. *American Journal of Physiology* 276, E352–E357.

Kimber, N.E., Heigenhauser, G.J., Spriet, L.L., et al. (2003) Skeletal muscle fat and carbohydrate metabolism during recovery from glycogen-depleting exercise in humans. *Journal of Physiology* 548, 919–927.

Maughan, R.J., Williams, C., Campbell, D.M., et al. (1978) Fat and carbohydrate metabolism during low intensity exercise: effects of the availability of muscle glycogen. *European Journal of Applied Physiology and Occupational Physiology* 39, 7–16.

Mendenhall, L.A., Swanson, S.C., Habash, D.L., et al. (1994) Ten days of exercise training reduces glucose production and utilization during moderate-intensity exercise. *American Journal of Physiology* 266, E136–E143.

Odland, L.M., Heigenhauser, G.J., Lopaschuk, G.D., et al. (1996) Human skeletal muscle malonyl-CoA at rest and during prolonged submaximal exercise. *American Journal of Physiology* 270, E541–E544.

Odland, L.M., Howlett, R.A., Heigenhauser, G.J., et al. (1998) Skeletal muscle malonyl-CoA content at the onset of exercise at varying power outputs in humans. *American Journal of Physiology* 274, E1080–E1085.

Roepstorff, C., Steffensen, C.H., Madsen, M., et al. (2002) Gender differences in substrate utilization during submaximal exercise in endurance-trained subjects. *American Journal of Physiology. Endocrinology and Metabolism* 282, E435–E447.

Roepstorff, C., Vistisen, B., Roepstorff, K., et al. (2004) Regulation of plasma long-chain fatty acid oxidation in relation to uptake in human skeletal muscle during exercise. *American Journal of Physiology. Endocrinology and Metabolism* 287, E696–E705.

Roepstorff, C., Halberg, N., Hillig, T., et al. (2005) Malonyl-CoA and carnitine in regulation of fat oxidation in human skeletal muscle during exercise. *American Journal of Physiology. Endocrinology and Metabolism* 288, E133–E142.

Roepstorff, C., Donsmark, M., Thiele, M., et al. (2006a) Sex differences in hormone-sensitive lipase expression, activity, and phosphorylation in skeletal muscle at rest and during exercise. *American Journal of Physiology. Endocrinology and Metabolism* 291, E1106–E1114.

Roepstorff, C., Thiele, M., Hillig, T., et al. (2006b) Higher skeletal muscle {alpha}2AMPK activation and lower energy charge and fat oxidation in men than in women during submaximal exercise. *Journal of Physiology* 574, 125–138.

Romijn, J.A., Coyle, E.F., Sidossis, L.S., et al. (1993) Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity and duration. *American Journal of Physiology* 265, E380–E391.

Romijn, J.A., Coyle, E.F., Sidossis, L.S., et al. (1995) Relationship between fatty acid delivery and fatty acid oxidation during strenuous exercise. *Journal of Applied Physiology* 79, 1939–1945.

Sahlin, K. (1990) Muscle carnitine metabolism during incremental dynamic exercise in humans. *Acta Physiologica Scandinavica* 138, 259–262.

Steffensen, C.H., Roepstorff, C., Madsen, M., et al. (2002) Myocellular triacylglycerol breakdown in females but not in males during exercise. *American Journal of Physiology. Endocrinology and Metabolism* 282, E634–E642.

Stephens, F.B., Constantin-Teodosiu, D., & Greenhaff, P.L. (2007) New insights concerning the role of carnitine in the regulation of fuel metabolism in skeletal muscle. *Journal of Physiology* 581, 431–444.

van Loon, L.J., Greenhaff, P.L., Constantin-Teodosiu, D., et al. (2001) The effects of increasing exercise intensity on muscle fuel utilisation in humans. *Journal of Physiology* 536, 295–304.

Vukovich, M.D., Costill, D.L., & Fink, W.J. (1994) Carnitine supplementation: effect on muscle carnitine and glycogen content during exercise. *Medicine & Science in Sports & Exercise* 26, 1122–1129.

Wachter, S., Vogt, M., Kreis, R., et al. (2002) Long-term administration of L-carnitine to humans: effect on skeletal muscle carnitine content and physical performance. *Clinica Chimica Acta* 318, 51–61.

Wall, B.T., Stephens, F.B., Constantin-Teodosiu, D., et al. (2011) Chronic oral ingestion of L-carnitine and carbohydrate increases muscle carnitine content and alters muscle fuel metabolism during exercise in humans. *Journal of Physiology* 589, 963–973.

Watt, M.J., Heigenhauser, G.J., Dyck, D.J., et al. (2002) Intramuscular triacylglycerol, glycogen and acetyl group metabolism during 4 h of moderate exercise in man. *Journal of Physiology* 541, 969–978.

White, L.J., Ferguson, M.A., McCoy, S.C., et al. (2006) Cardiovascular/non-insulin-dependent diabetes mellitus risk factors and intramyocellular lipid in healthy subjects: a sex comparison. *Metabolism* 55, 128–134.

Chapter 13

Metabolic Adaptations to a High-Fat Diet

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Introduction and Background

Regular endurance training results in skeletal muscle adaptations that increase rates of whole-body lipid oxidation and decrease sympathetic nervous system (SNS) activity in response to any given submaximal exercise intensity (Brooks & Mercier, 1984; Holloszy & Coyle, 1984). These adaptations enhance the ability of the trained musculature to oxidize all energy substrates. Overall though, they promote the oxidation of lipid-based fuels (i.e., adipose and intramuscular triglycerides (TGs) as well as blood-borne free fatty acids (FFAs) and TGs) in contrast to carbohydrate-based fuels (i.e., muscle and liver glycogen; blood glucose; and blood, muscle, and liver lactate), thereby attenuating the rate of muscle glycogenolysis (i.e., "glycogen sparing"). The increased reliance on lipid-based fuels is attributable to increased mitochondrial volume (Holloszy, 1967) along with increased mitochondrial enzymatic adaptations to transport and oxidize fat (Holloszy & Coyle, 1984). A reduction in the signals (free ADP and AMP) that activate the major enzymes that metabolize CHO (glycogen phosphorylase (PHOS), phosphofructokinase (PFK), and pyruvate dehydrogenase (PDH) (Spriet & Watt, 2003) also contributes to the increased reliance on lipid-based fuels. These adaptations to endurance training are well accepted. What is perhaps less well appreciated is that the short-term consumption of high-fat, low-carbohydrate (HFLC) diets by well-trained individuals in conjunction with daily training can also dramatically increase the rates of whole-body and muscle lipid oxidation, and "spare" muscle glycogen during submaximal exercise (for reviews, see Hawley, 2002, 2011; Hawley et al., 1998; Yeo et al., 2011).

A previous chapter in this series (Kiens & Helge, 2000) along with other reviews (Helge, 2000) focused on the effects of high-fat diets on endurance performance capacity in both rats and humans after dietary intervention periods lasting up to 7 weeks. This chapter provides a contemporary review on how short-term (~5 days) consumption of HFLC diets when undertaken in combination with daily physical activity can dramatically alter patterns of fuel utilization and subsequent exercise responses. Primary emphasis will be on the results of human studies and how HFLC diets interact with endurance training to modulate both acute and chronic responses to exercise, thereby promoting or inhibiting subsequent training capacity and performance.

Fat-Adaptation Protocols and Terminology: Dietary Periodization for the Athlete

The effect of consuming a high-fat (60–65% of energy intake), low-CHO (<20% of energy intake) diet for very short durations (<3 days) is to lower resting muscle and liver glycogen concentrations while concomitantly increasing rates of whole-body fat oxidation during low- to moderate-intensity

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(60–75% of maximal O_2 uptake, VO_2 max) exercise. Such short-term exposure to HFLC diets are detrimental to training capacity and subsequent endurance performance (Burke & Hawley, 2002; Helge, 2000). This is presumably due to the combination of lowered muscle glycogen availability and the absence of any substantial increase in the capacity of the muscle to oxidize fat during exercise, despite the increased FFA availability. Indeed, unlike carbohydrate oxidation, which is closely geared to the energy requirements of the working muscle, there are no mechanisms for matching the availability and utilization of FAs to the rate of energy expenditure. However, consumption of HFLC diets for longer periods elicits metabolic adaptations that significantly enhance rates of fat oxidation during both low- and moderate-intensity (<85% of VO_2 max) exercises (Burke & Hawley, 2002; Stepto et al., 2002) and, to a large extent, compensate for the short-term, diet-induced reduction in carbohydrate availability.

A shift to an increased oxidation of lipid-based fuels and decreased carbohydrate oxidation is desirable during prolonged, submaximal exercise and prolongs exercise capacity (i.e., the time taken to exercise to volitional fatigue at a given submaximal work rate (Pitsiladis et al., 1999). Carbohydrate is the more versatile fuel (it can contribute aerobic and anaerobic energy at all aerobic power outputs and contribute anaerobic energy when the power output exceeds the aerobic range). However, endogenous muscle and liver glycogen stores are limited to ~600 g (~10,000 kJ) in an endurance trained athlete and can be exhausted in as little as 2–3 hours of intense exercise when exercising in a fasted state (Coyle et al., 1986). Carbohydrate also has the advantage of a greater energy yield per unit of oxygen consumption—the energy yield per liter of oxygen consumed during fat oxidation is about 10% less than for carbohydrate (about 19 kJ/l O_2 for fat compared with 21 kJ/l O_2 for carbohydrate). Thus, any "sparing" of carbohydrate by upregulating lipid oxidation should, in theory, be advantageous assuming that the ultimate goal of prolonged exercise is to maintain muscle glycogen stores as long as possible and for the liver to provide glucose to the working muscles while maintaining euglycemia. Against this background, the concept of "dietary periodization" was proposed (Hawley & Hopkins, 1995) and refers to the strategy of consuming a high-fat, low-carbohydrate diet while undertaking an endurance-training program with the primary goal of promoting higher rates of fat utilization during exercise.

The term "fat adaptation" has been used generically to describe several diet–exercise protocols in which previously untrained and well-trained athletes consume an HFLC diet while undertaking endurance training (Helge, 2000; Lambert et al., 1994; Phinney et al., 1983). In recent years, HFLC dietary protocols have been refined and describe a diet–exercise practice whereby well-trained athletes ingest a fat-rich, low-carbohydrate diet for up to 5 days and then switch to a high-carbohydrate intake (i.e., carbohydrate restoration) 24–48 hours before an important event (Burke et al., 2000, 2002; Carey et al., 2001). Typically, studies of fat-adaptation and carbohydrate-restoration protocols have included a crossover design, whereby a control diet consisting of a high-carbohydrate diet (containing the same macronutrient composition as carbohydrate restoration) is compared with the fat adaptation and carbohydrate restoration diet (Figure 13.1). Furthermore, in these studies, participants were required to maintain their regular training program, including high-intensity training (HIT) sessions throughout the intervention period. Interestingly, even though the duration of various studies has ranged from 1–3 days with respect to the carbohydrate-restoration phase, it has been consistently shown that only 1 day of a high-carbohydrate diet and complete rest is necessary to restore muscle glycogen concentration in endurance-trained athletes (Burke et al., 2000; Stellingwerff et al., 2006; Yeo et al., 2008). From a practical perspective, this protocol (Figure 13.1) is an intervention that athletes/coaches are willing to adopt because of its brevity and lack of interference with a pre-event training/taper.

Fat Adaptation, Followed by Carbohydrate Restoration: Effects on Substrate Metabolism

Several recent and comprehensive reviews of the effects of an HFLC diet followed by carbohydrate restoration on fuel utilization during exercise

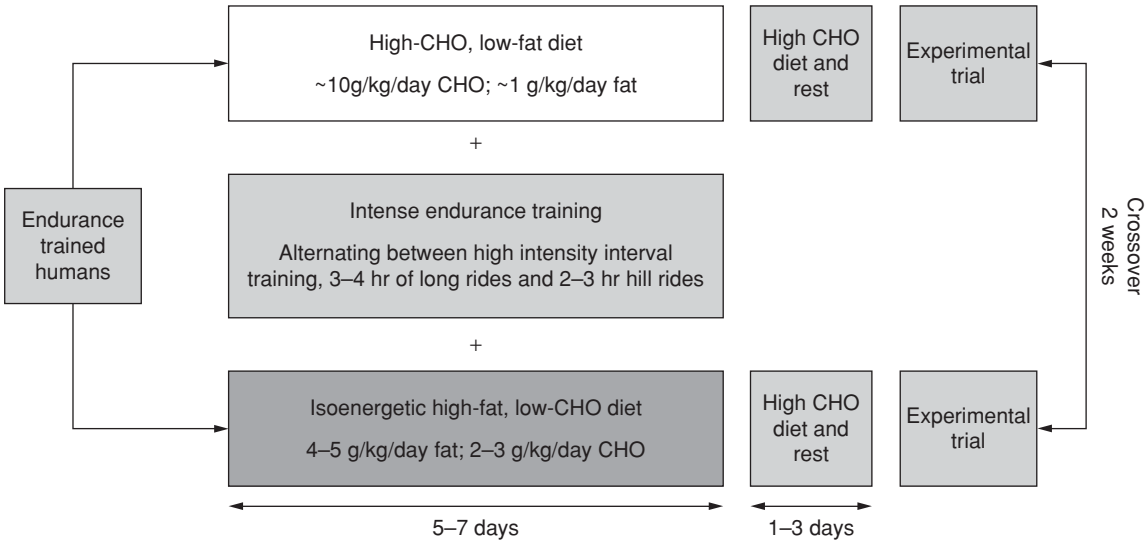

Figure 13.1 Schematic of the experimental design for studies utilizing the model of fat adaptation and carbohydrate restoration. CHO, carbohydrate.

can be found elsewhere (Hawley, 2011; Yeo et al., 2011). Of particular relevance here is that despite the short period of exposure (5 days) to a high-fat diet, rates of FA oxidation (compared to baseline) are substantially elevated in trained athletes. While acute alterations in carbohydrate and fat availability have previously been reported to shift patterns of fuel utilization in moderately trained individuals (Maughan et al., 1978), endurance-trained athletes already have a high capacity for fat oxidation and may have been expected to have maximized training-induced adaptations for FA metabolism. What is especially intriguing about the ability of this "dietary periodization" strategy to alter patterns of fuel utilization is that short-term exposure to an HFLC diet can increase rates of fat oxidation without a concomitant increase in mitochondrial volume. These results in humans are in contrast to those found in rats showing that prolonged (4 weeks) elevation of FFAs (i.e., a high-fat diet and injections of heparin to increase lipolysis and FFA availability) concurrently promote mitochondrial biogenesis and the capacity to oxidize FAs to a greater extent than chow-fed animals (Garcia-Roves et al., 2007). Taken collectively, these observations suggest that different mechanism(s) may be responsible for upregulating lipid oxidation after HFLC diets compared with traditional exercise training (i.e., going from an untrained to a trained state) and also that there are unique characteristics in the effects of dietary-training interactions between species. Of course, it is also important to note that fat-adaptation strategies represent as much a low-carbohydrate challenge (i.e., training in the face of low muscle glycogen availability) as they do a high-fat challenge (i.e., training with high fat availability) to cellular homeostasis as muscle glycogen contents are reduced during this adaptation phase. In this regard, training with low-carbohydrate availability (train low, compete high) has recently become popular in both scientific and athletic circles (for reviews, see Hawley & Burke, 2010; Hawley et al., 2011). Indeed, a major problem for the basic scientist when trying to unravel potential mechanism(s) underlying the benefit to training adaptation with either high-fat and/or reduced carbohydrate availability is the fact that carbohydrate restriction has reciprocal and pronounced effects on lipid availability, (i.e., increased circulating FFA concentrations and/or elevated muscle triacylglycerol levels).

Notwithstanding some of the problematic issues underlying the elucidation of the various mechanisms responsible for the shift to the oxidation of lipid- in preference to carbohydrate-based fuels, there are several issues that make the HFLC "dietary periodization" protocol a practical and attractive tool for athletes and coaches. First, the magnitude of increase in the rates of whole-body fat oxidation after short-term exposure to HFLC diets is similar to those observed after lengthier periods of dietary intervention. This is important from a health perspective because high-fat diets are associated with the development of muscle insulin resistance and pose risks even for individuals who undertake regular exercise (Hawley & Lessard, 2008). Second, elevated rates of fat oxidation persist even under conditions in which carbohydrate availability is increased by either having athletes consume a high-carbohydrate meal before commencing exercise and/or ingesting glucose solutions during exercise (Burke et al., 2002; Carey et al., 2001). This means an athlete could fat-adapt and then restore endogenous carbohydrate stores before a major event and then follow sport nutrition guidelines for increasing exogenous carbohydrate availability (i.e., pre-event meal and carbohydrate ingestion during exercise). While such a practice has a strong "metabolic underpinning" that should, theoretically, enhance performance, this is not the case! In laboratory-based settings in which a variety of exercise tests have been employed (i.e., the time to complete a fixed amount of work, or the time taken to complete a given distance), performance is similar between fat-adaptation protocols compared to isoenergetic high-carbohydrate diet interventions (discussed subsequently).

The majority of studies that have investigated the effects of HFLC diets have utilized competitive athletes who continue to train intensely while consuming diets containing a higher proportion of fat (4 g/kg/day, 65% of energy) than they would habitually ingest (Burke et al., 2000, 2002; Carey et al., 2001; Vogt et al., 2003; Yeo et al., 2008). To determine whether some of the metabolic adaptations that result from these extreme dietary perturbations can be obtained if athletes ingest diets in which the fat content is within the range typically chosen by trained individuals, Vogt et al. (2003) studied 11 endurance-trained athletes who ingested either a high-fat (50–55% of energy), high-carbohydrate or a low-fat (~20% of energy), high-carbohydrate diet for 5 weeks while undertaking their normal training regimen. Resting muscle glycogen content was not different between the two diets, but the high-fat diet resulted in a two-fold increase in resting muscle triacylglycerol content. Respiratory exchange ratio (RER) values were lower after the HFLC diet both at rest and during a range of submaximal exercise tests. However, despite similar glycogen values and metabolic adaptations favoring the use of lipid-based fuels, a variety of performance tests were not different after either diet interventions (Vogt et al., 2003).

Fat Adaptation Followed by Carbohydrate Restoration: Putative Mechanisms Underlying the Persistent Increase in Fat Oxidation

There are several potential cellular mechanisms that may explain the skeletal muscle adaptations induced by fat-adaptation strategies (Figure 13.2). These might reside with processes associated with either, or both, fat and carbohydrate oxidation and might be found at the level of substrate transport at the sarcolemma, substrate storage, breakdown of FA or glucose to acetyl-CoA, or mitochondrial transport.

Membrane Transport

The primary means of moving FAs into skeletal muscle cells is via the FA transporters, FA translocase (FAT/CD36) and plasma membrane fatty acid-binding protein (FABPpm). In one of the first studies to examine the cellular responses to HFLC diets, Cameron-Smith et al. (2003) reported that 5 days of fat adaptation increased both FAT/CD36 mRNA and protein content, although FABPpm mRNA and protein remained unchanged. At the time, these workers speculated that FAT/CD36 was more sensitive to changes in dietary fat content than FABPpm. In support of this contention, several studies have found that FAT/CD36 protein content returns to pre-fat-adaptation levels after a short

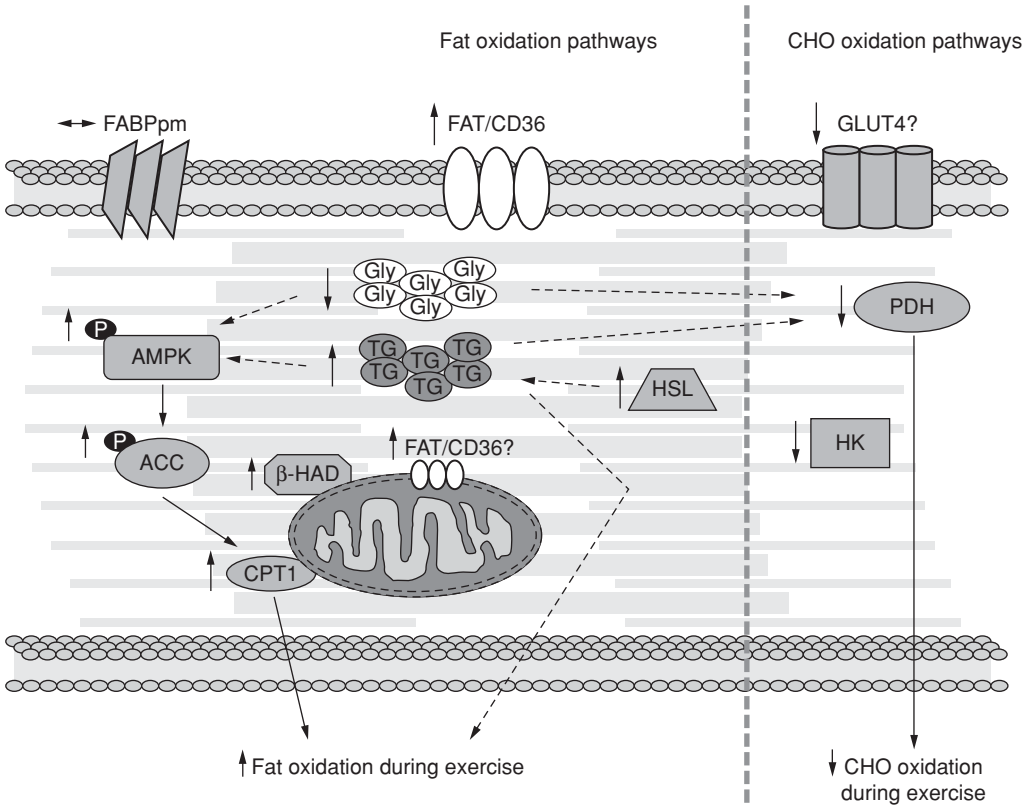

Figure 13.2 Fat adaptation-induced skeletal muscle adaptation with and/or without carbohydrate restoration. Solid lines signify experimentally verified evidence of valid perturbations after fat-adaptation studies. Dashed lines denote putative steps that are yet to be confirmed experimentally. FAT/CD36, fatty acid translocase; FABPpm, plasma membrane fatty acid-binding protein; CPT1, carnitine palmitoyltransferase I; GLUT4, glucose transporter type 4; Gly, glycogen; TG, intramuscular triglyceride; PDH, pyruvate dehydrogenase; HSL, hormone sensitive lipase; HK, hexokinase; AMPK, 5′ AMP-activated protein kinase; ACC, acetyl-CoA carboxylase; *β*-HAD, *β*-hydroxyacyl-CoA dehydrogenase; P, phosphorylation; ↑, increase; ↓, decrease; ↔, unchanged.

period of a high-carbohydrate diet (Yeo et al., 2008). However, it should be noted that these studies (Cameron-Smith et al., 2003; Yeo et al., 2008) have been limited to total muscle measures that provide no information regarding the compartmentalization or location at which the protein changes occurred. Recent work in humans has revealed that while high-intensity endurance training increased total muscle FABPpm and FAT/CD36, the changes at the sarcolemma were confined to FABPpm (Talanian et al., 2010). In addition, it has also been shown that the FA transport proteins can translocate to the membranes during exercise.

Mitochondrial FA Transport

Long-chain FAs require transport into mitochondria before they can be metabolized prior to their oxidation (Kiens, 2006). This process requires transport via the mitochondrial carnitine palmitoyl transferase (CPT) complex, with CPT1 believed to be the regulatory enzyme; the FA transport protein FAT/CD36 also appears to be involved. FABPpm on the mitochondrial membrane does not appear to play a role in FA transport but is structurally identical to aspartate aminotransferase, which is involved in shuttling reducing equivalents into

the mitochondria. Following training, the activity of CPT1 increased in the same proportion as the increase in mitochondrial content (Talanian et al., 2010). However, the amount of FAT/CD36 on the mitochondrial membrane increased to a greater extent than the increase in mitochondrial volume following training (Talanian et al., 2010). Increases in CPT1 activity have been demonstrated after 15 (Goedecke et al., 1999) and 28 days (Fisher et al., 1983) in response to HFLC diets.

The allosteric regulation of CPT1 activity is of great importance, at least at rest, in particular, the regulation of malonyl CoA (M-CoA)-mediated inhibition of CPT1 induced by the 5'AMP-activated protein kinase (AMPK) and the phosphorylation and suppression of the rate-limiting enzyme in M-CoA synthesis, acetyl-CoA carboxylase-b (ACC). Indeed the resting activation of AMPK and subsequent phosphorylation of ACC are increased by 5 days of a high-fat diet and 1 day of CHO restoration (Yeo et al., 2008). Given that the increase in AMPK activity and ACC phosphorylation were observed after carbohydrate availability was increased, it seems reasonable to suggest that these enzymes would be upregulated after fat adaptation alone. Thus, as noted previously (Yeo et al., 2011), the regulation of CPT1 activity by the AMPK-ACC-CPT1 axis might be an important mediator of the upregulation of FA oxidation induced by fat adaptation and might play a role in the persistent increase in fat oxidation observed after carbohydrate restoration.

Intramuscular Triacylglycerol Storage and Breakdown

Levels of glycogen and muscle TG storage can affect the rates of carbohydrate and fat oxidation during exercise (Spriet & Watt, 2003). The changes in muscle glycogen content in relation to fat adaptation and carbohydrate restoration have already been discussed. However, there are also likely to be many changes associated with the storage of TGs. Little current information exists, but TG levels have been reported to be increased by the dietary fat periodization protocol (Yeo et al., 2008). There was also a trend toward increased maximum activity of hormone-sensitive lipase (HSL) (20%) after fat adaptation followed by carbohydrate restoration (Stellingwerff et al., 2006). However, the overall TG content in muscles at rest following a training session ultimately depends on the balance between the rates of FA uptake, oxidation, and storage and the rate of TG hydrolysis. The esterification of FFA to TG requires acylation by acyl-CoA synthetase and the sequential addition of FFA to a glycerol backbone via a series of four enzymes, with the activities of glycerol-3-phosphate acyltransferase (GPAT) and diacylglycerol acyltransferase (DGAT) believed to be regulatory. On the breakdown side, adipose triacylglycerol lipase (ATGL) and HSL are believed to work hierarchically to regulate complete TG hydrolysis. ATGL initiates lipolysis by specifically removing the first FFA from TG to produce diacylglycerol substrate, which is then hydrolyzed by HSL to generate an additional FFA and monoacylglycerol (MG) substrate. MGs are converted to FFA and glycerol by MG lipase in the final step of lipolysis (Watt & Spriet, 2010). An increase in muscle TG levels following fat adaptation (Yeo et al., 2008) suggests increased synthesis over degradation during the rest periods between daily workouts.

Fat Adaptation: A Blunt Tool for Improving Endurance Performance?

A common finding from many of the studies reviewed is the mismatch between the changes in many of the laboratory-based measures (i.e., shifts in the patterns of substrate oxidation and/or increases in the expression of genes and proteins involved in fat metabolism and/or mitochondrial biogenesis) and whole-body functional outcomes (changes in training capacity or measures of performance). There could be several reasons to explain this disconnect. There may, of course, be no direct relationship between the performance of well-trained athletes and some of the diet–exercise-induced changes in selected variables that have been measured. Several diet–exercise interventions in both the acute and chronic setting can alter the balance between fat and carbohydrate oxidation at rest and during subsequent exercise, but they consistently fail to improve performance. In this regard, while it may be beneficial to increase rates of fat oxi-

dation and "spare" glycogen during training, top endurance athletes compete at such high speeds or power outputs that carbohydrate-based fuels predominate at the high exercise intensities they race at. Indeed, Spriet (2007) has speculated that "*it seems possible that top runners could complete the marathon using only carbohydrate as fuel.*"

It may be the fat-adaptation strategies that actually lead to an acute impairment in some measure of muscle/cellular function because of the complex interactions between pathways of substrate utilization; as systems to upregulate one pathway (i.e., lipid) occur, there may be a downregulation of others. In the case of "fat adaptation," there is direct mechanistic evidence to support such a contention. Stellingwerff et al. (2006) reported that 5 days of a high-fat diet followed by 1 day of "carbohydrate restoration" suppressed resting levels of pyruvate dehydrogenase (PDHa) activity by ~60% compared to an isoenergetic high-carbohydrate diet for 6 days. In response to a bout of submaximal cycling undertaken on day 7 (20 min at 70% of VO_2 max), there was a rapid increase in PDHa activity, which, although similar after both the diet interventions, remained 29% lower at the end of exercise after fat adaptation. Even in a 60-second sprint, an exercise challenge that would be expected to maximally activate muscle glycogenolysis, PDHa activity remained suppressed after fat adaptation ($p < 0.05$), even though the relative increase in PDH activation during the 1-minute sprint was similar between dietary treatments (~35%). As a result of less pyruvate oxidation (via a reduced PDH flux), estimates of glycogenolysis during both the first minute of submaximal exercise and the supramaximal sprint were also lower after fat adaptation. Clearly, the shift in muscle substrate use during submaximal and supramaximal cycling after fat adaptation involves both an upregulation of processes involved in FA metabolism, as well as downregulation of carbohydrate oxidation pathways.

Finally, there are many practical issues that surround the "fine-tuning" of fat-adaptation strategies before we can finally discount this intervention as a means to improve performance. For example, it remains to be tested experimentally whether a longer period of carbohydrate restoration is required in order to "rescue" the high-fat-diet-induced impairments to PDH activity so that rates of muscle glycogenolysis are not compromised during high-intensity work. The field-based observations of coaches suggest that there are positive responders to fat adaptation although scientists do not currently have evidence as to why this should be. For certain individual athletes, fat-adaptation protocols do enhance performance—it then becomes the job of exercise physiologists and sport nutritionists to optimize diet–exercise regimens for those athletes for whom dietary periodization strategies are beneficial.

References

Brooks, G.A. & Mercier, J. (1994) Balance of carbohydrate and lipid utilization during exercise: the "crossover" concept. *Journal of Applied Physiology* 76, 2253–2261.

Burke, L.M. & Hawley, J.A. (2002) Effects of short-term fat adaptation on metabolism and performance of prolonged exercise. *Medicine & Science in Sports & Exercise* 34, 1492–1498.

Burke, L.M., Angus, D.J., Cox, G.R., et al. (2000) Effect of fat adaptation and carbohydrate restoration on metabolism and performance during prolonged cycling. *Journal of Applied Physiology* 89, 2413–2421.

Burke, L.M., Hawley, J.A., Angus, D.J., et al. (2002) Adaptations to short-term high-fat diet persist during exercise despite high carbohydrate availability. *Medicine & Science in Sports & Exercise* 34, 83–91.

Cameron-Smith, D., Burke, L.M., Angus, D.J., et al. (2003) A short-term, high-fat diet up-regulates lipid metabolism and gene expression in human skeletal muscle. *American Journal of Clinical Nutrition* 77, 313–318.

Carey, A.L., Staudacher, H.M., Cummings, N.K., et al. (2001) Effects of fat adaptation and carbohydrate restoration on prolonged endurance exercise. *Journal of Applied Physiology* 91,115–122.

Coyle, E.F., Coggan, A.R., Hemmert, M.K., et al. (1986) Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *Journal of Applied Physiology* 61, 165–172.

Fisher, E.C., Evans, W.J., Phinney, S.D., et al. (1983) Changes in skeletal muscle metabolism induced by a eucaloric ketogenic diet. In *Biochemistry of exercise* (eds. H.G. Knuttgen, J.A. Vogel, & J. Poortmans), pp. 497–501. Human Kinetics, Champaign, IL.

Garcia-Roves, P., Huss, J.M., Han, D.H., et al. (2007) Raising plasma fatty acid concentration induces increased biogenesis of mitochondria in skeletal muscle. *Proceedings of the National Academy of Sciences of USA* 104, 10709–10713.

Goedecke, J.H., Christie, C., Wilson, G., et al. (1999) Metabolic adaptations to a high-fat diet in endurance cyclists. *Metabolism* 48, 1509–1517.

Hawley J.A. (2002) Effect of increased fat availability on metabolism and exercise capacity. *Medicine & Science in Sports & Exercise* 34, 1485–1491.

Hawley, J.A. (2011) Fat adaptation science: low-carbohydrate, high-fat diets to alter fuel utilization and promote training adaptation. *Nestle Nutrition Institute Workshop Series* 69, 59–77.

Hawley, J.A. & Burke, L.M. (2010) Carbohydrate availability and training adaptation: effects on cell metabolism. *Exercise and Sport Sciences Reviews* 38, 152–160.

Hawley, J.A. & Hopkins, W.G. (1995) Aerobic glycolytic and aerobic lipolytic power systems. A new paradigm with implications for endurance and ultraendurance events. *Sports Medicine* 19, 240–250.

Hawley, J.A. & Lessard, S.J. (2008) Exercise training-induced improvements in insulin action. *Acta Physiologica* 192, 127–135.

Hawley, J.A., Brouns, F., Jeukendrup, A. (1998) Strategies to enhance fat utilisation during exercise. *Sports Medicine* 25, 241–257.

Hawley, J.A., Burke, L.M., Phillips, S.M., et al. (2011) Nutritional modulation of training-induced skeletal muscle adaptations. *Journal of Applied Physiology* 110, 834–845.

Helge, J.W. (2000) Adaptation to a fat-rich diet: effects on endurance performance in humans. *Sports Medicine* 30, 347–357.

Holloszy, J.O. (1967) Biochemical adaptations in muscle. Effects of exercise on mitochondrial oxygen uptake and respiratory enzyme activity in skeletal muscle. *Journal of Biological Chemistry* 242, 2278–2282.

Holloszy, J.O. & Coyle E.F. (1984) Adaptations of skeletal muscle to endurance exercise and their metabolic consequences. *Journal of Applied Physiology* 56, 831–838.

Kiens, B. (2006) Skeletal muscle lipid metabolism in exercise and insulin resistance. *Physiological Reviews* 86, 205–243

Kiens, B. & Helge, J.W. (2000) Adaptations to a high-fat diet. In *Nutrition in Sport* (ed. R.J. Maughan), pp. 192–202. Blackwell Sciences, Oxford.

Lambert, E.V., Speechly, D.P., Dennis, S.C., et al. (1994) Enhanced endurance in trained cyclists during moderate intensity exercise following 2 weeks adaptation to a high fat diet. *European Journal of Applied Physiology and Occupational Physiology* 69, 287–293.

Maughan, R.J., Williams, C., & Campbell, D.M., et al. (1978) Fat and carbohydrate metabolism during low intensity exercise: effects of the availability of muscle glycogen. *European Journal of Applied Physiology and Occupational Physiology* 39(1), 7–16.

Phinney, S.D., Bistrian, B.R., Evans, W.J., et al. (1983) The human metabolic response to chronic ketosis without caloric restriction: preservation of submaximal exercise capability with reduced carbohydrate oxidation. *Metabolism* 32, 769–776.

Pitsiladis, Y.P., Smith, I., Maughan, R.J. (1999) Increased fat availability enhances the capacity of trained individuals to perform prolonged exercise. *Medicine & Science in Sports & Exercise* 31, 1570–1579.

Spriet, L.L. (2007) Regulation of substrate use during the marathon. *Sports Medicine* 37, 332–336.

Spriet, L.L. & Watt, M.J. (2003) Regulatory mechanisms in the interaction between carbohydrate and lipid oxidation during exercise. *Acta Physiologica Scandanavica* 178, 443–452.

Stellingwerff, T., Spriet, L.L., Watt, M.J., et al. (2006) Decreased PDH activation and glycogenolysis during exercise following fat adaptation with carbohydrate restoration. *American Journal of Physiology. Endocrinology and Metabolism* 290,E380–E388.

Stepto, N.K., Carey, A.L., Staudacher, H.M., et al. (2002) Effect of short-term fat adaptation on high-intensity training. *Medicine & Science in Sports & Exercise* 34, 449–455.

Talanian, J.L., Holloway, G.P., Snook, L.A., et al. (2010) Exercise training increases sarcolemmal and mitochondrial fatty acid transport proteins in human skeletal muscle. *American Journal of Physiology. Endocrinology and Metabolism* 299, E180–E188

Vogt, M., Puntschart, A., Howald, H., et al. (2003) Effects of dietary fat on muscle substrates, metabolism, and performance in athletes. *Medicine & Science in Sports & Exercise* 35,952–960

Watt, M.J., & Spriet, L.L. (2010) Triacylglycerol lipases and metabolic control: implications for health and disease. *American Journal of Physiology. Endocrinology and Metabolism* 299(2), E162–E168.

Yeo, W.K., Lessard, S.J., Chen, Z.P., et al. (2008) Fat adaptation followed by carbohydrate restoration increases AMPK activity in skeletal muscle from trained humans. *Journal of Applied Physiology* 105, 1519–1526.

Yeo, W.K., Carey, A.L., Burke, L., et al. (2011) Fat adaptation in well-trained athletes: effects on cell metabolism. *Applied Physiology, Nutrition, and Metabolism* 36, 12–22.

Chapter 14

Water and Electrolyte Loss and Replacement in Training and Competition

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Introduction

Water is often referred to as the "silent nutrient," reflecting the extent to which its presence and availability are taken for granted. As with all nutrients, an adequate intake is required for the body to maintain health, and deficiency symptoms and overdosage symptoms can both be observed. Water is the largest single component of the normal human body, typically accounting for about 50–60% of total body mass, and the turnover rate of water exceeds that of most other body components. For sedentary individuals living in a temperate climate, daily water turnover is typically about 2–2.5 liters, corresponding to the adequate daily intake established by the European Food Safety Authority (EFSA, 2010). This is about 5% of the total body water pool per day, but at times of thermal and exercise stress, such as occurs in men working in hot climates, daily sweat losses can reach 10–12 liters (Armstrong, 2000). In spite of its abundance and the high turnover, however, body water content must be maintained within narrow limits, and the body is much less able to cope with restriction of water intake than with restriction of food intake. A few days of total fasting has little impact on functional capacity if water is allowed and exercise is avoided, and even longer periods of abstinence from food are well tolerated. In contrast, cessation of water intake normally results in serious debilitation after times ranging from only a few hours to a few days at most. In only a very few reported cases have humans survived for longer than about 10 days without access to water.

The requirement for water is set by the total water losses from the body, for, except in the very short term, water intake must equal water loss. Water formed by the oxidation of foodstuffs will make some small contribution to meeting the daily water losses, but water in drinks and in foods will meet most of the demand. The major avenues of water loss are urine, feces, sweat, expired air, and through the skin: smaller losses occur in blood loss, tears etc., but these are normally trivial. When sweat losses are high, they can far exceed losses by all other routes combined. Along with water, several essential electrolytes are lost in sweat, and water balance is intimately associated with electrolyte (especially sodium) balance. The regulation of body water and electrolyte concentrations involves a number of neural and hormonal mechanisms that influence both intake and loss. Superimposed on these physiological control mechanisms are the various social and other factors that act to increase or restrict fluid intake.

Basal Water Requirements

All the major textbooks of nutrition and physiology include data on the different components of water intake and output, but it is difficult to find the original data on which these values are based. The Geigy Scientific Tables (Lentner, 1981) suggest that

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the minimum daily water requirement for adults to sustain physiological function is about 1.5 liters, including water from foods and water of oxidation, or about 1 ml per kcal of energy expenditure, assuming no unnecessary exercise. Body size clearly has a major influence on water turnover, but the total body water content will also be markedly affected by the body composition. The water content of lean tissue is about 72–75%, but adipose tissue contains only 10–20% water. It is expected, therefore, that there will be differences between men and women and between adults and children. Unless otherwise specified, the values given here will relate to the average 70 kg male with a moderate body fat content and a total body water content of about 42 liters. About two-thirds of this water is inside the cells (intracellular water) and about one-third in the interstitial space and plasma (extracellular water).

Environmental conditions will affect the basal water requirement by altering the losses that occur by the various routes. Water requirements for sedentary individuals living in the heat may be two- or threefold higher than the requirement when living in a temperate climate, even when this is not accompanied by pronounced sweating (Adolph et al., 1947). Transcutaneous and respiratory losses will be markedly influenced by the temperature and humidity of the ambient air. These losses are relatively small in the resting individual in a warm, moist environment (amounting to about 200 ml/day), but will be increased approximately twofold in regions of low humidity, and may be as high as 1500 ml/day during periods of hard work in the cold dry air at high altitudes (Ladell, 1965). To these losses must be added insensible loss through the skin (about 600 ml/day) and fecal loss (about 100 ml/day). Urine loss, which will not usually be less than about 800 ml/day, will include the obligatory volume required to excrete excess solute, plus any additional amount will account for the remaining fluid in excess of the body's requirement.

Variations in the amount and type of food eaten have some effect on water requirements because of the resulting demand for renal excretion of excess electrolytes and the products of metabolism. An intake of electrolytes in excess of the amounts lost in sweat and feces must be corrected by excretion in the urine, with a corresponding increase in the volume and/or osmolality of urine formed. The maximum urine concentrating capacity varies with individuals, and declines with age, but is typically about 1000–1200 mosmol/kg. The daily intake of electrolytes is subject to a wide variation between individuals, with strong trends for differences between different geographical regions. Daily dietary sodium chloride intakes for 95% of the young male UK population fall between 3.8 and 14.3 g with a mean of 8.4 g; the corresponding values for young women are 2.8–9.4 g, with a mean value of 6.0 g (Gregory et al., 1990). For the same population, mean urinary sodium losses were reported to amount to about 175 mmol/day (Gregory et al., 1990), which is equivalent to about 10.2 g of sodium chloride. Assuming a urine osmolality of 800 mosmol/kg, each extra gram of sodium chloride intake (assuming that none of this additional salt is lost in sweat) would require the formation of an extra 43 ml of urine to prevent a rise in urine osmolality.

There are also large differences between countries in the actual and recommended intakes of salt: the British advice is for a maximum of 6 g/day, but in Germany, a maximum of 10 g/day is recommended. In contrast, Sweden recommends a maximum of 2 g/day, and Poland recommends a minimum of 1.4 g/day. These differences in the recommendations by different expert committees reflect in part different interpretations with regard to the evidence linking salt intake and health, but also reflect regional consumption patterns. In addition to these differences between countries in salt intake, there are clearly large inter-individual differences that will have implications for daily water requirements. We have reported large inter-individual variations in sweat salt losses in football players in training (Maughan et al., 2005; Shirreffs et al., 2005); there is some evidence to suggest that high salt losses in sweat are not driven by high dietary intakes.

High-protein and high-salt diets require a greater urine output to allow excretion of nonvolatile solute: this effect is relatively small compared with other losses, but may become meaningful when water availability is limited or when renal function is compromised. Assuming a daily protein intake of about 113 g (corresponding to 15% of the energy content of

a 3000 kcal diet), and further assuming that all of this is oxidized, about 40 g of urea must be excreted. To excrete this at a urine osmolality of 800 mosmol/kg would require a urine output of about 800 ml. Some athletes, however, ingest a high-protein diet, and daily protein intakes of strength athletes may reach 2.5 g/kg or even more (see Chapter 10). This corresponds to a daily protein intake of 250 g for a 100 kg athlete; applying the same calculations as those used above, this will require a urine volume of 1770 ml. Even at the maximum urine concentration capacity of about 1200 mosmol/kg, a volume of 1060 ml would be required. In practice, however, even a wide range of sodium (10–400 mmol/day) or protein (80 or 180 g/day) intake was found to have no effect on water intake or urine volume in healthy men (Luft et al., 1983), reflecting the fact that urine output is normally well above the obligatory minimum volume.

The water content of food ingested will also be influenced greatly by the nature of the diet, and the water associated with food, typically, contributes 20–30% of daily intake (EFSA, 2010). The water content of a tomato, for example, is higher (93.1%) than that of a cola drink (89.8%) on a w/w basis (Holland et al., 1991). Some water is obtained from the oxidation of nutrients, and this will depend on the total metabolic rate, but will also be influenced by the nature of the substrate being oxidized (Table 14.1). Assuming an energy expenditure of 3000 kcal/day, a diet that is 50% carbohydrate, 35% fat, and 15% protein, will give about 400 ml of water per day (Table 14.2). Reducing the energy expenditure to 2000 kcal, but keeping the same diet composition will give about 275 ml of water. The contribution of this water of oxidation to water requirements is thus appreciable when water turnover is low, but becomes rather insignificant when water turnover is high.

Table 14.1 Energy and water of oxidation from nutrients

Foodstuff	Calories (cal/g)	Water of oxidation (ml/g)
Protein	4.1	0.42
Carbohydrate	4.1	0.60
Fat	9.3	1.07
Alcohol	7.0	1.17

Table 14.2 Water from food oxidation

1500 kcal from CHO	400 g CHO	240 g water
1050 kcal from fat	117 g fat	125 g water
450 kcal from protein	113 g protein	47 g water
		Total = 412 g

These calculations are based on a total energy intake of 3000 kcal/day, with 50% of the total energy intake provided by carbohydrate (CHO), 35% by fat, and 15% by protein, and assume that the fuels oxidized match exactly the dietary intake.

Control of Water Balance

Water intake and excretion are driven by a complex interaction of neural and hormonal factors, which respond to a number of different inputs. Under normal conditions, the blood volume and the osmolality of the extracellular fluid are maintained within narrow limits: a rise or fall of about 5 mosmol/l in the plasma osmolality is sufficient to switch the kidney from maximum conservation of water to a maximal diuresis (Levy & Lightman, 1997). Sodium, as the major ion of the extracellular space, accounts for about 50% of the total plasma osmolality, with the result that maintenance of osmotic balance is closely coupled to the intake and excretion of both sodium and water. The profound diuresis that ensues when plasma osmolality falls is normally sufficient to prevent water overload, but hyponatremia can occur when a conscious effort is made to override the physiological signals and ingest large volumes of plain water or other low-electrolyte fluids. Some degree of hyponatremia, for example, may be a normal accompaniment to ingestion of large volumes of beer, which is essentially sodium-free (Flear et al., 1981).

The kidneys can conserve water or electrolytes by reducing the rate of loss, but cannot restore a deficit. The subjective sensation of thirst initiates the desire to drink and hence plays a key role in the control of fluid balance. Thirst, whether measured as a perceived response or as the outcome (i.e., the volume of fluid consumed) appears to be relatively insensitive to acute changes in hydration status in man, but the overall stability of the total water volume of an individual indicates that the desire to drink is a powerful regulatory factor over the long term (Adolph et al., 1947).

Thirst may not be a direct consequence of the physiological need for water intake, but can be initiated by a number of unrelated factors, including habit, ritual, taste or desire for nutrients, stimulants, or a warming or cooling effect. A number of the sensations associated with thirst are learned, with signals such as dryness of the mouth or throat inducing drinking, while distention of the stomach can stop ingestion before a fluid deficit has been restored (Grossman, 1990). However, the underlying regulation of thirst is done separately by both the osmolality and volume of the body fluids and is, therefore, regulated by the same mechanisms that affect water and solute reabsorption in the kidneys and control central blood pressure. The thirst control centers, located in the hypothalamus and forebrain, play a key role in the regulation of both thirst and diuresis. Receptors in these centers respond directly to changes in plasma osmolality, blood pressure, and blood volume, while others are stimulated by the fluid balance hormones, which also regulate renal excretion (Phillips et al., 1985). The level of neural activity in the thirst control centers regulates the relative sensations of thirst and satiety, and can also influence urine output. Input from the higher centers of the brain can override these physiological mechanisms, and may lead to inappropriate drinking behavior. Where excessive volumes of fluid are consumed, dilutional hyponatremia ensues, with consequent morbidity and mortality (see Chapter 44).

A rise of 2–3% in plasma osmolality from the normal level of 285–290 mosmol/l is sufficient to evoke a profound sensation of thirst coupled with an increase in the circulating concentration of ADH (Grossman, 1990). The mechanisms that respond to changes in intravascular volume and pressure are less sensitive, reflecting the large fluctuations in blood volume and pressure that occur during normal daily activity.

When a water deficit has incurred, the normal drinking response in man involves a period of rapid ingestion during which 50% or more of the total intake is consumed, followed by a longer period during which intermittent consumption of relatively small volumes of the drink occurs (Verbalis, 1990). The initial alleviation of thirst occurs before significant amounts of the beverage have been absorbed into the body pools. This indicates a role for receptors in the mouth, esophagus, and stomach, which are thought to be responsive to the volume of fluid ingested; distension of the stomach also tends to reduce the perception of thirst (Verbalis, 1990). These pre-absorptive signals appear to be behavioral, learned responses and may be disrupted in unfamiliar situations. This may partly explain the inappropriate voluntary fluid intake in individuals exposed to an acute increase in environmental temperature or to exercise-induced dehydration. In the longer term, a fall in plasma osmolality and increase in the extracellular volume inhibit the sensation of thirst.

Impact of Exercise on Water Balance

Exercise elevates the metabolic rate, and only about 20–25% of the energy made available by the metabolic pathways is used to perform external work, with the remainder being dissipated as heat. The normal resting oxygen consumption is about 4 ml/kg body mass/min; for a 70 kg individual, this gives a resting rate of heat production of about 60–70 W. Running a marathon in 2 hours 30 minutes results in an average rate of heat production of about 1100 W sustained throughout the race for the average runner with a body mass of 70 kg (Maughan & Leiper, 1983). Heat loss mechanisms are invoked to limit the potentially harmful rise in core temperature. A high skin temperature facilitates heat loss by radiation and convection, but these mechanisms are effective only when the ambient temperature is low and the rate of air movement over the skin is high (Leithead & Lind, 1964). At high ambient temperatures (above about 35°C), skin temperature will be below ambient temperature, so heat is gained from the environment by this route, leaving evaporation as the only mechanism by which heat can be lost from the body. Evaporation of 1 liter of water from the skin will remove 2.4 MJ (580 kcal) of heat from the body. For the 2 hour 30 minute marathon runner with a body mass of 70 kg to balance the rate of metabolic heat production by evaporative loss alone, requires evaporation from the skin at about 1.6 liters/h. At such high sweat rates, some sweat drips from the skin without evaporating, and a

sweat secretion rate of about 2 liters/h is likely to be necessary to achieve this rate of evaporative heat loss. This is possible, but would result in the loss of 5 liters of body water over the course of the whole race, corresponding to a loss of more than 7% of body weight for a 70 kg runner. Water will also be lost by evaporation from the respiratory tract. During hard exercise in a hot, dry environment, this can amount to a significant water loss, although it is not generally considered to be a major heat loss mechanism in man.

In most activities, the energy demand varies continuously; average sweat losses for various sporting activities in different environments are well categorized (Rehrer & Burke, 1996). Even at low ambient temperatures, high sweat losses are sometimes observed when the energy demand is high, as in marathon running. Galloway and Maughan (1997) reported a sweat rate of 0.55 liters/h in subjects exercising at 80% of maximum oxygen uptake in a cold (4°C) environment while wearing only shorts and shoes. It cannot be concluded, therefore, that dehydration is a problem only when the ambient temperature and humidity are high. The sweat loss is, however, closely related to the environmental conditions, and substantial fluid deficits are much more common in the summer months and in tropical climates. In a group of professional soccer players training in a warm (32°C) environment, sweating rate was reported to vary from 1.1 to 2.2 liters/h, even though all completed the same training session (Shirreffs et al., 2005). Body mass losses of 6 liters or more have been reported for marathon runners in warm-weather competition (Costill, 1977), corresponding to a water deficit of about 8% of body mass, or about 12–15% of total body water. In spite of the large variation between individuals, sweating rate was found to be related to running speed in a heterogeneous group of marathon runners (Figure 14.1); there was, however, no relationship between total sweat loss and time to complete the race (Maughan, 1985).

Women tend to sweat less than men under standardized conditions, even after a period of acclimatization (Wyndham et al., 1965). It is likely, however, that a large part of the apparent sex difference can be accounted for by differences in body size, fitness and training, and acclimation status. There is a limited amount of information on the effects of age on the sweating response, and again levels of fitness and acclimation are confounding factors, but the sweating response to a standardized challenge generally decreases with age (Kenney, 1995). These observations should not be interpreted as suggesting an inability to exercise in the heat, nor should they be taken to indicate a decreased need for women or older individuals to pay attention to fluid intake during exposure to heat stress. The sweating capacity of children is low, when expressed per unit surface area, and the sweat electrolyte content is low relative to that of adults (Meyer et al., 1992), but the need for fluid and electrolyte replacement is no less important than in adults. Indeed, in view of the evidence that core temperature increases to a greater extent in children than in adults at a given level of dehydration, the need for fluid replacement may be greater in children (Bar-Or, 1989).

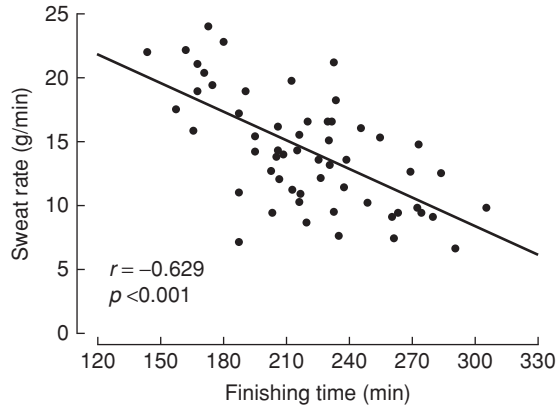

Figure 14.1 Estimated mean sweat rate in relation to average running speed (expressed as the time taken to complete the race) in a heterogeneous group of male runners who completed a marathon race (42.2 km) in times ranging from 2 hours 20 minutes to 5 hours 15 minutes. Redrawn from Maughan (1985), with permission from Georg/Thieme Verlag.

Dehydration, if sufficiently severe, is harmful to athletic performance, and both endurance sports and high-intensity events are adversely affected (see Chapter 15). Fluid losses are distributed in varying proportions among the plasma, extracellular water, and intracellular water. The decrease in plasma

volume, which accompanies dehydration, may be of particular importance in influencing work capacity; blood flow to the muscles must be maintained at a high level to supply oxygen and substrates, but a high blood flow to the skin is also necessary to convect heat to the body surface where it can be dissipated (Nadel, 1980). When the ambient temperature is high and blood volume has been decreased by sweat loss during prolonged exercise, there may be difficulty in meeting the requirement for a high blood flow to both these tissues. In this situation, skin blood flow is likely to be compromised, allowing central venous pressure and muscle blood flow to be maintained, but reducing heat loss and causing body temperature to rise (Rowell, 1986).

The preferential loss of water from the extracellular space reflects the relatively high sodium and chloride concentration in sweat. Electrolyte losses in sweat are a function of sweating rate and sweat composition, and both of these vary over time as well as being substantially influenced by the exercise intensity, environmental conditions, and the physiology of the individual. Added to this variability is the difficulty in obtaining a reliable estimate of sweat composition (Shirreffs & Maughan, 1997), and these methodological problems have contributed, at least in part, to the diversity of the results reported in the literature. Sweat is invariably isotonic with respect to plasma, and the major electrolytes are sodium and chloride, as in the extracellular space (Table 14.3). It is usual to present the composition in mmol/l, and the extent of the sodium losses in relation to daily dietary intake, which is usually expressed in grams, is not widely appreciated. Loss of 1 liter of sweat with a sodium content of 50 mmol/l represents a loss of 2.9 g of sodium chloride; the athlete who sweats 5 liters in a day, perhaps in two or more training sessions, may, therefore, lose almost 15 g of salt. We have reported losses of almost 9 g of sodium chloride in a footballer taking part in the second of two daily training sessions (Shirreffs et al., 2005). Even allowing for a reduced sweat sodium concentration and a decreased urinary output when sodium losses in sweat are large, this amount is large in comparison with the reported normal intakes quoted above. It is clear that the salt balance of individuals exercising in the heat may be precarious, and additional salt may usefully be added to food.

Table 14.3 Electrolyte concentration of normal human sweat in comparison with the composition of plasma

	Sweat (mmol/l)	Plasma (mmol/l)	Loss in 1 l of sweat (mg)
Sodium	20–80	130–155	460–1840
Potassium	4–8	3.2–5.5	160–320
Calcium	0–1	2.1–2.9	0–40
Magnesium	<0.2	0.7–1.5	<5
Chloride	20–60	96–110	710–2130

Source: Values for sweat composition are taken from Shirreffs and Maughan (1997).
The plasma composition is normally regulated within a narrow range, although some disease states are accompanied by large deviations from this.

Assessing Hydration Status and Water Loss

A simple, convenient method for assessment of hydration status would be useful, but there is no universal agreement on the optimum pre-exercise hydration status, nor is there a good index of euhydration that can be applied. The various options that can be used to assess hydration status have been described in detail by many authors, including Armstrong et al. (1994), Cheuvront and Sawka (2005), and Shirreffs (2000). The primary variables that are homeostatically regulated are blood volume and plasma osmolality, but both are subject to short-term variation in response to posture change, exercise, food and fluid intake, and a number of other factors, so neither is a good index of hydration status (Armstrong et al., 1994). Urine osmolality and specific gravity are less sensitive than plasma osmolality and show delayed responses, but Armstrong et al. (1994) showed that urine indices may be more sensitive to small changes in hydration status than are blood-derived indices, when measures are made over a period of days rather than minutes or hours. Using a scale of eight colors (Armstrong, 2000), it was concluded that a linear relationship existed between urine color, and both specific gravity and osmolality of the urine and that urine color could be

used in field settings to estimate hydration status. A few dietary factors may confound urine color, but these can normally be accounted for. A urine osmolality of more than about 900 mosmol/kg is consistent with a body water deficit of about 2% of body mass (Shirreffs & Maughan, 1998). The American College of Sports Medicine position stand suggests that a urine osmolality equal to or less than 700 mosmol/kg is indicative of euhydration (Sawka et al., 2007). Bioimpedance methods can be used to estimate total body water content, and have attracted much attention, as the equipment required is relatively inexpensive, and the technique is minimally invasive. However, acute changes in body water content are not reliably detected by the method, and it is sensitive to changes in posture, skin temperature, and other factors unrelated to body water, limiting its use for hydration monitoring (Institute of Medicine, 2005).

The first sample passed in the morning on rising is frequently selected as the criterion sample (Cheuvront & Sawka, 2005), but hydration status may change markedly between waking and the first training session of the day, especially if this takes place later in the day. The longer the interval between waking and training, the greater the probability that the waking urine sample will not reflect hydration status at the beginning of training. Collection of urine samples from football players reporting for training (Shirreffs et al., 2005) or match play (Maughan et al., 2007a) suggest that many players arrive for training or competition already dehydrated.

Urine measurements of hydration status will always be rather imprecise and single values may be of limited usefulness, but an individual who has a consistently high urine osmolality when about to begin a training session or competition is likely to be hypohydrated to some degree. For this reason, regular monitoring of playing squads has become routine in some sports. There is some evidence of a positive correlation between pre-training urine osmolality and the volume of fluid ingested during a training session where fluids are freely available (Maughan et al., 2005). This seems logical, as athletes who begin training with a higher urine osmolality may experience a greater sensation of thirst and, therefore, drink more, but this relationship has not been seen in all populations studied.

Acute changes in body water content in training or competition lasting not more than a few hours can be estimated from changes in body mass, though there are some qualifications (Maughan et al., 2007b).

Water Replacement

When the rate of water loss is low, substantial periods of time may elapse before the water deficit reaches a level where there may be an impact on physiological function. Water replacement can, therefore, be episodic, and replacement can be achieved by increasing the fluid intake with meals or by increasing the intake of foods with high water content. An increase in water requirement of 2 liters per day may represent a 100% increase in water turnover for the average individual, but, where food and water of oxidation contribute 50% of the baseline total daily water turnover, this means a threefold increase in the amount of liquids that must be drunk. This requires a major behavioral change and it is likely that euhydration will not be maintained without a conscious effort to increase drinking.

The major issues in fluid replacement for active individuals and for athletes are optimization of hydration status prior to exercise, provision of fluid and substrate (and possibly also other nutrients) during exercise, and rehydration and recovery after exercise. The kidneys effectively prevent attempts to increase the body water content prior to exercise, and although the addition of osmotically active substances such as glycerol to ingested fluids can decrease the fraction of ingested fluid that appears promptly in the urine, exercise performance is probably not enhanced (Latzka et al., 1996).

Dilute glucose-electrolyte solutions are the most effective way of replacing water losses when rapid rehydration is desired; variations on this formulation are the basis of oral rehydration solutions for the treatment of infectious diarrhea and of sports drinks (Maughan, 1997). The rate of gastric emptying of such solutions is slowed in proportion to the carbohydrate content, and concentrated solutions will be unable to deliver water at high rates.

Intestinal water absorption is driven by osmotic gradients and by solvent drag resulting from active absorption of solute, especially glucose and sodium, which are cotransported by an ATP-dependent mechanism. Hypotonic (200–250 mosmol/kg) solutions containing glucose and sodium will maximize the rate of water absorption, but hypertonic solutions will cause a temporary secretion of water into the intestinal lumen, exacerbating any existing hypohydration (Schedl et al., 1994).

The ingestion of water during exercise can help reduce the fall in plasma volume that normally occurs; this in turn helps to maintain cardiac output by maintaining stroke volume and increases skin blood flow, which helps promote heat loss and limits the rise in core temperature (Montain & Coyle, 1992; Montain et al., 1995). Although ingestion of plain water can help to improve exercise performance, further benefits are observed if glucose or glucose plus electrolytes are added (Maughan, 1997).

Both sweat losses and drinking behaviors vary greatly, even when environmental conditions, exercise demands, and fitness levels are similar, but sweat loss generally exceeds fluid intake (Maughan et al., 2005). Football (soccer) players in training may lose 1–3 liters of sweat in a single training session, but most of these players successfully maintain fluid balance on a daily basis even when training twice daily (Shirreffs et al., 2005). It is apparent, however, that some participants in very prolonged events, such as triathletes, ultramarathon runners, and the slower participants in marathon runs, consume excessive amounts of fluid (Almond et al., 2005). These amounts may be sufficient to induce hyponatremia, which is observed primarily in slower, female runners (Hew, 2005). It is also clear from studies of marathon runners that the incidence of hypohydration and hypernatremia is likely to be greater than that of hyperhydration and hyponatremia (Kratz et al., 2005), albeit serious adverse consequences are rare.

Rehydration is a vital part of the recovery process after exercise that has resulted in sweat loss in excess of fluid intake (Maughan et al., 1997). There is now compelling evidence that it is necessary to ingest significantly more volume than the net fluid deficit if euhydration is to be achieved, and it is also essential to replace the electrolytes lost in sweat if the ingested fluid is to be retained rather than being lost in the urine (see Chapter 16).

Measurement of Water Turnover

There appear to be few reliable measurements of water turnover in normal healthy individuals, and there are formidable obstacles that tend to confound the results of measurements of the various components of water intake and water loss. Intake is usually estimated from weighed or measured food and fluid intake diaries, but these data are complicated by uncertainties in the water content of foodstuffs, and are subject to the usual underreporting and other reliability issues. Measurement of the various avenues of water loss is beset by similar problems.

Application of isotopic tracer methodologies allows the noninvasive measurement of water turnover, using deuterium as a tracer for body water. Body water is labeled with deuterium, and the water turnover is measured over a period of a few days from the rate of decrease in the tracer concentration in body water. The measurement can be made conveniently on blood or urine samples. Collection of 24-hour urine output allows nonrenal losses (consisting mainly of sweat and transcutaneous losses, respiratory water loss, and fecal loss) to be calculated by difference.

Application of this method to two groups of subjects, one sedentary and one physically active, showed a higher rate of water turnover in the exercising group (Leiper et al., 1996). The active group comprised men with sedentary occupations who ran or jogged a mean distance of 103 km during the week of the study; subjects in the sedentary group had a similar age, height, weight, and body water content, and were engaged in similar occupations, but undertook no physically demanding activities in their leisure time. The median daily water turnover (averaged over 7 days) in the active group was 4673 ml/day, which was higher than that of the sedentary subjects (3256 ml/day). These data are shown in Figure 14.2. The average daily urine loss was greater in the exercising group (3021 ml) than in the sedentary group (1883 ml). It might have been expected that the runners would have a greater

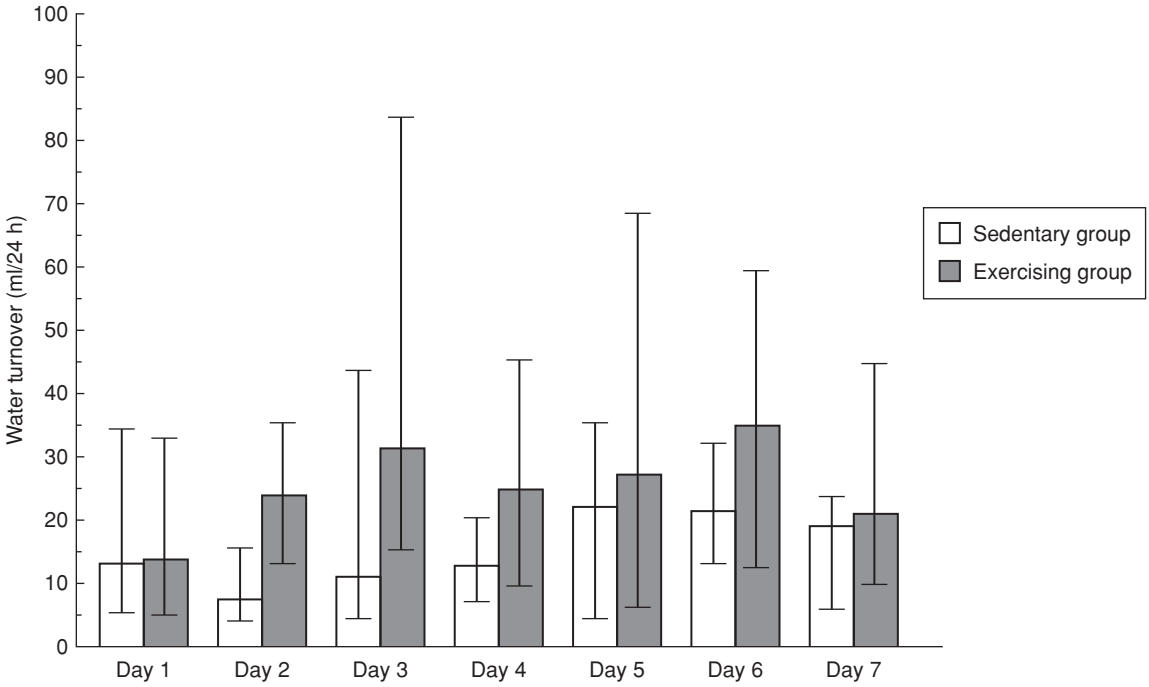

Figure 14.2 Median (range) daily water turnover rates for sedentary and physically active middle-aged men over a 7-day period. Data from Leiper et al. (1996).

daily sweat loss than the sedentary group, and that this would be reflected in a greater total nonrenal loss. There was, however, no significant difference between the groups in nonrenal water losses (runners, 1746 ml; sedentary, 1223 ml), although some tendency for there to be a difference ($p < 0.08$) was observed. These results seem surprising, but may reflect the relatively low total exercise load of the runners and the temperate climate (mean maximum daytime temperature of 14 (7–21)°C) at the time of the study. The results also suggest that the runners were habituated to drinking a volume of fluid in excess of that required to match the sweat loss incurred during exercise.

The same methodology has been applied to another physically active group, in this case cyclists covering an average daily distance of 50 km in training for competition, and another matched sedentary group (Leiper et al., 2001). Again, the median water turnover rate was higher in the active group (3.38 liters/day) than in the sedentary individuals (2.22 liters/day), but there was no difference between the groups in the daily urine output (cyclists, 1.96 liters; controls, 1.90 liters). The nonrenal losses were greater in the cyclists (1.46 liters/day) than in the sedentary group (0.53 liters/day). It was again rather cool during the measurement period, with maximum daily temperatures of 10 (4–18)°C, which might account for the rather low sweat rates in spite of the high physical activity level of the cyclists.

These studies emphasize the variability in the normal pattern of fluid intake and loss in both sedentary and active individuals. In all cases, body mass remained rather constant throughout the 7-day measurement period, suggesting that subjects were maintaining normal hydration status.

Diuretic Agents

Caffeine and alcohol are both part of the normal diet of a large part of the population, including many athletes. Both of these compounds act as diuretics to stimulate water loss in urine. It has been estimated

that 1 g of alcohol causes an extra 10 ml of urine to be formed and that each mg of caffeine causes an excess urine output of 1.1 ml, but these values are very crude approximations. In individuals habituated to caffeine intake, a dose of less than about 250–300 mg is unlikely to have a significant diuretic effect, though such doses can enhance exercise performance (see Chapter 25). The effects of alcohol depend on many factors, including the form in which it is consumed. Dilute alcohol solutions, such as weak beer will provide water in excess of the diuretic effect of their alcohol content, though stronger drinks such as wines and spirits should generally be avoided where hydration might be compromised (see Chapter 27).

Conclusion

High levels of physical activity are associated with high rates of metabolic heat production, and the sweating response is invoked to limit the rise in body temperature, especially when the ambient temperature is high. Basal levels of water turnover for healthy humans living in a temperate climate are about 2–4 liters per day, but this can be increased to something in excess of 10–12 liters per day during periods of hard physical work in hot climates. Such situations are encountered in occupational and military tasks as well as in sports training and competition. A water deficit is poorly tolerated, and a deficit of as little as 1% of body mass may impair exercise tolerance. Replacement of losses is, therefore, vital, but the relative insensitivity of the thirst mechanism may make this difficult to achieve in practice. Control of fluid balance is intimately linked with electrolyte (especially sodium) balance, and maintenance of hydration when sweat rates are high and requires replacement of electrolyte losses as well as the volume loss.

References

Adolph, A. & Associates (1947) *Physiology of Man in the Desert*. John Wiley & Sons, New York.

Almond, C.S., Shin, A.Y., Foretscus, E.B., et al. (2005) Hyponatremia among runners in the Boston marathon. *New England Journal of Medicine* 352, 1550–1556.

Armstrong, L.E. (2000) *Performing in Extreme Environments*. Human Kinetics, Champaign, IL.

Armstrong, L.E, Maresh, C.M., Castellani, J.W., et al. (1994) Urinary indices of hydration status. *International Journal of Sport Nutrition* 4, 265–279.

Bar-Or, O. (1989) Temperature regulation during exercise in children and adolescents. In *Perspectives in Exercise Science and Sports Medicine. Vol 2: Youth, Exercise, and Sport* (ed. C.V. Gisolfi & D.R. Lamb), pp. 335–362. Benchmark Press, Indianapolis, IN.

Cheuvront, S.N. & Sawka, M.N. (2005) Hydration assessment of athletes. *Sports Science Exchange* 97(2), 1–8.

Costill, D.L. (1977) Sweating: its composition and effects on body fluids. *Annals of the New York Academy of Sciences* 301, 160–174.

EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA). (2010) Scientific opinion on dietary reference values for water. *EFSA Journal* 8(3), 1459. doi:10.2903/j.efsa.2010.1459

Flear, C.T.G., Gill, C.V., & Burn, J. (1981) Beer drinking and hyponatraemia. *Lancet* 2, 477.

Galloway, S.D.R. & Maughan, R.J. (1997) Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Medicine & Science in Sports & Exercise* 29, 1240–1249.

Gregory, J., Foster, K., Tyler, H., et al. (1990) *The Dietary and Nutritional Survey of British Adults*. HMSO, London.

Grossman, S.P. (1990) *Thirst and Sodium Appetite*. Academic Press, San Diego, CA.

Hew, T.D. (2005) Women hydrate more than men during a marathon race: hyponatraemia in the Houston marathon: a report on 60 cases. *Clinical Journal of Sports Medicine* 15, 148–153.

Holland, B., Welch, A.A., Unwin, I.D., et al. (1991) *McCance and Widdowson's The Composition of Foods*, 5th edn. Royal Society of Chemistry, London.

Institute of Medicine. (2005) *Dietary Reference Intakes for Water, Potassium, Sodium, Chloride and Sulfate*, pp. 73–185. National Academy Press, Washington, DC.

Kenney, W.L. (1995) Body fluid and temperature regulation as a function of age. In *Perspectives in Exercise Science and Sports Medicine. Vol 8: Exercise in Older Adults* (eds. C.V. Gisolfi, D.R. Lamb, & E. Nadel), pp. 305–352. Cooper Publishing Group, Carmel, IN.

Kratz A., Siegel, A.J., Verbalis, J.G., et al. (2005) Sodium status of collapsed marathon runners. *Archives of Pathology and Laboratory Medicine* 129, 227–230.

Ladell, W.S.S. (1965) Water and salt (sodium chloride) intakes. In *The Physiology of Human Survival* (eds. O. Edholm & A. Bacharach), pp. 235–299. Academic Press, New York.

Latzka, W.A., Sawka, M.N., Matott, R.P., et al. (1996) Hyperhydration: Physiologic and Thermoregulatory Effects During Compensable and Uncompensable Exercise-Heat Stress. *Tech. Rep. No. T96-6*. US Army Research Institute of Environmental Medicine, Natick, MA.

Leiper, J.B., Carnie, A., & Maughan, R.J. (1996) Water turnover rates in sedentary and exercising middle-aged men. *British Journal of Sports Medicine* 30, 24–26.

Leiper, J.B., Pitsiladis, Y., & Maughan, R.J. (2001) Comparison of water turnover rates in men undertaking prolonged cycling exercise and in sedentary men. *International Journal of Sports Medicine* 22, 181–185.

Leithead, C.S. & Lind, A.R. (1964) *Heat Stress and Heat Disorders*. Casell, London.

Lentner, C. (1981) *Geigy Scientific Tables*, 8th edn. Ciba-Geigy Limited, Basle, Switzerland.

Levy, A. & Lightman, S. (1997) *Endocrinology*. Oxford University Press, Oxford.

Luft, F.C., Fineberg, N.S., Sloan, R.S., et al. (1983) The effect of dietary sodium and protein on urine volume and water intake. *Journal of Laboratory and Clinical Medicine* 101, 605–610

Maughan, R.J. (1985) Thermoregulation in marathon competition at low ambient temperature. *International Journal of Sports Medicine* 6, 15–19

Maughan, R.J. (1997) Optimising hydration for competitive sport. In *Perspectives in Exercise Science and Sports Medicine*, Vol. 10 (ed. D.R. Lamb & R. Murray), pp. 139–184. Cooper Publishing Group, Carmel, IN.

Maughan, R.J. & Leiper, J.B. (1983) Aerobic capacity and fractional utilisation of aerobic capacity in elite and non-elite male and female marathon runners. *European Journal of Applied Physiology* 52, 80–87

Maughan, R.J., Leiper, J.B., & Shirreffs, S.M. (1997) Factors influencing the restoration of fluid and electrolyte balance after exercise in the heat. *British Journal of Sports Medicine* 31, 175–182

Maughan, R.J., Shirreffs, S.M., & Merson, S.J. (2005) Fluid and electrolyte balance in elite male football (soccer) players training in a cool environment. *Journal of Sports Sciences* 23, 73–79.

Maughan, R.J., Watson, P., Evans, G.H., et al. (2007a) Water balance and salt losses in competitive football. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 583–594.

Maughan, R.J., Shirreffs, S.M., & Leiper, J.B. (2007b) Errors in the estimation of sweat loss and changes in hydration status from changes in body mass during exercise. *Journal of Sports Sciences* 25, 797–804.

Meyer, F., Bar-Or, O., MacDougall, D., et al. (1992) Sweat electrolyte loss during exercise in the heat: effects of gender and maturation. *Medicine & Science in Sports & Exercise* 24, 776–781.

Montain, S.J. & Coyle, E.F. (1992) Influence of graded dehydration on hyperthermia and cardiovascular drift during exercise. *Journal of Applied Physiology* 73, 1340–1350.

Montain, S.J., Latzka, W.A., & Sawka, M.N. (1995) Control of thermoregulatory sweating is altered by hydration level and exercise intensity. *Journal of Applied Physiology* 79, 1434–1439.

Nadel, E.R. (1980) Circulatory and thermal regulations during exercise. *Federation Proceedings* 39, 1491–1497.

Phillips, P.A., Rolls, B.J., Ledingham, J.G.G., et al. (1985) Osmotic thirst and vasopressin release in humans: a double-blind crossover study. *American Journal of Physiology* 248, R645–R650.

Rehrer, N.J. & Burke, L.M. (1996) Sweat losses during various sports. *Australian Journal of Nutrition and Dietetics* 53 (Suppl. 4), S13–S16.

Rowell, L.B. (1986) *Human Circulation*. Oxford University Press, New York.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.

Schedl, H.P., Maughan, R.J., & Gisolfi, C.V. (1994) Intestinal absorption during rest and exercise: implications for formulating oral rehydration beverages. *Medicine & Science in Sports & Exercise* 26, 267–280.

Shirreffs, S.M. (2000) Markers of hydration status. *Journal of Sports Medicine and Physical Fitness* 40, 80–84.

Shirreffs, S.M. & Maughan, R.J. (1997) Whole body sweat collection in man: an improved method with some preliminary data on electrolyte composition. *Journal of Applied Physiology* 82, 336–341.

Shirreffs, S.M. & Maughan, R.J. (1998) Urine osmolality and conductivity as markers of hydration status. *Medicine & Science in Sports & Exercise* 30, 1598–1602.

Shirreffs, S.M., Aragon-Vargas, L.F., Chamorro, M., et al. (2005) The sweating response of elite professional soccer players to training in the heat. *International Journal of Sports Medicine* 26, 90–95.

Verbalis, J.G., (1990) Inhibitory controls of drinking: satiation of thirst. In *Thirst: Physiological and Psychological Aspects* (eds. D.J. Ramsay & D.A. Booth), pp. 313–334. . ILSI Human Nutrition Reviews, Springer-Verlag, London.

Wyndham, C.H., Morrison, J.F., & Williams, C.G. (1965) Heat reactions of male and female Caucasians. *Journal of Applied Physiology* 20, 357–364.

Chapter 15

Performance Effects of Dehydration

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Introduction

The human body is a marvelously precise, efficient, and resistant machine capable of achieving outstanding physical performances when appropriately and specifically trained to achieve a given task. For instance, in 2011, Andreas Raelert established the Ironman triathlon world record with a time of 7 hours 41 minutes and 33 seconds. Patrick Makau, in 2011, set the marathon world record with a time of 2 hours 3 minutes and 38 seconds. In 2009, Usain Bolt ran the 100 m dash in a world record time of 9.58 seconds. In 1993, Javier Sotomayor established the high jump world record with a leap of 2.45 m (8 ft). Finally, Behdad Salimi, in 2011, set the snatch lift world record in the 105+ kg category with a lift of 214 kg. Few would disagree that such physical performances, encompassing the pinnacle of aerobic fitness and muscular endurance, strength, and power, require that all body's cells work in perfect unison under the most favorable internal environment possible.

In healthy adult humans, body water constitutes ~60% of body weight and is the most abundant compound in the body. It is essential for the transport of material inside the body and for biochemical reactions within cells. It is the most important solvent of the organism and is vital for lubrication of organs and joints, thermoregulatory processes, and the maintenance of the integrity of the cardiovascular system. Without drinking, a healthy individual is expected to die within 7–10 days, whereas one can go without foods for weeks or even months. Given the importance of body water in terms of physiological effects and the total mass it occupies within the body, it is reasonable to believe that, under particular circumstances, decreasing the body water content may have an impact on human physical performance.

Metabolism increases several fold during exercise, which produces heat that must be dissipated to the surrounding environment in order to prevent an excessive rise in body temperature. During hard exercise, especially in warm environments, heat is lost mainly through the evaporation of sweat and, to a lesser extent, by convection and radiation. Because athletes usually drink less than their rate of sweat production during exercise (Noakes, 1993), a phenomenon referred to as involuntary dehydration, hypohydration develops. On the other hand, several studies have reported that individuals often start exercising in an already hypohydrated state. Athletes, coaches, physical trainers, and exercise scientists need to have a deep and exercise-specific understanding of how hypohydration impacts human performance in order to maximize performance.

On these bases, the goal of the present chapter is to review and report the latest findings regarding the impact of exercise-induced dehydration on endurance performance and exercise capacity, and the effects of pre-exercise hypohydration on subsequent endurance performance, high-intensity

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aerobic exercise performance, and muscular strength, power, and endurance performances. A critical review of guidelines dedicated to improving athletes' performance through hydration strategies is performed. At the end of the chapter, performance-oriented fluid intake strategies with real-world applicability are proposed to athletes. There are many research gaps in the literature that, unfortunately, limit the capacity to make solid, evidence-based recommendations that apply to all exercise conditions, and those will be identified. It is hoped that they will serve as beacons for future research. Purposefully, few inferences are made as to how hypohydration influences physiological functions during exercise; this aspect has been covered in depth before (Casa, 1999; Cheuvront et al., 2003; Horswill, 1998; Noakes, 1993). Complete rehydration between exercise bouts may be critical to an athlete's performance. To prevent redundancy, this topic has been deliberately omitted and is thoroughly covered in Chapter 16.

Euhydration refers to normal body water content, hypohydration to reduced body water content, and hyperhydration to increased body water content. The term aerobic exercise performance is used to refer both to measurements of endurance performance and endurance capacity. For convenience, throughout it is considered that a loss or gain in 1 g of body water is equivalent to a loss or gain in 1 g of body weight, though there may be significant errors in this assumption (Maughan et al., 2007a).

Fluid Intake Guidelines: History of the Past 15 Years

Closely inspecting and carefully reviewing the various fluid replacement guidelines that have been developed over the past years by sporting- and nutrition-related organizations with the main objective of maximizing athletes' performance is a powerful exercise to help identify the continuum of research developments, research progress, and lines of thinking that have defined this particular research field. But perhaps most importantly, when meticulously performed, it allows some of the statements made by organizations to be put into perspective and research gaps and limits of our understanding to be pinpointed, thereby providing guidance for where future research efforts should be heading.

By the 1940s it had already been demonstrated that physical performance was compromised by exercise-induced dehydration (Barr, 1999). However, it was not before 1996 that a worldwide-recognized organization, i.e., the American College of Sports Medicine (ACSM), decided to engage in the development and diffusion of fluid guidelines promoting the health, safety, and optimal physical performance of individuals participating in regular physical activity. Based on the evidence available at that time, it was ACSM's recommendation that "during exercise athletes should consume fluids at a rate sufficient to replace the water lost through sweating or consume the maximum amount that can be tolerated" (Convertino et al., 1996). Performance-wise, it was likely premature for ACSM to arrive at such a conclusion since, in 1996 there was a limited amount of research data on the effect of exercise-induced dehydration upon aerobic exercise performance and, additionally, findings were not entirely consistent (Below et al., 1995; Maughan et al., 1989; Robinson et al., 1995; Walsh et al., 1994).

In 2000, the National Athletic Trainers' Association (NATA) (Casa et al., 2000) advocated that "fluid replacement should approximate sweat and urine losses and at least maintain hydration at less than 2% body weight reduction." NATA's position should be viewed with caution, since their interpretation of research findings was not based on all the relevant information available and was confounded by studies in which hypohydration was induced before exercise.

In an update of their 1996 Position Stand, the ACSM proposed in 2007 "that the goal of drinking during exercise is to prevent excessive dehydration (>2% body weight loss from water deficit) … to avert compromised exercise performance" (Sawka et al., 2007). It is not clear why such a conclusion was reached. In fact, results of studies published up until 2007, which validly evaluated the effect of exercise-induced dehydration of less than 2% body weight on aerobic exercise performance were equivocal or unclear.

In 2009, the ACSM, along with the American Dietetic Association (ADA) and Dietitians of Canada

(DC) (Rodriguez et al., 2009), issued a Position Stand with a confusing and irregular message in that, in the abstract section it was written, "athletes should drink enough fluid during exercise to balance fluid losses." whereas in the key points section it stated that "the goal of drinking is to prevent dehydration (water deficit in excess of 2–3% body mass) from occurring during exercise."

Since the ACSM, NATA, ADA, and DC Position Stand releases, several other organizations also felt the need to develop and disseminate their own guidelines for fluid replacement during exercise. The International Marathon Medical Director Association (IMMDA) (Hew-Butler et al., 2006) issued in 2006 a provocative Position Stand in which it recommended that athletes should "always defer to physiologic cues to increase fluid intake (thirst) or decrease fluid consumption (increased urination, bloating, weight gain) while running." At that time, there was no direct scientific evidence supporting the idea that the act of drinking according to thirst could optimize endurance performance.

In their 2010 consensus, the International Olympic Committee recommended that athletes should "drink sufficient fluid during exercise to limit dehydration to less than about 2% of body mass," agreeing with ACSM's 2007 Position Stand.

Finally, in 2011, the International Association of Athletics Federations (IAAF) amalgamated the 2007 ACSM and 2006 IMMDA recommendations and concluded that both thirst-driven and programmed fluid consumption could be used complementarily by athletes to maximize endurance performance, depending on exercise circumstances and fluid availability. Unlike IMMDA's Position Stand, by the time the IAAF published their consensus there was evidence in the literature to support the idea that drinking to thirst could optimize endurance performance (Dugas et al., 2009; Goulet, 2011).

From the information provided by the different positions reviewed above, it is clear that the jury is still out, and that no clear and well-defined consensus has yet been reached as to how endurance athletes should hydrate during exercise to optimize performance. On the one hand, there are organizations that support the idea that athletes should enter an exercise while being fully aware of their fluid needs and try to do whatever they can to prevent a weight loss >2%. On the other hand, some believe that protecting body weight during exercise is irrelevant, and that all that is necessary for optimal performance is to follow thirst sensation to gauge the need for fluid replacement.

The well-accepted and vigorously-defended (Sawka & Noakes, 2007) ideas that (1) weight loss should be minimized during exercise, and that (2) by the time thirst has developed, performance has already started to decrease, are at the moment severely challenged by many. This, in large part, is a direct consequence of the fact that prior to 2009 researchers had developed no study decisively excluding the possibility that drinking to thirst (independent of weight loss) could not maximize aerobic exercise performance. The storm this situation has created will likely not calm down before the balance of scientific proof favors one strategy over the other or shows that a niche can exist for both. Unfortunately, such a debate in the literature is extremely perplexing and frustrating for athletes, coaches, and health practitioners and very clearly highlights the need for scientists to focus research efforts to resolve this issue in a timely manner.

There are, however, some recent findings, not taken into account by the aforementioned Position Stands or consensuses, that have substantially advanced our understanding of the effect of exercise-induce dehydration on endurance performance.

Aerobic Exercise Performance: Effect of Exercise-Induced Dehydration

The idea that exercise-induced dehydration >2% body weight decreases endurance performance originates from a 2003 review study published by researchers from the United State Army Research Institute of Environmental Medicine (USARIEM) (Cheuvront et al., 2003). Without discriminating between the different research constructs used by the various studies and relying only on a vote counting procedure, researchers established that the dehydration threshold for endurance performance decrement was 2% body weight. But as will be demonstrated, if it had not been for the presence

of studies using non-ecological research designs, i.e., exercise protocols where athletes are forced to exercise at a fixed-work rate throughout or for a part of the exercise protocol, such a conclusion could not and would not have been reached (Sawka & Noakes, 2007).

To examine and evaluate the effect of exercise-induced dehydration on aerobic exercise performance, over the past 23 years researchers have turned to the use of two laboratory-based exercise tests: (1) the traditional fixed-power output protocol, which provides a measure of endurance capacity and (2) the time-trial protocol, which provides an outcome of endurance performance (Goulet, 2011). The difference between the two exercise protocols is not trivial from a practical and scientific point of view, and has a major implication for the interpretation of research findings in this field. Fixed-exercise intensity protocols require subjects to either exercise at a fixed-power output until complete exhaustion or complete a high-intensity time-trial type exercise preceded by a bout of exercise conducted at a fixed-work rate. Obviously, the latter testing models have an inherently low ecological validity (Mündel, 2011), as under most training and racing situations susceptible to be affected by dehydration, athletes utilize pacing strategies in which speed varies throughout either on a macro- or micro-scale (Abbiss et al., 2010; Angus & Waterhouse, 2011; Lambert et al., 2004; Thomas et al., 2012). Moreover, there is no sporting event that requires athletes to exercise until complete exhaustion. Additionally, it has been demonstrated that fixed-power output tests to exhaustion have a poor reliability (Currell & Jeukendrup, 2008) as well as an unclear sensitivity (Amann et al., 2008; Laursen et al., 2002, 2003).

On the other hand, during time-trial type protocols, subjects are asked to complete a set distance or a given amount of work as fast as possible. Therefore, a time-trial protocol can be considered ecologically valid because it reflects what athletes are required to do during an actual race. This applies at least to a time-trial race: in a true race, the pace is often set by another competitor and the athlete must choose to follow this pace or to disregard it. Moreover, it has been demonstrated that laboratory-based cycling time-trial performances correlate well with outdoor cycling time trials (Palmer et al., 1996). Hence, when wishing to evaluate, comment, and make inferences about the effect of an intervention on performance during field conditions, only those studies using time-trial type research protocols can provide valid and meaningful information for competitive endurance athletes.

Fluid intake guidelines are primarily designed for, of interest to, and mainly used by competitive athletes exercising under real-world exercise conditions. Consequently, it makes sense that only findings deriving from studies using research designs closely mimicking outdoor exercise conditions should be utilized for the formulation of fluid replacement recommendations. Based on this premise, it appears quite clear that dehydration and aerobic exercise performance-related research findings must be reported not in an agglomerated fashion, but rather separately based on whether studies used time-trial or fixed-intensity exercise protocols.

Time-Trial Exercise Conditions and Exercise-Induced Dehydration: Effect on Endurance Performance

A careful review of the literature reveals that five laboratory-based studies have examined the effect of exercise-induced dehydration upon time-trial type exercise performance (Bachle et al., 2001; Backx et al., 2003; Dugas et al., 2009; Kay & Marino, 2003; Robinson et al., 1995). All of these studies were conducted between 1995 and 2009 and cycling was used as the exercise. The mean ambient temperature, relative humidity, exercise intensity, and duration of cycling trials were 26ºC (range: 20–33ºC), 61% (range: 50–72%), 68% of maximal oxygen consumption (VO_2 max) (range: 52–85% of VO_2 max), and 86 minutes (range: 60–127 minutes), respectively, which is concordant with exercise conditions athletes could face in everyday situations. The range of exercise-induced dehydration varied from 1% to 4% of body weight. Strikingly, none of these studies demonstrated that dehydration significantly hinders endurance performance.

In order to pin down the magnitude of the effect of exercise-induced dehydration on endurance performance, Goulet (2011) performed a meta-analysis

demonstrating that during cycling time trial, exercise-induced dehydration on average increased, albeit non-significantly, endurance performance by 0.06% (95% confidence interval: –1.42% to 1.54%), compared with a euhydrated state. Mean end-of-exercise weight loss was 0.4% for the well-hydrated condition, compared with 2.2% for the dehydrated condition. Moreover, Goulet (2011) showed that there was no significant difference in performance between studies with an end-of-exercise dehydration level of more than 2% or less than 2% of body weight. Using magnitude-based inference statistics and considering that the smallest deleterious change in power output that would matter to the performance of competitive long-distance cyclists is 1.6% (Paton & Hopkins 2006), Goulet (2011) concluded that an end-of-exercise dehydration level of 2.2% body weight should produce a trivial change in the performance of long-distance cyclists under field conditions.

As demonstrated in Figure 15.1, Goulet's meta-analysis established no significant relationship between changes in exercise-induced dehydration and changes in endurance performance (power output). However, it was observed that both exercise intensity and duration correlated with endurance performance, implying that these variables may have had a greater impact on endurance performance than exercise-induced dehydration in these studies.

Consequently, as of 2011, laboratory-based science has still been unable to show that exercise-induced dehydration of 1–4% of body weight impairs endurance performance in athletes undertaking exercise simulating real-world conditions. These findings corroborate those that have been observed in field-based studies, which will be reviewed in a later section.

Time-Trial Exercise Conditions and Exercise-Induced Dehydration: Endurance Performance and Thirst Sensation

It can be read in almost all popular magazines and scientific articles reviewing the topic of hydration and endurance performance that athletes should never feel thirsty during exercise and, hence, force fluid intake to prevent this sensation developing, which supposedly indicates that dehydration has reached a critical level and performance has already started to diminish. From a scientific point of view, this discourse makes sense for those believing that the underlying reason behind why exercise-induced dehydration diminishes endurance performance is

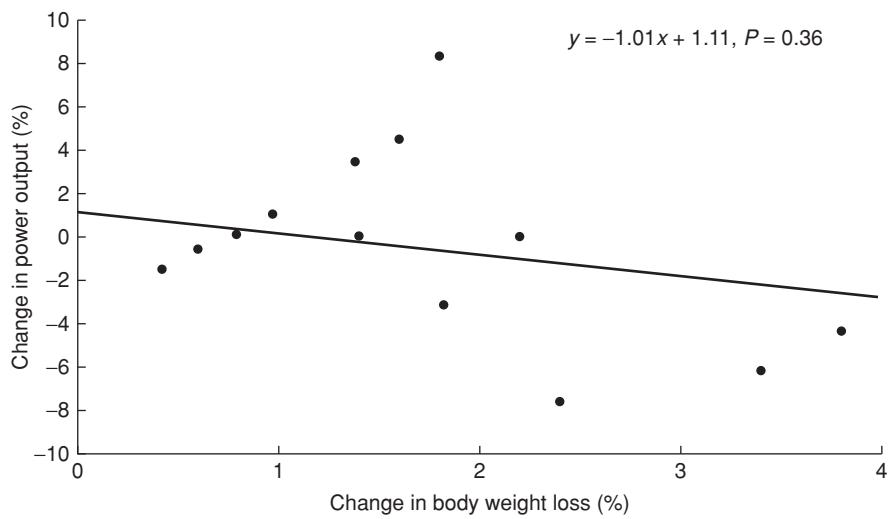

Figure 15.1 Correlation between the % changes in weight loss and the % changes in power output. From Goulet (2011), with permission from BMJ Publishing Group Ltd.

linked to body weight reduction and associated cardiovascular, thermoregulatory, neural, and metabolic changes, as implies the 2% body weight loss rule (Cheuvront et al., 2003). Given that research has clearly established that drinking according to thirst during exercise is insufficient to maintain body weight, then this message is critically important for athletes.

However, if it is the "internal milieu," or extracellular fluid osmolality, that is regulated during exercise, then drinking according to thirst sensation may be all that is necessary to maximize endurance performance, independent of the extent of the exercise-induced changes in body weight (Sawka & Noakes, 2007). Indeed, research has shown that the feeling of thirst is initiated with a plasma osmolality level that is still well within the normal range (Valtin, 2002). Moreover, drinking to thirst maintains plasma osmolality within physiological range for those with low as well as high sweat-sodium concentration (Brown et al., 2011).

As most studies in the literature have compared exercise situations where athletes either replaced all or none of their sweat losses, neither of which actually mimics the practice of most athletes, few data are therefore available to evaluate how endurance performance is affected when athletes rely on their thirst sensation to replace lost fluids. In that respect, only two studies, using time-trial type exercise protocols, provide evidence on this issue (Backx et al., 2003; Dugas et al., 2009). Using a meta-analytic procedure, Goulet (2011) has demonstrated (Figure 15.2) that drinking below thirst sensation significantly decreased (–5.2%), and drinking above thirst non-significantly decreased (–2.4%), endurance performance. Overall, compared with drinking below or above thirst sensation, following thirst to decide whether or not fluid is required during exercise, significantly improved endurance performance by 4.3%. From a practical point of view, these findings make sense and can be interpreted to suggest that prolonged, unquenched thirst as well as forced fluid intake reduce endurance performance.

Athletes are therefore encouraged to rely on their thirst sensation during exercise if the goal is to optimize performance. Moreover, drinking to thirst not only preserves extracellular fluid osmolality (Armstrong et al., 1997; Maresh et al., 2004; Merry et al., 2008), but it also prevents athletes from overdrinking during exercise, which may lead to severe gastrointestinal symptoms and ultimately, but rarely, lethal dilutional hyponatremia.

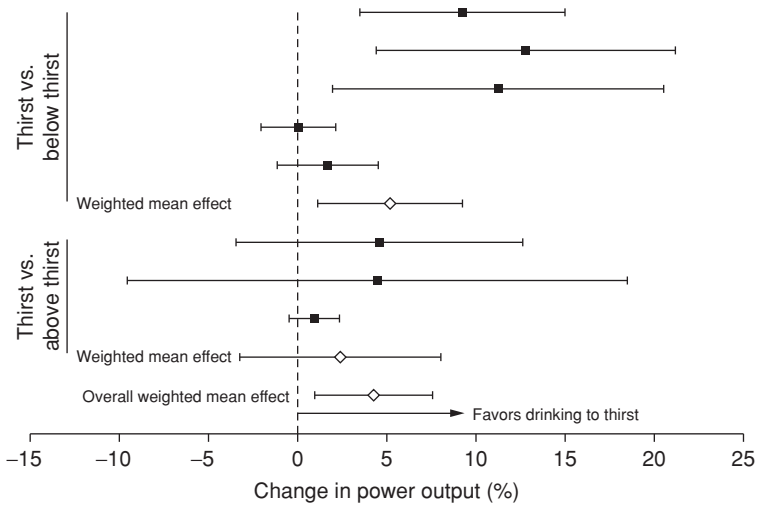

Figure 15.2 Forest plot showing how following thirst sensation as a means to gauge fluid replacement affects endurance performance, compared with rates of drinking below or above thirst sensation. The overall weighted mean effect represents the magnitude of effect observed when all effect estimates are combined together. ◊ represents the weighted mean effect estimate. A random-effects model was used. Results are means ± 95% confidence intervals. Based on data from Goulet (2011).

Time-Trial Exercise Conditions and Exercise-Induced Dehydration: 1 hour Endurance Performance

Time-trial exercises of 1 hour are conducted at very high exercise intensities, which drastically slows gastric emptying, intestinal absorption, and fluid integration rates (Maughan et al., 1990). Given these, some may wonder whether it would not be wiser for endurance athletes to limit or even completely eliminate fluid consumption during a 1 hour, high-intensity exercise.

In order to provide an objective answer to this question, Goulet (2011) performed a meta-analysis of relevant studies using time-trial type exercise protocols and found that fluid abstinence during such exercises increased (+0.6%), although non-significantly, endurance performance. These results clearly point to the fact that fluid intake is not required during 1 hour, high-intensity exercises, or that if fluid is consumed, the overall volume is not important (i.e., ≤150 ml).

Real-World Exercise Conditions and Exercise-Induced Dehydration: Effect on Endurance Performance

Although they have received little media or scientific attention, several field-based studies over the past 45 years have observed and reported the relationship between exercise-induced body weight loss and endurance performance. Albeit by the nature of their design, such studies do not allow the establishment of causal relationships, they nevertheless can help identify whether exercise-induced weight loss is associated either negatively or positively with endurance performance, especially if a pattern emerges across studies.

According to the well-accepted dogma, athletes with weight losses >2% should necessarily and systematically show degraded performances compared with those following drinking patterns preventing weight losses >2%. Results acquired under real-world exercise conditions do not support this assertion, and are in line with those of Goulet (2011).

In marathon runners, Zouhal et al. (2011) recently demonstrated a significant linear relationship between the degree of weight loss and race finishing time, such that athletes with the greatest weight loss had the best racing times (Figure 15.3). It should be noted, though, that only 26 of the 643 participants in this study completed the event in less than 3 hours, so the description of these individuals as athletes is perhaps misleading. Similarly, Sharwood et al. (2004) observed a significant relationship between Ironman-triathlon race finishing time and weight loss, so that athletes who finished the race with the highest dehydration level were also the fastest (Figure 15.4). The relationship between exercise-induced weight loss and 24-hour ultra-marathon performance was examined in 23 athletes by Kao et al. (2008). Again, authors identified a significant relationship between weight loss and performance. (Figure 15.5). However, over a 12-hour race, Kao et al. (2008) identified no relationship between exercise-induced weight loss and total distance run.

The Marathon of Sands is a 7-day, multi-stage running race conducted under extreme conditions in the Moroccan desert. During such a race, Zouhal et al. (2009) observed that the faster the racing times the greater the weight losses were at the end of the race. The fastest subject they studied completed the marathon in sixth position out of a total of 650 participants and his end-of-exercise body weight was 9% lower than his starting weight.

The top three finishers of the 1970 Commonwealth Games marathon consumed less than 100 ml of fluid during the race and became dehydrated by 4–5% of their body mass, yet completed the race

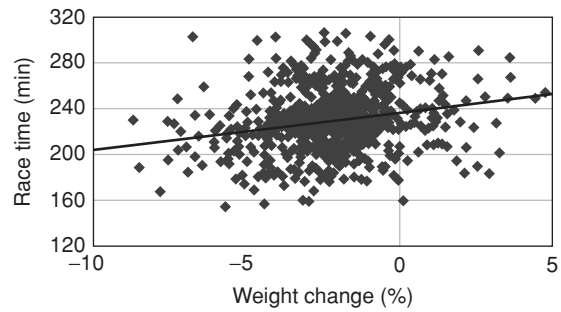

Figure 15.3 Degree of body weight change plotted against the race finishing time (minutes) for the entire group of 643 marathon finishers (p <0.0000001; r = 0.217). From Zouhal et al. (2011), with permission from BMJ Publishing Group Ltd.

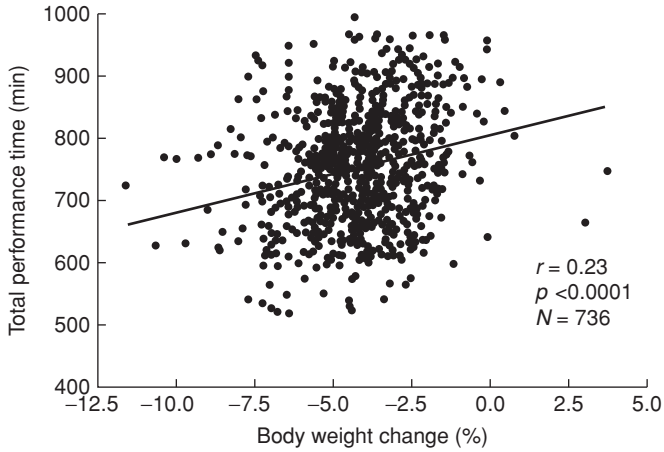

Figure 15.4 Relation between the total performance time (minutes) and percentage change in body weight. From Sharwood et al. (2004), with permission from BMJ Publishing Group Ltd.

with times below 2 hours and 13 minutes (Muir et al., 1970). In 1967, Pugh et al. reported that the winner (2 hours 38 minutes) of a national marathon held in England lost 5.23 kg, or in relative terms, 6.9% of his body weight. Moreover, it was reported that the first four athletes to finish the race had lost on average 5.8% of their weight.

Altogether, these findings suggest that athletes can achieve outstanding performance while being dehydrated by more than 2% body weight.

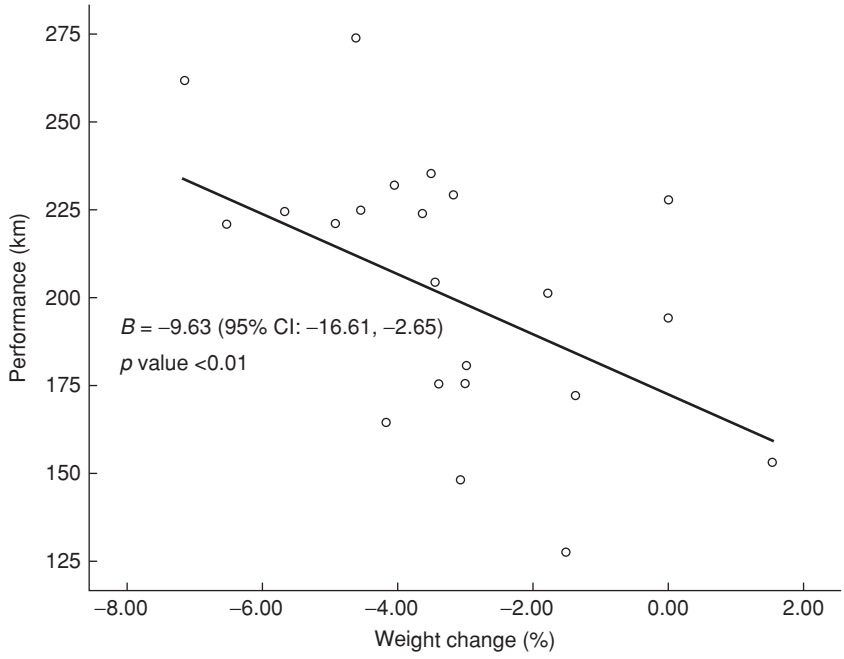

Figure 15.5 Relationship between the percentage change in relative body weight and performance (km) for athletes who successfully completed the 24-hour ultra-marathon ($n = 23$). B = the regression coefficient of the slope. From Kao et al. (2008).

Although these observations cannot be interpreted to suggest that dehydration helps performance, they nevertheless cast doubts about the validity of the 2% body weight loss rule.

Fixed-Exercise Intensity Conditions: Effects of Exercise-Induced Dehydration

Far more studies have examined the effect of exercise-induced dehydration using time-to-exhaustion type protocols or exercise protocols comprising bouts of fixed-exercise intensity rather than time-trial type protocols (Below et al., 1995; Ebert et al., 2007; Edwards et al., 2007; Fallowfield et al., 1996; Maughan et al., 1989, 1996; McConell et al., 1997, 1999; Van Schuylenbergh et al., 2005; Walsh et al., 1994). As underlined by Currel and Jeukendrup (2008), time-to-exhaustion protocols have likely evolved from early animal studies where it is impossible to ask the animal to complete a set distance as fast as possible. Additionally, in comparison with time-trials, fixed-exercise conditions offer the advantage of more easily dissecting the impact of an intervention on physiological functions. However, as argued previously, these tests are not appropriately designed to evaluate endurance performance. Nevertheless, they may have some relevance for military and occupational settings where fixed work rates may be imposed on individuals.

Overall, results of 10 research manuscripts comprising 15 individual studies can be used to determine how exercise-induced dehydration influences performance during exercise at constant power output. When each study is analyzed individually, two out of three (66%) show a statistically significant negative effect of exercise-induced dehydration. This represents a completely different portrait than that reflecting the effect of dehydration during time-trial type exercises. Reasons why athletes respond differently to the different protocols need to be investigated in future research.

It is apparent that the perception and belief of scientists, and by extension that of the general public, about the effects of exercise-induced dehydration on aerobic exercise capacity is in part based on findings deriving from studies using exercise protocols with fixed exercise intensity conditions. However, another part of the belief rests on studies that examined the influence of starting an exercise already hypohydrated on subsequent aerobic exercise performance.

Aerobic Exercise Performance: Effect of Pre-Exercise Hypohydration

Several studies have observed that individuals start exercising while being in a state of hypohydration (Maughan et al., 2007b; Stover et al., 2006). It is therefore important to examine the impact of such a condition on aerobic exercise performance. Sufficient studies have been completed to quite clearly delineate how an acute body water loss prior to exercise influences VO_2 max and high-intensity aerobic exercise performance (Sawka & Noakes, 2007). However, the quantity and quality of studies examining the influence of pre-exercise hypohydration on subsequent prolonged exercise are not sufficient to allow clear conclusions to be drawn.

Maximal Oxygen Consumption and Aerobic Exercise Performance: Effect of Pre-Exercise Hypohydration

In 2010, Gigou et al. reported the results of a meta-analysis evaluating the effect of pre-exercise hypohydration on VO_2 max and high-intensity, short-term aerobic exercise performance (i.e., ≤30 minutes). They demonstrated that both VO_2 max (–2.4%) and performance (–3.2%) were impaired in a statistically significant manner by pre-exercise hypohydration ≥3% body weight. Although there was no association between the extent of hypohydration and changes in aerobic exercise performances, there was a significant association (random-effects model meta-regression) between hypohydration and VO_2 max, with each percent of body weight loss above a threshold loss of 3% decreasing VO_2 max by 2.9% (Figure 15.6).

In an effort to provide results with the best real-world applicability possible, Gigou et al. (2010) excluded from their analysis all those studies that induced weight losses using either diuretics or energy-restricted diets. Moreover, to eliminate the possible confounding effects of residual body-heat

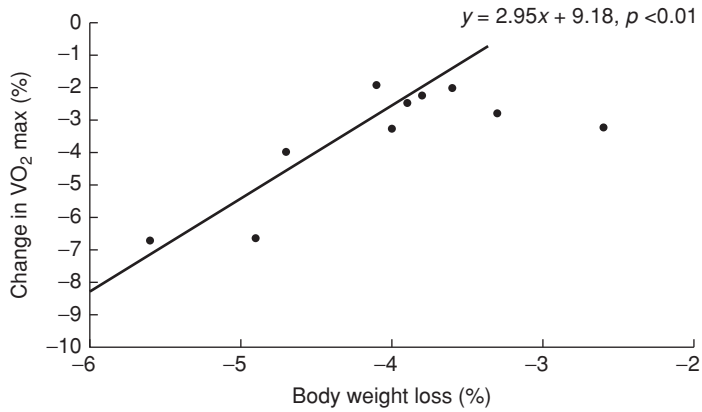

Figure 15.6 Correlation between the % changes in weight and the % changes in maximal oxygen consumption. From Gigou et al. (2010).

accumulation or muscular and systemic fatigue resulting either from heat exposure, physical exercise, or a combination of both factors to provoke acute weight losses, only studies with an elapse time ≥1 hour between the end of the dehydration and testing period were included.

In the only study of its kind, Casa et al. (2010) demonstrated that pre-exercise hypohydration of 2.3% significantly decreased time-trial running performance of ~1 hour, compared with a well-hydrated state. However, inferences from this study are difficult to make, since athletes who started the exercise well hydrated were permitted to drink during the time trial, whereas hypohydrated athletes were not. Moreover, the hypohydrated athletes were substantially thirstier than the well-hydrated athletes before, during, and after exercise. Given the results of Goulet's meta-analysis (2011), it therefore cannot be ruled out that the highest thirst sensation rating in the hypohydrated group played a significant role in the decrease in performance.

Results of the above studies suggest that pre-exercise hypohydration ≥3% body weight decreases subsequent high-intensity, ≤30 minute aerobic exercise performances as well as VO_2 max. It is however not clear how these variables would be affected by milder hypohydration levels (1–2%). Such results are very important and may have important performance implications, since during short-duration, high-intensity exercises no amount of fluid drunk during exercise will be able to reverse hypohydration.

Pre-exercise hypohydration as low as 2% body weight is likely to impair 1 hour, running time-trial performance under situations preventing fluid replacement during exercise. The effect pre-exercise hypohydration would have on prolonged exercise performance under situations where athletes are able to drink remains to be determined. In a brilliantly-designed study, Maresh et al. (2004) demonstrated that athletes who start an exercise significantly hypohydrated drink more and more frequently during exercise compared with athletes who undergo the exercise in a euhydrated state, such that they are able to reestablish their plasma osmolality to normal, baseline levels. Therefore, if allowed to drink during exercise, it is possible that athletes could maintain their performance although exercise was started in a hypohydrated state.

Jumping Ability and Muscle Strength, Power and Anaerobic Endurance Capacity: Effect of Pre-Exercise Hypohydration

Despite the existence of an important and relevant volume of individual research into the effects of hypohydration on muscle performance, relatively

little information can be found in the lay press or influential research papers about this very topic. This is puzzling for many reasons: (1) For strength-related sports, this can potentially have a direct and important implication on performance. (2) For most, competitive endurance-trained athletes, strength training is an integral part of a well-rounded yearly training plan and is critical to performance success. (3) It is well known that strength-trained athletes are continuously seeking ways to maximize training gains.

In 2007, Judelson et al. published the first systematic review paper looking at the influence of hypohydration on muscular strength, power, and high-intensity endurance. After accounting for several subtle methodological issues that either exacerbate or attenuate the effect of hypohydration, they demonstrated that hypohydration consistently diminishes strength (–2%), power (–3%), and high-intensity endurance (–10%) performance. Unfortunately, in their computation of the intervention effects, the authors included results of studies that induced hypohydration with diuretics, which produce a hypovolemic, isosmotic-type of dehydration. Under real-world conditions, sweat- or water deprivation-induced body water losses increase plasma osmolality level, which better protects and defends plasma volume, compared with the use of diuretics. Moreover, although the work of Judelson et al. (2007) provides great insight into how pre-exercise hypohydration influences various muscle-related performance outcomes, it is worth noting that a meta-analytic procedure was not utilized to determine hypohydration effects. From a scientific and statistical point of view, this represents an important weakness and limitation of this study.

Nevertheless, in a meta-analysis that excluded studies that induced hypohydration with the use of diuretics or energy restriction diets, Goulet et al. (2010) arrived at similar conclusions to those reached by Judelson et al. (2007). Additionally, they observed that leg and arm muscle strength were similarly and significantly reduced by pre-exercise hypohydration. Finally, it was demonstrated by Goulet et al. (2010) that pre-exercise hypohydration did not significantly alter vertical jumping capacity, a reflection of muscle power. This finding was observed despite the fact that hypohydration has been recently shown to lower the product of applied force and time during the propulsion phase of jumping, suggesting that the negative impact of hypohydration on leaping capacity is offset by an increase in the strength-to-mass ratio (Cheuvront et al., 2010).

Taken altogether, the results of the analyses of Judelson et al. (2007) and Goulet et al. (2010) strongly suggest that pre-exercise hypohydration decreases muscle strength, endurance, and power in a significant fashion. This, in addition of being directly relevant to endurance- and strength-trained athletes, may also be relevant for military and industrial personnel.

Hydration Guidelines

Based on the best and most recent available evidence, the following fluid replacement strategies were developed for strength- as well as endurance-trained athletes wishing to get the most out of training or competitive performance. Since our understanding of the effects of hypohydration on exercise performance is rather incomplete, these recommendations are not absolute but only tentative.

Before Exercise

- All athletes should begin exercise in a well-hydrated state. This can be achieved by paying close attention to thirst sensation (Casa et al., 2010), which should be kept as low as possible over the last 3 hours prior to exercise start. Given that it does not interfere with pre-competition preparations, in the last 2 hours before exercise, athletes should drink enough water (usually about 5–10 ml of water per kg body weight) to produce at least one micturition, which is very pale yellow to pale yellow in color (Armstrong et al., 1998). Such a level of hydration before exercise will ensure maintenance of normal plasma osmolality level, hence optimizing thirst regulation at the start of exercise.
- Under exercise situations where fluid access is limited, impossible, or not practical, and where

meaningful weight loss and thirst sensation could develop, athletes are encouraged to hyperhydrate before exercise. In fact, pre-exercise hyperhydration delays the onset of hypohydration and thirst development (Goulet et al., 2008). Different strategies may be used by athletes. In its simplest form, fluid overloading may be achieved by ingesting 500–1000 ml of cool water within 20 minutes of exercise start. The famous American runner and Boston marathon winner Alberto Salazar reportedly consumed 1000 ml of water 5 minutes prior to the 1984 Olympic marathon start (Armstrong et al., 1986). Alternately, commencing 60–80 minutes before exercise, athletes could hyperhydrate with 500–1000 ml of a sodium-containing solution (7 g/l of table salt). Sodium-induced hyperhydration has been demonstrated to improve endurance capacity (Sims et al., 2007) and time-trial capacity following a fixed-intensity bout of exercise (Coles & Luetkemeier, 2005). Importantly, before using these strategies during key races, athletes are encouraged to use and fully test them during training and low key races.

During Exercise

- Strength-trained athletes are encouraged to maintain euhydration by drinking quantities of fluid approximating sweat losses. Hourly sweating rate can be calculated with the following formula: nude pre-exercise body weight (kg) – nude post-exercise body weight (kg) + fluid intake during exercise (ml) – urine volume produced during exercise (ml)/exercise time (min) × 60.
- During aerobic exercise >1 hour, where plenty of fluid is available and work rate can be altered as wished, athletes are encouraged to drink according to the dictates of their thirst to gauge fluid replacement needs.
- Drinking according to thirst may not allow sufficient fluid replacement for elderly athletes (Ainslie et al., 2002) or for athletes exercising under cold conditions or who are undergoing a heat-acclimatization process. Therefore, they are encouraged to program their fluid intake during aerobic exercise such to prevent a body weight loss >2–3%. The same rule would apply (1) for endurance athletes perceiving that their thirst sensation is unreliable to optimize fluid intake; (2) in exercise conditions where work rate is fixed and continuous; and (3) when psychological stress or repeated food intake may blunt thirst sensation.
- Particularly under hot training or racing conditions, and whenever conditions allow, athletes are encouraged to consume the coolest fluid they can tolerate. Drinking cold fluid has been shown to reduce core temperature, which may benefit endurance performance (Burdon et al., 2010).
- During high-intensity, 1 hour exercise duration, athletes are encouraged to drink minimally. Specifically, every 8–10 minutes, athletes could rinse their mouths (~5–10 seconds) with, and subsequently swallow or spit, a small volume (20–25 ml) of sports drink solution. Rollo and Williams recently (2011) concluded that the available evidence suggests that mouth rinsing with carbohydrate solutions during 1 hour exercise enhances endurance performance.

Conclusion

This chapter has highlighted that the relationship between hypohydration and exercise performance is far more complicated than was previously thought. Nevertheless, inadequate hydration appears to systematically impair muscular performance, though dehydration induced during exercise does not appear to hinder time-trial exercise performance. Moreover, current research indicates that endurance performance may be maximized when athletes follow their thirst sensation to gauge the need for fluid replacement. Further well-conducted and ecologically valid studies need to be undertaken before solid, evidence-based recommendations can confidently be made to athletes regarding how they should be hydrating before and during exercise to maximize performance. However, readers should be reassured that the present chapter attempted to present and integrate the forefront of research in the field of hydration and performance.

References

Abbiss, C.R., Straker, L., Quod, M.J., et al. (2010) Examining pacing profiles in elite female road cyclists using exposure variation analysis. *British Journal of Sports Medicine* 44, 437–442.

Ainslie, P.N., Campbell, I.T., Frayn, K.N., et al. (2002) Energy balance, metabolism, hydration, and performance during strenuous hill walking: the effect of age. *Journal of Applied Physiology* 93, 714–723.

Amann, M., Hopkins, W.G., & Marcora, S.M. (2008) Similar sensitivity of time to exhaustion and time-trial time to changes in endurance. *Medicine & Science in Sports & Exercise* 40(3), 574–578.

Angus, S.D. & Waterhouse, B.J. (2011) Pacing strategy from high frequency field data: more evidence for neural regulation? *Medicine & Science in Sports & Exercise* 43, 2405–2411.

Armstrong, L.E., Hubbard, R.W., Jones, B.H., et al. (1986) Preparing Alberto Salazar for the heat of the 1984 Olympic marathon. *The Physician and Sports Medicine* 14, 73–81.

Armstrong, L.E., Maresh, C.M., Gabaree, C.V., et al. (1997) Thermal and circulatory responses during exercise: effects of hypohydration, dehydration, and water intake. *Journal of Applied Physiology* 82, 2028–2035.

Armstrong, L.E., Soto, J.A., Hacker Jr, F.T., et al. (1998) Urinary indices during dehydration, exercise, and rehydration. *International Journal of Sport Nutrition* 8, 345–355.

Bachle, L., Eckerson, J., Albertson, L., et al. (2001) The effect of fluid replacement on endurance performance. *Journal of Strength and Conditioning Research* 15, 217–224.

Backx, K., van Someren, K.A., & Palmer, G.S. (2003) One hour cycling performance is not affected by ingested fluid volume. *International Journal of Sport Nutrition and Exercise Metabolism* 13, 333–342.

Barr, S.I. (1999) Effects of dehydration on exercise performance. *Canadian Journal of Applied Physiology* 24, 164–172.

Below, P.R., Mora-Rodríguez, R., González-Alonso, J., et al. (1995) Fluid and carbohydrate ingestion independently improve performance during 1 h of intense exercise. *Medicine & Science in Sports & Exercise* 27, 200–210.

Brown, M.B., McCarty, N.A., & Millard-Stafford, M. (2011) High-sweat Na+ in cystic fibrosis and healthy individuals does not diminish thirst during exercise in the heat. *American Journal of Physiology—Regulatory, Integrative, and Comparative Physiology* 301, R1177–R1185.

Burdon, C.A., O'Connor, H.T., Gifford, J.A., et al. (2010) Influence of beverage temperature on exercise performance in the heat: a systematic review. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 166–174.

Casa, D.J. (1999) Exercise in the heat. I. Fundamentals of thermal physiology, performance implications, and dehydration. *Journal of Athletic Training* 34, 246–252.

Casa, D.J., Armstrong, L.E., Hillman, S.K., et al. (2000) National athletic trainers' association position statement: fluid replacement for athletes. *Journal of Athletic Training* 35, 212–224.

Casa, D.J., Stearns, R.L., Lopez, R.M., et al. (2010) Influence of hydration on physiological function and performance during trail running in the heat. *Journal of Athletic Training* 45, 147–156.

Cheuvront, S.N., Carter 3rd, R., & Sawka, M.N. (2003) Fluid balance and endurance exercise performance. *Current Sports Medicine Reports* 2, 202–208.

Cheuvront, S.N., Kenefick, R.W., Ely, B.R., et al. (2010) Hypohydration reduces vertical ground reaction impulse but not jump height. *European Journal of Applied Physiology* 109, 1163–1170.

Coles, M.G. & Luetkemeier, M.J. (2005) Sodium-facilitated hypervolemia, endurance performance, and thermoregulation. *International Journal of Sports Medicine* 26, 182–187.

Convertino, V.A., Armstrong, L.E., Coyle, E.F., et al. (1996) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 28, 1–7.

Currell, K. & Jeukendrup, A.E. (2008) Validity, reliability and sensitivity of measures of sporting performance. *Sports Medicine* 38, 297–316.

Dugas, J.P., Oosthuizen, U., Tucker, R., et al. (2009) Rates of fluid ingestion alter pacing but not thermoregulatory responses during prolonged exercise in hot and humid conditions with appropriate convective cooling. *European Journal of Applied Physiology* 105, 69–80.

Ebert, T.R., Martin, D.T., Bullock, N., et al. (2007) Influence of hydration status on thermoregulation and cycling hill climbing. *Medicine & Science in Sports & Exercise* 39, 323–329.

Edwards, A.M., Mann, M.E., Marfell-Jones, M.J., et al. (2007) Influence of moderate dehydration on soccer performance: physiological responses to 45 min of outdoor match-play and the immediate subsequent performance of sport-specific and mental concentration tests. *British Journal of Sports Medicine* 41, 385–391.

Fallowfield, J.L., Williams, C., Booth, J., et al. (1996) Effect of water ingestion on endurance capacity during prolonged running. *Journal of Sports Sciences* 14, 497–502.

Gigou, P.Y., Lamontagne-Lacasse, M., & Goulet, E.D.B. (2010) Meta-analysis of the effects of pre-exercise hypohydration on endurance performance, lactate threshold and VO_{2max}. *Medicine & Science in Sports & Exercise* 42, 361–362.

Goulet, E.D. (2011) Effect of exercise-induced dehydration on time-trial exercise performance: a meta-analysis. *British Journal of Sports Medicine* 45, 1149–1156.

Goulet, E.D., Rousseau, S.F., Lamboley, C.R., et al. (2008) Pre-exercise hyperhydration delays dehydration and improves endurance capacity during 2 h of cycling in a temperate climate. *Journal of Physiological Anthropology* 27, 263–271.

Goulet, E.D.B., Lamontagne-Lacasse, M., Gigou, P.Y., et al. (2010) Pre-exercise hypohydration effects on skeletal muscle strength, endurance, anaerobic capacity and jumping performance?: a meta-analysis. *Medicine & Science in Sports & Exercise* 42, 362.

Hew-Butler, T., Verbalis, J.G., & Noakes, T.D. (2006) International Marathon Medical Directors Association. Updated fluid recommendation: position statement from the International Marathon Medical Directors Association (IMMDA). *Clinical Journal of Sport Medicine* 16, 283–292.

Horswill, C.A. (1998) Effective fluid replacement. *International Journal of Sport Nutrition* 8, 175–195.

International Association of Athletics Federations (2011) *Nutrition for Athletics*, http://www.iaaf.org/mm/Document/imported/42817.pdf (accessed July 4, 2011).

International Olympic Committee (2010) *Consensus Statement on Sports Nutrition*, http://www.olympic.org/Documents/Reports/EN/CONSENSUS-FINAL-v8-en.pdf (accessed November 26, 2011).

Judelson, D.A., Maresh, C.M., Anderson, J.M., et al. (2007) Hydration and

muscular performance: does fluid balance affect strength, power and high-intensity endurance? *Sports Medicine* 37, 907–921.

Kao, W.F., Shyu, C.L., Yang, X.W., et al. (2008) Athletic performance and serial weight changes during 12-and 24-hour ultra-marathons. *Clinical Journal of Sport Medicine* 18, 155–158.

Kay, D. & Marino, E.F. (2003) Failure of fluid ingestion to improve self-paced exercise performance in moderate-to-warm humid environments. *Journal of Thermal Biology* 28, 29–34.

Lambert, M.I., Dugas, J.P., Kirkman, M.C., et al. (2004) Changes in running speeds in a 100 km ultra-marathon race. *Journal of Sports Science and Medicine* 3, 167–173.

Laursen, P.B., Rhodes, E.C., Langill, R.H., et al. (2002) Relationship of exercise test variables to cycling performance in an Ironman triathlon. *European Journal of Applied Physiology* 87, 433–440.

Laursen, P.B., Shing, C.M., Tennant, S.C., et al. (2003) A comparison of the cycling performance of cyclists and triathletes. *Journal of Sports Sciences* 21, 411–418.

Maresh, C.M., Gabaree-Boulant, C.L., Armstrong, L.E., et al. (2004) Effect of hydration status on thirst, drinking, and related hormonal responses during low-intensity exercise in the heat. *Journal of Applied Physiology* 97, 39–44.

Maughan, R.J., Fenn, C.E., & Leiper, J.B. (1989) Effects of fluid, electrolyte and substrate ingestion on endurance capacity. *European Journal of Applied Physiology and Occupational Physiology* 58, 481–486.

Maughan, R.J., Leiper, J.B., & McGaw, B.A. (1990) Effects of exercise intensity on absorption of ingested fluids in man. *Experimental Physiology* 75, 419–421.

Maughan, R.J., Bethell, L.R., & Leiper, J.B. (1996) Effects of ingested fluids on exercise capacity and on cardiovascular and metabolic responses to prolonged exercise in man. *Experimental Physiology* 81, 847–859.

Maughan, R.J., Shirreffs, S.M., & Leiper, J.B. (2007a) Errors in the estimation of sweat loss and changes in hydration status from changes in body mass during exercise. *Journal of Sports Sciences* 25, 797–804.

Maughan, R.J., Watson, P., Evans, G.H., et al. (2007b) Water balance and salt losses in competitive football. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 583–594.

McConell, G.K., Burge, C.M., Skinner, S.L., et al. (1997) Influence of ingested fluid volume on physiological responses during prolonged exercise. *Acta Physiologica Scandinavica* 160, 149–156.

McConell, G.K., Stephens, T.J., & Canny, B.J. (1999) Fluid ingestion does not influence intense 1-h exercise performance in a mild environment. *Medicine & Science in Sports & Exercise* 31, 386–392.

Merry, T.L., Ainslie, P.N., Walker, R., et al. (2008) Fitness alters fluid regulatory but not behavioural responses to hypohydrated exercise. *Physiology and Behavior* 20, 348–352.

Muir, A.L., Percy-Robb, I.W., Davidson, I.A., et al. (1970) Physiological aspects of the Edinburgh commonwealth games. *Lancet* 296(7683), 1125–1128.

Mündel, T. (2011) To drink or not to drink? Explaining "contradictory findings" in fluid replacement and exercise performance: evidence from a more valid model for real-life competition. *British Journal of Sports Medicine* 45, 2.

Noakes, T.D. (1993) Fluid replacement during exercise. *Exercise and Sport Science Review* 21, 297–330.

Palmer, G.S., Dennis, S.C., Noakes, T.D., et al. (1996) Assessment of the reproducibility of performance testing on an air-braked cycle ergometer. *International Journal of Sports Medicine* 17, 293–298.

Paton, C. & Hopkins, W. (2006) Variation in performance of elite cyclists from race to race. *European Journal of Sport Science* 6, 25–31.

Pugh, L.G., Corbett, J.L., & Johnson, R.H. (1967) Rectal temperatures, weight losses, and sweat rates in marathon running. *Journal of Applied Physiology* 23, 347–352.

Robinson, T.A., Hawley, J.A., Palmer, G.S., et al. (1995) Water ingestion does not improve 1-h cycling performance in moderate ambient temperatures. *European Journal of Applied Physiology and Occupational Physiology* 71, 153–160.

Rodriguez, N.R., Di Marco, N.M., & Langley, S. (2009) Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41, 709–731.

Rollo, I. & Williams, C. (2011) Effect of mouth-rinsing carbohydrate solutions on endurance performance. *Sports Medicine* 41, 449–461.

Sawka, M.N. & Noakes, T.D. (2007) Does dehydration impair exercise performance? *Medicine & Science in Sports & Exercise* 39, 1209–1217.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.

Sharwood, K.A., Collins, M., Goedecke, J.H., et al. (2004) Weight changes, medical complications, and performance during an Ironman triathlon. *British Journal of Sports Medicine* 38, 718–724.

Sims, S.T., van Vliet, L., Cotter, J.D., et al. (2007) Sodium loading aids fluid balance and reduces physiological strain of trained men exercising in the heat. *Medicine & Science in Sports & Exercise* 39(1), 123–130.

Stover, E.A., Petrie, H.J., Passe, D., et al. (2006) Urine specific gravity in exercisers prior to physical training. *Applied Physiology, Nutrition, and Metabolism* 31, 320–327.

Thomas, K., Stone, M.R., Thompson, K.G., et al. (2012) Reproducibility of pacing strategy during simulated 20-km cycling time trials in well-trained cyclists. *European Journal of Applied Physiology* 112, 223–229.

Valtin H. (2002) "Drink at least eight glasses of water a day." Really? Is there scientific evidence for "8 × 8"? *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology* 283, R993–R1004.

Van Schuylenbergh, R., Vanden Eynde, B., & Hespel, P. (2005) Effect of exercise-induced dehydration on lactate parameters during incremental exercise. *International Journal of Sports Medicine* 26, 854–858.

Walsh, R.M., Noakes, T.D., Hawley, J.A., et al. (1994) Impaired high-intensity cycling performance time at low levels of dehydration. *International Journal of Sports Medicine* 15, 392–398.

Zouhal, H., Groussard, C., Vincent, S., et al. (2009) Athletic performance and weight changes during the "Marathon of Sands" in athletes well-trained in endurance. *International Journal of Sports Medicine* 30, 516–521.

Zouhal, H., Groussard, C., Minter, G., et al. (2011) Inverse relationship between percentage body weight change and finishing time in 643 forty-two-kilometre marathon runners. *British Journal of Sports Medicine* 45, 1101–1105.

Chapter 16

Rehydration and Recovery After Exercise

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Introduction

Chapter 14 of this book describes the impact of exercise on water balance. Given the substantial sweat losses that some people experience during exercise, becoming dehydrated is sometimes inevitable, even where there is free access to water and other fluids during the exercise period. Hard exercise is often combined with limited opportunities for fluid intake, and in this situation, large water deficits may be incurred. In some cases there will be no urgency to restore hydration status to euhydration, and thus it can be achieved by consumption of food and drinks taken as part of the normal diet. In other situations, however, a planned rehydration strategy can lead to a faster and more efficient restoration of body water content.

It is also important to recognize that restoration of water balance is only one of the nutritional priorities after exercise. Other concerns of the athlete and the recreational exerciser alike include the replenishment of muscle and liver glycogen stores if the exercise has caused these to be depleted (Burke et al., 2011) and the provision of a sufficient amount of the essential amino acids to stimulate the process of adaptation that takes place in muscle and other tissues in the hours after exercise (Phillips & van Loon, 2011). Restoration of water balance after extensive sweat losses also requires replacement of the electrolytes—especially sodium—lost in the sweat.

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The fluid deficit that exists at the end of an exercise session will depend on the water losses incurred, mostly in the form of sweat, and on the amount of fluid ingested. Where the net fluid deficit is small (perhaps about 1–2% of body mass or less), rehydration will not generally be a priority during the immediate post-exercise recovery period. The recovery strategy will also be influenced, however, by the time available before the next exercise session and by whether this is likely to be affected if there is incomplete recovery of fluid balance.

There is good evidence that anabolic and catabolic processes within the muscle cell are influenced by cell volume, with an expanded cell volume favoring anabolism (Lang, 2011). Although it seems intuitively obvious that fluid ingestion will favor cell expansion and thus contribute to the achievement of recovery goals, evidence as to how this can be most effectively achieved is almost entirely absent at the present time.

Factors Influencing Rehydration after Exercise

The primary factors influencing the rehydration process after exercise are the volume and composition of the fluid consumed. The volume consumed will be influenced by many factors, including the palatability of the drink and its effects on the thirst mechanism, although some people may choose to drink large quantities of an unpalatable drink even when they are not thirsty or to avoid drinking even when a palatable drink is available and they are

thirsty. These examples illustrate that conscious decisions can override the usual physiological signals. The availability of fluid may also be an important factor: if drinks are not available, they will not be consumed. Also, if there is not easy access to drinks, some individuals will not make the effort to obtain them. The ingestion of solid food and the composition of that food may also be important factors influencing rehydration after exercise, but there are many situations where solid food is avoided between exercise sessions or immediately after exercise because of real or perceived gastrointestinal discomfort. Where no solid food is consumed, all of the recovery aims must be met from drinks: this is entirely possible, but requires careful consideration of both the nutrition priorities and the composition of drinks to be consumed.

Drink Volume and Rate of Drink Ingestion

Restoration of fluid balance after exercise requires fluid ingestion, but will also depend on ongoing body water losses. Some sweat loss may continue after exercise and there will certainly be ongoing losses of water from the lungs and through the skin. In addition, obligatory urine losses persist even in the dehydrated state, because of the need for elimination of metabolic waste products. Thus, the volume of fluid consumed after exercise-induced or thermal sweating must be greater than the fluid deficit that exists at the end of the exercise period if effective rehydration is to be achieved. This has been demonstrated in a number of research studies including those of Mitchell et al. (1994) and Shirreffs et al. (1996) where volumes equivalent to 150% of the sweat volumes lost were shown to be effective at promoting rehydration after exercise. When a volume equal to the net fluid deficit at the end of exercise, or only slightly greater, is consumed, the ongoing water losses mean that a state of euhydration is not maintained for more than a very short period of time (Shirreffs & Maughan, 1998). It was also demonstrated that there was an interaction between the drink volume consumed and its sodium content, and the role of sodium in rehydration after exercise is described more fully in the section Beverage Composition.

Subsequent to this research, it has become clear that the rate of ingestion of a large rehydration bolus can have important implications for the subsequent fate of the ingested fluid (Archer & Shirreffs, 2001; Kovacs et al., 2002). Rapid ingestion of a large volume of water or other energy-free and sodium-free drinks will lead to a prompt and marked increase of plasma volume and a corresponding decrease in the plasma sodium concentration: this in turn will lead to a pronounced diuretic response, causing a significant part of the ingested water to be lost in the urine, even when a state of net negative fluid balance is present. As described by Shafiee et al. (2005), reducing the overall rate of fluid absorption by reducing the rate at which water is ingested or by taking other steps to slow the appearance of this water in the vascular space can aid the retention of the ingested fluid by avoiding the hemodilution that occurs with rapid ingestion. These effects may be important in situations where only a limited amount of fluid is available, as in wilderness treks or military operations where all water supplies must be carried. An unnecessary diuresis is wasteful of water.

Outside the laboratory, it is normal for individuals to have a free choice as to how much they wish to drink and how quickly or slowly they do so. This is particularly true for recovery after exercise when any rules or restrictions imposed by the sport will likely have ceased. Their individual physiology may, however, influence their drinking behavior, as a large volume drink which is emptied slowly from the stomach is likely to be consumed at a slower rate than one which empties rapidly as a result of the discomfort experienced due to an excessively full stomach. A number of drink characteristics will result in a slower emptying from the stomach, including a high energy density resulting from a high carbohydrate content. This issue is revisited in the section on the roles of carbohydrate and protein later in this chapter.

Beverage Composition

Sodium Plain water is not the ideal post-exercise rehydration beverage when rapid and complete restoration of fluid balance is necessary and where all intake is in liquid form. The reasons for this were

described some time ago (e.g., Costill & Sparks, 1973; Nielsen et al., 1986) when it was observed that the high urine flow that followed ingestion of large volumes of electrolyte-free drinks did not allow subjects to remain in positive fluid balance for more than a very short time. These studies also established that the plasma volume was better maintained when electrolytes were present in the fluid ingested, and this effect was attributed to the presence of sodium in the drinks (see Chapter 44).

The first systematic studies to investigate the mechanisms of post-exercise rehydration showed that the ingestion of large volumes of plain water after exercise-induced dehydration resulted in a rapid fall in plasma osmolality and sodium concentration (Nose et al., 1988a), leading to a prompt and marked diuresis caused by a rapid return to control levels of plasma renin activity and aldosterone concentration (Nose et al., 1988b). The fall in plasma osmolality and sodium concentration that occurs in response to ingestion of low-electrolyte drinks results in a stimulation of urine output. If, for whatever reason, diuresis does not occur, there may be potentially more serious consequences such as hyponatremia.

As sodium is the major ion lost in sweat (Table 16.1), it is intuitive that sweat sodium losses should be replaced. It is not logical to think that the salty water we lose as sweat would be best replaced in the body by plain water. This area has been systematically investigated, and Shirreffs and Maughan (1998) showed that, provided that an adequate volume is consumed, sustained euhydration is achieved only when the sodium intake is greater than the sweat sodium loss. After ingestion of a large bolus of fluid with varying sodium content (0–100 mmol/l), an approximately linear relationship was observed between hydration status a few hours later and the sodium content of the ingested drinks. The addition of sodium to a rehydration beverage is therefore justified on the basis that sodium is lost in sweat and must be replaced to achieve full restoration of fluid balance. The sodium concentration of most commercially available sports drinks, however, is less than the typical sodium concentration of sweat and although it will contribute to rehydration, it is insufficient for those individuals with a high sweat sodium concentration.

Table 16.1 Concentration (mmol/l) of the major electrolytes in sweat, plasma, and intracellular water

	Sweat	Plasma	Intracellular
Sodium	20–80	130–155	10
Potassium	4–8	3.2–5.5	150
Calcium	0–1	2.1–2.9	0
Magnesium	<0.2	0.7–1.5	15
Chloride	20–60	96–110	8
Bicarbonate	0–35	23–28	10
Phosphate	0.1–0.2	0.7–1.6	65
Sulfate	0.1–2	0.3–0.9	10

These values are taken from a variety of sources.

Potassium and Magnesium Potassium, as the major ion in the intracellular fluid, has been postulated to have a role in optimizing post-exercise rehydration by aiding the retention of water in the intracellular space. Potassium is lost in sweat in concentrations of about 5–10 mmol/l which is a little higher than the typical extracellular potassium concentration of about 4–6 mmol/l. Early work using dehydrated rats supported this idea (Yawata, 1990) indicating that the role of potassium in restoring intracellular volume is more modest than that of sodium in restoring extracellular volume. However, subsequent work in man, either directly investigating the role of potassium, or indirectly providing some data due to the inclusion of drinks with a high potassium content, has proved to be less conclusive (e.g., Maughan et al., 1994; Saat et al., 2002; Shirreffs et al., 2007a, 2007b). Potassium may therefore be important in enhancing rehydration by aiding intracellular rehydration, but further investigation is required to provide conclusive evidence. Importantly, however, no negative effect of including modest amounts of potassium in rehydration drinks has been demonstrated and indeed potassium in small quantities is an ingredient in most commercially available sports drinks that are promoted for post-exercise rehydration. As indicated above, the focus of hydration strategies has tended to be on the maintenance of vascular volume during exercise, but the restoration and even expansion of cellular volume after exercise may be equally important due

to the effects of cell volume on a wide range of anabolic and catabolic functions (Lang, 2011).

The importance of including magnesium in sports drinks has been the subject of much discussion. Magnesium is lost in sweat in only small amounts (<0.2 mmol/l) relative to the total body magnesium content, but these losses or any resulting effects on circulating levels (Schwellnus et al., 2004) have been implicated in muscle cramp though the evidence for a causal role is absent (Garrison et al., 2012). Even though there may be a decline in plasma magnesium concentration during exercise, this is most likely to be due to the redistribution of magnesium and water between body water compartments rather than to sweat loss. There does not seem to be any good reason for including magnesium in post-exercise rehydration and recovery sports drinks (Nielsen & Lukaski, 2006).

Carbohydrate The inclusion of carbohydrate in post-exercise recovery drinks is not essential for restoration of fluid balance, though replacement of glycogen stores is an important aspect of recovery if the exercise has resulted in depletion of glycogen stores. However, if it is included, carbohydrate may significantly affect the rehydration process. One role of carbohydrate added at low concentrations (about 2–6%) may be to promote intestinal absorption of the drink via sodium–glucose cotransport in the small intestine (Schedl et al., 1994). Where the rest interval between successive exercise sessions is short and rapid rehydration is essential, this role may be of major importance. However, a second, more recently researched role relates to the effects of higher concentrations of carbohydrate on the rate of gastric emptying and thus on the rate at which ingested drinks are available to the body after drinking (Evans et al., 2011).

In a study by Evans et al. (2009), restoration of water balance was assessed following sweat loss equivalent to about 2% of body mass induced by intermittent exercise in the heat. Rehydration was achieved more effectively by the ingestion of a hypertonic 10% glucose electrolyte solution than by the ingestion of the same volume of a hypotonic 2% glucose electrolyte solution or an electrolyte-only solution. The greater efficacy of the 10% glucose electrolyte solution was attributed to the avoidance of large reductions in serum osmolality and increases in plasma volume that resulted from the rapid absorption of the other drinks. Similarly, another recent study by Osterberg et al. (2010) demonstrated that the addition of carbohydrate to a rehydration solution resulted in an enhanced retention of fluid over a 4-hour recovery period compared to a carbohydrate-free placebo. It should be noted, however, that the participants in this latter study ingested a volume of fluid equivalent to 100% of the body mass lost during exercise over a 60-minute period, and thus remained hypohydrated throughout the recovery period on all trials.

Recovery of muscle glycogen stores after exercise will also involve an increase in the water content of muscle. There is some debate as to how much water is stored in association with muscle glycogen, but a value of about 3 g of water per gram of glycogen is commonly accepted (Maughan et al., 2007). Given that a long, hard training session typical of that completed by cyclists, rowers, or runners might involve the use of up to 400 g of endogenous carbohydrate, it is clear that the ingestion of large amounts of carbohydrate in the immediate post-exercise period should be accompanied by an intake of water that is sufficient to allow for the replacement of water associated with glycogen storage.

Protein In recent years, a number of studies investigating recovery of water losses after exercise have included drinks that contain protein (Seifert et al., 2006; Shirreffs et al., 2007a; Watson et al., 2008) and some of these initial studies suggested that these protein-containing drinks may promote rehydration after exercise. However, these studies were investigating "off-the-shelf" drinks, and the study designs did not allow the impact of protein to be specifically determined. In the case of studies that used milk-based drinks, any beneficial effect of the protein could have been due to a slower rate of gastric emptying due to the milk proteins, fat, lactose, and other components (Burn-Murdoch et al., 1978). As described above, slowing the rate of gastric emptying will limit excursions in plasma volume and electrolyte concentrations and thus reduce the diuretic response.

More recently, James et al. (2011) demonstrated that post-exercise fluid retention was greater after ingestion of a carbohydrate plus milk protein solution than after consuming a carbohydrate solution that was matched for energy density and electrolyte content. The mechanism for this effect was again proposed to relate to the slowing of gastric emptying, in this case caused by the coagulation of the casein fraction of milk protein in the acid environment of the stomach. Subsequent research investigating the effects of a whey protein isolate on rehydration after exercise-induced sweat loss did not replicate the findings achieved with the milk proteins, so the picture remains unclear (James et al., 2012).

Food and Fluid Consumption

In many situations, there may be opportunities to consume solid food between exercise bouts, and this should be encouraged to meet other nutritional goals unless it is likely to result in gastrointestinal disturbances. Maughan et al. (1996) investigated the role of solid food intake in promoting rehydration from a sweat loss equivalent to 2.1% of body mass with consumption of either a solid meal plus flavored water or a commercially available sports drink: the two treatments were matched for total water content. The urine volume produced following food and water ingestion was almost 300 ml less than that when the sports drink was consumed, resulting in a more favorable recovery and maintenance of hydration status. This was attributed to the higher electrolyte (sodium and potassium) content of the meal relative to the sports drink. Subsequent studies have also highlighted a role for food products in post-exercise fluid balance restoration and have reinforced the finding that plain water is an adequate post-exercise rehydration beverage if salt-containing foods are ingested at the same time (Ray et al., 1998).

The beneficial effects are likely to be due to the relatively large amounts of electrolytes in foods and/or the delayed gastric emptying that occurs with solids in comparison to liquids (Maughan et al., 1996).

Beverage Palatability and Voluntary Fluid Intake

The majority of scientific studies in the area of post-exercise rehydration and recovery, including those described above, have prescribed a fixed volume of fluid that was consumed within a fixed period of time according to a predetermined schedule. However, in most of the everyday situations that athletes find themselves in, intake is determined by the interaction of physiological and psychological factors. When the effect of palatability, together with the solute content of beverages in promoting rehydration after sweat loss, was studied (Wemple et al., 1997), subjects drank 123% of their sweat volume losses with flavored water and 163% and 133% when the solution had 25 and 50 mmol/l sodium. Because of the higher urine output with the flavored water, however, 3 hours after starting the rehydration process, the subjects were in a better whole body hydration status after drinking the sodium-containing beverages than the flavored water. Similar results had previously been reported by Maughan and Leiper (1993) and together these studies demonstrate the importance of palatability for promoting consumption. They also confirm earlier results showing that a moderately high electrolyte content is essential if the ingested fluid is to be retained in the body. The benefit of the higher intake with the more palatable drinks was lost because of the higher urine output. Other drink characteristics, including carbonation, influence drink palatability and therefore need to be considered when a beverage is being considered for effective post-exercise rehydration (Passe et al., 1997).

Post-exercise Rehydration and Restoration of Exercise Capacity or Performance

A relatively small number of studies have investigated the influence of post-exercise rehydration on acute restoration of exercise capacity or performance (e.g., Bilzon et al., 2000; Lee et al., 2011; Watson et al., 2008; Williams et al., 2003; Wong & Williams, 2000; Wong et al., 1998, 2000, 2009). The results of these studies are generally inconclusive and most provided carbohydrate as well as water: isolating the

effects of recovery of water balance from carbohydrate replacement effects is not always possible. This remains an area where significant systematic research is required. It does, though, seem sensible to make use of the immediate post-exercise period to begin the process of rehydration and replenishment of glycogen stores. Whether this is achieved through drinks alone or by a combination of drinks and solid will depend on the circumstances and on individual preferences.

Practical Considerations, Summary, and Conclusions

It is normal for athletes to finish training or competition with a substantial body water and sodium deficit because water and salt intake during exercise seldom match sweat losses during hard exercise sessions. When an active rehydration strategy for rapid but effective and sustained recovery of sweat losses after exercise is desired, careful consideration should be given to the drink composition, its palatability, the volume to be consumed, and the rate of consumption. An assessment of the water deficit can be made from the change in body mass that has occurred during exercise (Maughan et al., 2007). It is suggested that the volume of fluid consumed in the post-exercise period should be more than the net water deficit to account for obligatory urine losses and ongoing sweating. Replacement of sodium losses is also a prerequisite for restoration of hydration status, so ensuring a source of sodium to consume is needed, but palatability of the drink consumed is key, particularly, if a large volume needs to be consumed. However, drinking at a rapid rate may be less effective than slower drinking because a rapid expansion of blood volume and/or dilution of extracellular fluid may induce a diuresis, which will affect the volume retained.

References

Archer, D.T. & Shirreffs, S.M. (2001) Effect of fluid ingestion rate on post-exercise rehydration in man. *Proceedings of the Nutrition Society* 60, 200A.

Bilzon, J.L.J., Allsopp, A.J., & Williams, C. (2000) Short-term recovery from prolonged constant pace running in a warm environment: the effectiveness of a carbohydrate–electrolyte solution. *European Journal of Applied Physiology* 82(4), 305–312.

Burke, L.M., Hawley, J.A., Wong, H.S.S., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Sciences* 29, S17–S27.

Burn-Murdoch, R.A., Fisher, M.A., & Hunt, J.N. (1978) The slowing of gastric emptying by proteins in test meals. *Journal of Physiology* 274, 477–485.

Costill, D.L. & Sparks, K.E. (1973) Rapid fluid replacement following thermal dehydration. *Journal of Applied Physiology* 34, 299–303.

Evans, G.H., Shirreffs, S.M., & Maughan, R.J. (2009) Post-exercise rehydration in man: the effects of osmolality and carbohydrate content of ingested drinks. *Nutrition* 25, 905–913.

Evans, G.H., Shirreffs, S.M., & Maughan, R.J. (2011) The effects of repeated ingestion of high and low glucose–electrolyte solutions on gastric emptying and blood $2H_2O$ concentration after an overnight fast. *British Journal of Nutrition* 106, 1732–1739. doi:10.1017/S0007114511002169

Garrison, S.R., Allan, G.M., Sekhon, R.K., et al. (2012) Magnesium for skeletal muscle cramps. *Cochrane Database of Systematic Reviews*, 9 (Art. No. CD009402). doi:10.1002/14651858.CD009402.pub2

James, L.J., Clayton, D., & Evans, G.H. (2011) Effect of milk protein addition to a carbohydrate–electrolyte rehydration solution ingested after exercise in the heat. *British Journal of Nutrition* 105, 393–399. doi:10.1017/S0007114510003545

James, L.J., Gingell, R., & Evans, G.H. (2012) Whey protein addition to a carbohydrate–electrolyte rehydration solution ingested after exercise in the heat. *Journal of Athletic Training* 47(1), 61–66.

Kovacs, E.M., Schmahl, R.M., Denden, J.M., et al. (2002) Effect of high and low rates of fluid intake on post-exercise rehydration. *International Journal of Sport Nutrition and Exercise Metabolism* 12, 14–23.

Lang, F. (2011) Effect of cell hydration on metabolism. In *Sports Nutrition: More Than Just Calories—Triggers for Adaptation* (eds. R.J. Maughan & L.M. Burke), pp. 115–130. Karger, Basel, Switzerland.

Lee, J.K.W., Nio, A.Q.X., Ang, W.H., et al. (2011) Effects of ingesting a sports drink during exercise and recovery on subsequent endurance capacity. *European Journal of Sport Science* 11(2), 77–86.

Maughan, R.J. & Leiper, J.B. (1993) Post-exercise rehydration in man: effects of voluntary intake of four different beverages. *Medicine & Science in Sports & Exercise* 25(Suppl.), S2.

Maughan, R.J., Owen, J.H., Shirreffs, S.M., et al. (1994) Post-exercise rehydration in man: effects of electrolyte addition to ingested fluids. *European Journal of Applied Physiology* 69, 209–215.

Maughan, R.J., Leiper, J.B., & Shirreffs, S.M. (1996) Restoration of fluid balance after exercise-induced dehydration: effects of food and fluid intake. *European Journal of Applied Physiology* 73, 317–325.

Maughan, R.J., Shirreffs, S.M., & Leiper, J.B. (2007) Errors in the estimation of sweat loss and changes in hydration status from changes in body mass during exercise. *Journal of Sports Sciences* 25, 797–804.

Mitchell, J.B., Grandjean, P.W., Pizza, F.X., et al. (1994) The effect of volume ingested on rehydration and gastric emptying following exercise-induced dehydration. *Medicine & Science in Sports & Exercise* 26, 1135–1143.

Nielsen, F.H. & Lukaski, H.C. (2006) Update on the relationship between magnesium and exercise. *Magnesium Research* 19, 180–189.

Nielsen, B., Sjogaard, G., Ugelvig, J., et al. (1986) Fluid balance in exercise dehydration and rehydration with different glucose–electrolyte drinks. *European Journal of Applied Physiology* 55, 318–325.

Nose, H., Mack, G.W., Shi, X., et al. (1988a) Role of osmolality and plasma volume during rehydration in humans. *Journal of Applied Physiology* 65, 325–331.

Nose, H., Mack, G.W., Shi, X.R., et al. (1988b) Involvement of sodium retention hormones during rehydration in humans. *Journal of Applied Physiology* 65, 332–336.

Osterberg, K.L., Pallardy, S.E., Johnson, R.J., et al. (2010) Carbohydrate exerts a mild influence on fluid retention following exercise-induced dehydration. *Journal of Applied Physiology* 108, 245–250.

Passe, D.H., Horn, M., & Murray, R. (1997). The effects of beverage carbonation on sensory responses and voluntary fluid intake following exercise. *International Journal of Sports Nutrition* 7, 286–297.

Phillips, S.M. & van Loon, L.J.C. (2011) Dietary protein for athletes: from requirements to optimum adaptation. *Journal of Sports Sciences* 29, S29–S38.

Ray, M.L., Bryan, M.W., Ruden, T.M., et al. (1998) Effect of sodium in a rehydration beverage when consumed as a fluid or meal. *Journal of Applied Physiology* 85(4), 1329–1336.

Saat, M., Singh, R., Sirisinghe, R.G., et al. (2002) Rehydration after exercise with fresh young coconut water, carbohydrate–electrolyte beverage and plain water. *Journal of Physiology and Anthropology of Applied Human Science* 21(2), 93–104.

Schedl, H.P., Maughan, R.J., & Gisolfi, C.V. (1994) Intestinal absorption during rest and exercise: implications for formulating oral rehydration beverages. *Medicine & Science in Sports & Exercise* 26, 267–280.

Schwellnus, M.P., Nicol, J., Laubscher, R., et al. (2004) Serum electrolyte concentrations and hydration status are not associated with exercise associated muscle cramping (EAMC) in distance runners. *British Journal of Sports Medicine* 38, 488–492.

Seifert, J., Harmon, J., & DeClercq, P. (2006) Protein added to a sports drink improves fluid retention. *International Journal of Sport Nutrition and Exercise Metabolism* 16, 420–429.

Shafiee, M.A., Charest, A.F., & Cheema-Dhadli, S., et al. (2005) Defining conditions that lead to the retention of water: the importance of the arterial sodium concentration. *Kidney International* 67, 613–621.

Shirreffs, S.M. & Maughan, R.J. (1998) Volume repletion after exercise-induced volume depletion in humans: replacement of water and sodium losses. *American Journal of Physiology* 274, F868–F875.

Shirreffs, S.M., Taylor, A.J., Leiper, J.B., et al. (1996) Post-exercise rehydration in man: effects of volume consumed and drink sodium content. *Medicine & Science in Sports & Exercise* 28, 1260–1271.

Shirreffs, S.M., Watson, P., & Maughan, R.J. (2007a) Milk as an effective postexercise rehydration drink. *British Journal of Nutrition* 98, 173–180. doi:10.1017/S0007114507695543

Shirreffs, S.M., Aragon-Vargas, L.F., Keil, M., et al. (2007b) Rehydration after exercise in the heat: a comparison of 4 commonly used drinks. *International Journal of Sport Nutrition and Exercise Metabolism* 17(3), 244–258.

Watson, P., Love, T.D., Maughan, R.J., et al. (2008) A comparison of the effects of milk and a carbohydrate–electrolyte drink on the restoration of fluid balance and exercise capacity in a hot, humid environment. *European Journal of Applied Physiology* 104, 633–642.

Wemple, R.D., Morocco, T.S., & Mack, G.W. (1997) Influence of sodium replacement on fluid ingestion following exercise-induced dehydration. *International Journal of Sport Nutrition* 7(2), 104–116.

Williams, M.B., Raven, P.B., Fogt, D.L., et al. (2003) Effects of recovery beverages on glycogen restoration and endurance exercise performance. *Journal of Strength and Conditioning Research* 17(1), 12–19.

Wong, S.H. & Williams, C. (2000) Influence of different amounts of carbohydrate on endurance running capacity following short term recovery. *International Journal of Sports Medicine* 21(6), 444–452.

Wong, S.H., Williams, C., Simpson, M., et al. (1998) Influence of fluid intake pattern on short-term recovery from prolonged, submaximal running and subsequent exercise capacity. *Journal of Sports Sciences* 16(2), 143–152.

Wong, S.H., Williams, C., & Adams, N. (2000) Effects of ingesting a large volume of carbohydrate–electrolyte solution on rehydration during recovery and subsequent exercise capacity. *International Journal of Sport Nutrition and Exercise Metabolism* 10(4), 375–393.

Wong, S.H., Chen, Y.J., Fung, W.M., et al. (2009) Effect of glycemic index of meals on recovery and subsequent endurance capacity. *International Journal of Sports Medicine* 30(12), 898–905.

Yawata, T. (1990) Effect of potassium solution on rehydration in rats: comparison with sodium solution and water. *Japan Journal of Physiology* 40(3), 369–381.

Chapter 17

Nutritional Effects on Central Fatigue

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Definitions of Fatigue

In exercise physiology, fatigue has been defined as an acute impairment of exercise performance which leads to an inability to produce the maximum force output possible due to metabolite accumulation or substrate depletion (St Clair Gibson et al., 2003). However, fatigue will not only occur at the peripheral level, since there is ample evidence that mechanisms at the central nervous system (CNS) are also implicated in the genesis of fatigue. Fatigue has many definitions. Be it a "failure to maintain the required force" or an "inability to continue working at a given exercise intensity," fatigue results in an acute impairment of exercise performance that includes both an increase in the perceived effort necessary to exert a desired force or power output and the eventual inability to produce that force or power output (Davis & Bailey, 1997).

It is obvious that fatigue not only occurs at the peripheral level, but that the "perception" of fatigue is processed at the central level (St Clair Gibson et al., 2003) and that cerebral metabolism and neurohumoral or neurotransmitter responses during exercise can be disturbed leading to fatigue (Marino, 2004). It seems thus that many parameters affect the capacity to "perform" during exercise and all will depend on the type of exercise, the duration of exercise, and environmental factors. The processes that lead to decrements in performance can occur at every level of the brain–muscle pathway and although literature usually makes a distinction between peripheral and central fatigue, one should be aware that both pathways are possibly integrated.

As substrate availability is important, the depletion of muscle glycogen is often linked with muscle fatigue. At a certain time point during prolonged exercise, there will be an inability to maintain a sufficient rate of ATP resynthesis, secondary to reduced availability of pyruvate and key metabolic intermediates with the appearance of fatigue. Besides the depletion of energy sources, the accumulation of metabolic by-products such as lactate and the hydrogen ion after breakdown of glycogen and glucose and ammonia after the breakdown of both ATP and amino acids, has also been implicated in the onset of peripheral fatigue.

Much research has been performed on the involvement of the motor pathways in fatigue. Neuromuscular fatigue occurs when a progressive exercise-induced failure of voluntary activation of the muscle exists together with a gradual failure to drive motor neurons. Taylor stated that central fatigue can be demonstrated by an increase in the increment of force induced by nerve stimulation during a maximal voluntary contraction (Taylor et al., 2006). If extra force can be evoked by stimulating the motor neurons (superimposed twitch), central fatigue takes place, possibly due to supraspinal mechanisms (Taylor et al., 2006).

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Brain neurotransmitter activity has been implicated in the regulation of cardiovascular and endocrine responses during exercise (Meeusen, 1999). A number of neurotransmitters, and especially the central monoamines, are strong candidates for inducing the centrally mediated effects of fatigue during exercise. The monoamines serotonin (5-hydroxytryptamine; 5-HT), dopamine (DA), and noradrenaline (NA) play a key role in signal transduction between neurons, and exercise-induced changes in the concentrations of these neurotransmitters (especially 5-HT & DA) have been linked to central fatigue. Newsholme et al. (1987) developed the first hypothesis based on changes in central neurotransmission to explain fatigue i.e., the "central fatigue hypothesis." This hypothesis is based on disturbances in brain 5-HT concentrations, as this neurotransmitter is involved in changes in sleep-wakefulness, emotion, sleep, appetite, the hypothalamic–pituitary axis, and numerous physiological functions (Meeusen et al., 2006). During exercise, the entry of tryptophan (TRP), precursor of 5-HT, into the CNS through the blood–brain barrier is favored by increased muscle use of branched-chain amino acids (BCAAs) and elevated plasma fatty acids as this elevates the ratio of unbound TRP to BCAA. This increases the amount of TRP crossing the blood–brain barrier, leading to higher 5-HT concentrations in the brain (Davis et al., 2000; Meeusen et al., 2006; Roelands & Meeusen, 2010).

Events arising entirely from within the brain can influence an individual's sensation of fatigue and thus potentially affect performance. This opens an opportunity to manipulate the CNS through changes in diet or supplementation with specific nutrients, including amino acids (BCAA, tyrosine), carbohydrates (CHO), and caffeine.

Nutritional Influences on Central Fatigue

Branched-Chain Amino Acids

Fernstrom (2005) clearly indicated the importance of the BCAA. Leucine, isoleucine, and valine participate directly and indirectly in a variety of important biochemical functions in the brain. These include protein synthesis, the production of energy, and the synthesis of the amine neurotransmitters 5-HT and the catecholamines DA and NA, which are derived from the aromatic amino acids TRP, phenylalanine, and tyrosine (Fernstrom, 2005). In relation to a connection between dietary BCAA intake and brain function, however, only the production of the amine neurotransmitters appears clearly to have been linked to diet. This link occurs for several metabolic reasons: first, dietary protein contains considerable amounts of the BCAA (e.g., 15–20% of the amino acid content of animal-based proteins); second, a major fraction of ingested BCAA is not metabolized by the liver and passes into the systemic circulation after a meal, causing plasma concentrations to rise appreciably and in proportion to the protein content of the meal; third, plasma BCAA are transported into the CNS by a transporter, located at the blood–brain barrier on CNS capillary endothelial cells, shared by a number of large neutral amino acids, including the aromatic amino acids, TRP, phenylalanine, and tyrosine. Thus, the ingestion of BCAA causes rapid elevation of their plasma concentrations, increases their uptake into brain, and decreases the brain uptake and levels of TRP, phenylalanine, and tyrosine (Fernstrom, 2005). Consequently, there is a decrease in the synthesis of 5-HT, DA, and NA.

It was hypothesized that by reducing the production of 5-HT in the brain, feelings of fatigue could be attenuated and performance enhanced. Supplementation of BCAA has been proposed as a possible strategy to limit the development of central fatigue. BCAAs compete with the precursor of 5-HT (TRP) for transport across the blood–brain barrier. If more BCAAs are available in the circulation, then more will be transported across the blood–brain barrier at the cost of TRP. This would imply that with increased BCAA availability, less TRP will be available in the brain, less 5-HT would be formed, and consequently fatigue would be reduced. The link between the dietary BCAA and the production of the amine neurotransmitters has been demonstrated in several animal studies employing brain microdialysis techniques. The development of *in vivo* brain microdialysis has enabled the direct analyses of extracellular neurotransmitters and metabolites from the brain of resting and active animals with limited tissue trauma. Meeusen et al. (1996) demonstrated that

increased TRP availability resulted in an elevation in extracellular 5-HT and 5-hydroxyindoleacetic acid concentrations in 24-hour fasted rats. Exercise for 1 hour further increased extracellular levels of 5-HT. Good evidence for the role of BCAA in limiting TRP entry into the CNS and attenuating the increase in 5-HT has also been reported (Gomez-Merino et al., 2001). During the placebo trial (saline infusion) a progressive increase in extracellular 5-HT was apparent in the hippocampus as exercise continued, but this elevation was abolished when exercise was preceded by an infusion of valine.

Although this is a very attractive theory, there is limited or only circumstantial evidence to suggest that exercise performance in humans can be altered by nutritional manipulation through BCAA supplements. Madsen et al. (1996), Struder et al. (1998), and van Hall et al. (1995) all attempted to influence the plasma free TRP to BCAA ratio by BCAA supplementation but failed during exercise in normal ambient temperature (Watson et al., 2004). However, one laboratory study conducted by Mittleman et al. (1998) reported a marked improvement in exercise capacity. Male and female subjects performed cycle exercise in the heat (34°C). The purpose of studying exercise in a warm environment was to increase the central component of fatigue. A significant improvement in physical performance was found: the average exercise time to exhaustion during ergometer cycle exercise at 40% of the maximal oxygen uptake increased from 137 to 153 minutes when the subjects received BCAA as compared with the placebo. The BCAAs were given to the subjects before and during exercise. Watson et al. (2004) were however unable to find an ergogenic effect of BCAA supplementation in the heat (30°C) and found a large variation in response to the BCAA supplementation. These authors (Watson et al., 2004) concluded that it may be that the effect of the nutritional supplementation is simply too small to produce a functional change in central neurotransmission in some individuals.

BCAAs have also been given in combination with CHO during exercise. It is a well-known fact that ingestion of CHO during prolonged exercise can delay fatigue, which is often suggested to be due to the maintenance of blood glucose levels and the supply of energy when muscle glycogen levels are low. However, it is possible that CHO might also have an effect on central fatigue by delaying the rise in plasma concentration of free TRP (Blomstrand, 2001). Intake of CHO before or during exercise has been reported to reduce the exercise-induced increase in the plasma free fatty acid concentration (McConell et al., 1999), probably owing to a stimulation of insulin secretion, which is known to inhibit lipolysis. Furthermore, Davis et al. (1992) showed that besides the delayed rise in free fatty acids, CHO are also responsible for the delay in the free TRP concentration during prolonged exercise. However, when the exercise continues for longer than 2–3 hours, there is an increase in the plasma concentration of free fatty acids and free TRP even when CHO are consumed (Davis et al., 1992; McConell et al., 1999). No difference in performance was discovered between intake of BCAAs plus CHO and CHO alone, i.e., no additional benefit of BCAA on physical performance could be found (for review see Blomstrand, 2001). An early field study suggested that both physical (race time) and mental (color and word tests) performance were enhanced in those subjects receiving BCAA prior to a marathon or cross-country race. However, enhanced performance was witnessed only in subjects completing the marathon in times slower than 3 hours 5 minutes. The authors suggested that a lack of effect in the faster runners may have been due to their increased resistance to the feelings associated with central and peripheral fatigue (Blomstrand et al., 1991). While there is some evidence of BCAA ingestion influencing ratings of perceived exertion and mental performance, the results of several well-controlled laboratory studies have failed to demonstrate a clear positive effect on exercise capacity or performance during prolonged fixed intensity exercise to exhaustion, prolonged time trial performance, incremental exercise, or intermittent shuttle-running.

Tyrosine

Tyrosine, or 4-hydroxyphenylalanine, is one of the 22 amino acids that are used by cells to synthesize proteins. It can be synthesized in the body from phenylalanine, and is found in many high-protein

foods such as soy products, chicken, turkey, fish, peanuts, almonds, avocados, milk, cheese, yogurt, and sesame seeds. Acute consumption of tyrosine increases the plasma concentration ratio of tyrosine to other large neutral amino acids such as leucine, isoleucine, valine, and TRP. Tyrosine shares a common transport molecule with large neutral amino acids at the blood–brain barrier, so an increase in the tyrosine ratio causes an increase in brain tyrosine and a decrease in large neutral amino acid concentration. Consequently, this would lead to an increase in brain DA and NA concentration (Tumilty et al., 2011). A review by Lieberman (2003) identified tyrosine as a leading candidate for use as a cognitive performance enhancer in military operations.

A series of preclinical, animal studies has been conducted with this amino acid. In aggregate, these studies clearly indicate that tyrosine reduces many of the adverse effects of acute stress on cognitive performance in a wide variety of stressful environments. Given this unique potential use, it is not surprising that tyrosine has been the focus of considerable military interest for its cognitive "antistress" effects (Lieberman, 1994). Both DA and NA play a key role in a variety of stress-related behaviors. Furthermore, NA is critical for modulating the central stress response. Many forms of stress cause the release of large amounts of NA in several brain regions, which play a central role in the regulation of stress-related behaviors. During acute stress, NA is depleted, and when additional substrate in the form of tyrosine is provided, the release of NA increases (Yeghiayan et al., 2001).

Tyrosine affects the same neurotransmitter systems as the amphetamines and related drugs, which are potent performance-enhancing compounds, although they have many side effects (Yeghiayan et al., 2001). In a series of animal studies conducted at several laboratories, it has been shown that the effects of heat, cold, high altitude, and psychological stress are all mitigated by the administration of tyrosine (for review see Lieberman, 2003). Although it has been difficult to conclusively demonstrate that tyrosine has beneficial effects in humans, in part due to ethical concerns, the preponderance of evidence suggests that tyrosine is useful as an acute treatment to prevent stress-related declines in cognitive function. Stressors that have been evaluated in human studies include the psychological stress of military operations, cold stress, the combination of cold and high altitude stress, cardiovascular stress designed to simulate space flight, and sleep deprivation. A wide variety of cognitive tasks, symptoms, and moods have been shown to be affected by tyrosine administration in acutely stressed volunteers. Banderet and Lieberman (1989) found that vigilance, choice reaction time, pattern recognition, coding, and complex behaviors, such as map-compass reading, were all improved by tyrosine administration when volunteers were exposed to the combination of cold and high altitude (hypoxia). Many of the adverse symptoms associated with these stressors, such as perceived coldness and headache, were reduced by tyrosine administration and many mood states, such as fatigue, confusion, and tension improved (Lieberman, 2003).

One study has measured a net brain uptake of tyrosine during prolonged exercise in humans (Nybo et al., 2003); however, acute tyrosine supplementation did not improve either prolonged exercise capacity (Struder et al., 1998) or performance (Chinevere et al., 2002) in temperate conditions. Exercise in the heat on the other hand represents a specific demand on brain DA which is not apparent in temperate conditions (Roelands et al., 2008; Watson et al., 2005). Therefore, brain tyrosine requirement may be greater with the cumulative demands of exercise and heat stress and may become limiting to DA synthesis and release. Very recently, Tumilty et al. (2011) assessed the effects of acute tyrosine supplementation on exercise capacity in the heat. Eight healthy male subjects cycled until exhaustion at an intensity above the lactate threshold but below critical power threshold. This study indicates, for the first time, that supplementing a nutritional DA precursor 1 hour before exercise is associated with increased exercise capacity in the heat, and shows that tyrosine availability, at least in part, may influence prolonged exercise tolerance in conditions of heat stress (Tumilty et al., 2011). Further studies are needed to identify the influence of regular supplementation of large amounts of tyrosine (5–10 g) on health due to chronic changes in sympathetic nervous system activity.

Carbohydrate

Another nutritional strategy that may influence the development of central fatigue is CHO feeding. Glucose ingestion stimulates the secretion of insulin and blunts the exercise-induced rise in both plasma free fatty acids and free TRP (Karelis et al., 2010). Work by Davis et al. (1992) demonstrated that in a control situation, plasma free TRP increased by about sevenfold, when subjects performed prolonged exercise for 200 minutes at 68% VO_2 max. This was associated with an increase in plasma free fatty acid levels and reduced blood glucose levels from 5 to 4 mmol/l. When subjects ingested ~1 g/min of CHO, the blood glucose level was maintained at 5.5 mmol/l, changes in plasma free TRP as well as free fatty acids were significantly attenuated, and fatigue was delayed by ~1 hour (Davis et al., 1992). In line with this, Nybo (2003) showed that the average force production during a sustained maximal muscle contraction was decreased after 3 hours of exercise at 60% VO_2 max in endurance-trained subjects. Blood glucose levels decreased from 4.5 to 3 mmol/l after exercise and a diminished activation drive from the CNS was apparent. This central fatigue was reversed when euglycemia (4.5 mmol/l) was maintained with the ingestion of 200 g of CHO. In addition, it was easier for the subjects to retain power output at the end of prolonged exercise when hypoglycemia was prevented (Karelis et al., 2010). CHO ingestion during exercise attenuates the cerebral uptake of TRP as well as prevents the development of hypoglycemia, but the peripheral role of CHO ingestion in acting as a substrate for muscle metabolism cannot be ruled out. The beneficial effect of CHO supplementation during prolonged exercise could also relate to increased (or maintained) substrate delivery for the brain, with a number of studies indicating that hypoglycemia affects brain function and cognitive performance.

CHO feeding has been shown to improve higher intensity exercise performance lasting approximately 60 minutes even though the estimated amount of glucose delivered to the muscle during this period was estimated to be very small (Jeukendrup et al., 1997). The same research group infused glucose during a time trial to study the performance effects with increased CHO availability. Failure to observe a benefit of glucose infusion on time trial performance (Carter et al., 2004a) prompted this group to suggest an alternative mechanism for the ergogenic effect of CHO centered around the activation of CHO receptors found in the mouth. Carter et al. (2004b) reported a 3% (placebo (PLA) 61.37 minutes; CHO 59.57 minutes) improvement in time trial performance with the rinsing of a maltodextrin solution around the mouth before and during exercise. No solution was actually swallowed during the protocol, suggesting that this performance benefit may have been mediated through direct communication between receptors present in the mouth and the brain. Other groups have also looked into the effects of a CHO mouth rinse on performance. Pottier et al. (2010) found a performance improvement on a 60-minute time trial rinsing with a CHO-electrolyte solution, while Rollo et al. (2008) showed ergogenic effects on a 30-minute time trial. Interestingly, most studies that found an effect were carried out in the fasted state. When a CHO mouth rinse was performed in a fed state, no effect on performance in a 45-minute (Whitham & McKinney, 2007) or a 60-minute time trial was observed (Beelen et al., 2009). The authors suggested that oral perception of CHO perhaps only plays a role when muscle and liver glycogen stores are reduced. This finding was, however, not replicated in a very recent study by Fares and Kayser (2011). In this study, a mouth rinse with a maltodextrin solution increased time to exhaustion in both fed and fasted states in nonathletic male subjects (Fares & Kayser, 2011). The concept of the CHO mouth rinse has been supported by work investigating brain activity following the ingestion of a bolus of glucose (Liu et al., 2000) and research demonstrating activation of several brain regions after rinsing CHO solutions within the mouth (Chambers et al., 2009). These studies highlight a marked increase in brain activation, occurring immediately after CHO enters the mouth, with a second spike in activity observed 10 minutes following ingestion, presumably occurring as the substrate enters the circulation. These findings are very novel and suggest an interesting mechanism of action. Further investigation of CHO receptors in the mouth is certainly warranted.

Caffeine

Caffeine has long been recognized as an ergogenic aid. It is one of the substances that lie in the grey area between a nutritional supplement and a drug. For a while, caffeine use was restricted for athletes and it was only removed from the list of controlled substances in January 2004, when it was put on the monitoring list. Current research supports a CNS effect mediated by antagonism of adenosine receptors as the most likely cause (Davis et al., 2003). Caffeine influences both adenosine A(1) and A(2A) receptors, and the modulation of DA transmission through A2A receptors has been implicated as one of the most important CNS effects. Since caffeine is known to antagonize adenosine receptors in the brain, and adenosine inhibits the release of DA, logically, caffeine will induce higher brain DA concentrations (Davis et al., 2003). Human studies using a variety of protocols have shown performance improvements after caffeine intake (Roelands & Meeusen, 2010).

Besides ergogenic effects, caffeine also increases resting energy expenditure, mental energy, cognitive function, and neuromuscular coordination; elevates mood; and relieves anxiety (Glade, 2010). Caffeine may thus reduce perception of effort and pain during exercise, thereby allowing subjects to perform at higher workloads for a longer period of time. Caffeine has been shown to be effective in relatively low doses (3 mg/kg) and its effect seems to level off at 6 mg/kg, and therefore very high doses should not be recommended. Given the widespread use of caffeine by many, the level of habitual intake may be an important factor to consider when undertaking caffeine supplementation with the view to enhance performance. In some naive individuals, caffeine can produce several side effects, such as tachycardia and palpitations, nervousness, dizziness, and gastrointestinal symptoms that may be detrimental to performance. The positive (and possible negative) effects of caffeine seem very individually determined so prior experience with doses and timing is essential before using supplementation in competitive environments. Table 17.1 provides an overview of the performance effects of supplements that act on the CNS.

Table 17.1 Effects of supplements that act on the central nervous system

Supplement	Dose(s) studied	Proposed effect on the brain	Does it influence performance?
Branched-chain amino acids (BCAAs)	5–20 g	Reduces brain serotonin production	• Evidence is generally weak. A few studies suggest an effect, but many more find no benefit • May reduce perception of effort and enhance mental performance during exercise
Caffeine	2–10 mg/kg BM	Reduces effect of adenosine in the brain	• Performance in events lasting more than a couple of minutes can be enhanced by caffeine • Alters mood; increases alertness and reaction times • Large individual variation in sensitivity to caffeine
Carbohydrate (CHO)	30–90 g/h	Increased energy for the brain. Influences neurotransmission and cerebral metabolism	• Evidence suggests a benefit to performance in most cases • Possible that part of this ergogenic effect is due to influence on the CNS
Tyrosine	5–10 g	Increases production of brain dopamine and noradrenaline	• Few studies investigating effect of tyrosine on performance, but show no effect on physical performance • Some evidence of a benefit to mood, memory, and cognitive function

Conclusions

Originally, fatigue was attributed solely to peripheral factors, such as muscular and cardiovascular factors. There is now proof that fatigue can also occur at the level of the brain. This so-called "central fatigue" compromises specific alterations in the functioning of the CNS. Although the rationale for the central fatigue hypothesis is solid, the largely inconsistent findings of nutritional manipulation studies with BCAA and tyrosine make it difficult to draw any firm conclusions regarding the role of central neurotransmission in the fatigue process. Both CHO and caffeine have been shown to be ergogenic in different exercise protocols. The mechanism by which this happens is, however, not entirely clear and might very well be a combination of both central and peripheral aspects. Recent studies have begun to incorporate new technologies such as transcranial magnetic stimulation, *in vivo* microdialysis, and other dynamic imaging technologies to better understand what happens in the brain, but the search for the central mechanisms of fatigue and the role of nutrition in the development of fatigue remain to be unraveled!

References

Banderet, L.E. & Lieberman, H.R. (1989) Treatment with tyrosine, a neurotransmitter precursor, reduces environmental stress in humans. *Brain Research Bulletin* 22, 759–762.

Beelen, M., Berghuis, J., Bonaparte, B., et al. (2009) Carbohydrate mouth rinsing in the fed state: lack of enhancement of time-trial performance. *International Journal of Sport Nutrition and Exercise Metabolism* 19(4), 400–409.

Blomstrand, E. (2001) Amino acids and central fatigue. *Amino Acids* 20(1), 25–34.

Blomstrand, E., Hassmén, P., Ekblom, B., et al. (1991) Administration of branched-chain amino acids during sustained exercise—effects on performance and on plasma concentration of some amino acids. *European Journal of Applied Physiology* 63, 83–88.

Carter, J.M., Jeukendrup, A.E., Mann, C.H., et al. (2004a) The effect of glucose infusion on glucose kinetics during a 1-h time trial. *Medicine & Science in Sports & Exercise* 36(9), 1543–1550.

Carter, J.M., Jeukendrup, A.E., & Jones, D.A. (2004b) The effect of carbohydrate mouth rinse on 1-h cycle time trial performance. *Medicine & Science in Sports & Exercise* 36(12), 2107–2111.

Chambers, E.S., Bridge, M.W., & Jones, D.A. (2009) Carbohydrate sensing in the human mouth: effects on exercise performance and brain activity. *Journal of Physiology* 587(Pt 8), 1779–1794.

Chinevere, T.D., Sawyer, R.D., Creer, A.R., et al. (2002) Effects of L-tyrosine and carbohydrate ingestion on endurance exercise performance. *Journal of Applied Physiology* 93, 1590–1597.

Davis, J.M. & Bailey, S.P. (1997) Possible mechanisms of central nervous system fatigue during exercise. *Medicine & Science in Sports & Exercise* 29(1), 45–57.

Davis, J.M., Bailey, S.P., Woods, J.A., et al. (1992) Effects of carbohydrate feedings on plasma free-tryptophan and branched-chain amino acids during prolonged cycling. *European Journal of Applied Physiology* 65, 513–519.

Davis, J.M., Alderson, N.L., & Welsh, R.S. (2000) Serotonin and central nervous system fatigue: nutritional considerations. *American Journal of Clinical Nutrition* 72(2), 573S–578S.

Davis, J.M., Zhao, Z., Stock, H.S., et al. (2003) Central nervous system effects of caffeine and adenosine on fatigue. *American Journal of Physiology Regulatory, Integrative and Comparative Physiology* 284, R399–R404.

Fares, E.J. & Kayser, B. (2011) Carbohydrate mouth rinse effects on exercise capacity in pre- and postprandial states. *Journal of Nutrition and Metabolism*, Article ID 385926, 6 pages.

Fernstrom, J.D. (2005) Branched-chain amino acids and the brain. *Journal of Nutrition* 135(6), 1539S–1546S.

Glade, M.J. (2010) Caffeine—not just a stimulant. *Nutrition* 26, 932–938.

Gomez-Merino, D., Bequet, F., Berthelot, M., et al. (2001) Evidence that the branched-chain amino acid L-valine prevents exercise-induced release of 5-HT in rat hippocampus. *International Journal of Sports Medicine* 22(5), 317–322.

Jeukendrup, A., Brouns, F., Wagenmakers, A.J., et al. (1997) Carbohydrate-electrolyte feedings improve 1h time trial cycling performance. *International Journal of Sports Medicine* 18(2), 125–129.

Karelis, A.D., Smith, J.W., Passe, D.H., et al. (2010) Carbohydrate administration and exercise performance. What are the potential mechanisms involved? *Sports Medicine* 40(9), 747–763.

Lieberman, H.R. (1994) Tyrosine and stress: human and animal studies. In *Food Components to Enhance Performance* (ed. B. Marriott), pp. 277–299. National Academy Press, Washington, DC.

Lieberman, H.R. (2003) Nutrition, brain function and cognitive performance. *Appetite* 40, 245–254.

Liu, Y., Gao, J.H., Liu, H.L., et al. (2000) The temporal response of the brain after eating revealed by functional MRI. *Nature* 405(6790), 1058–1062.

Madsen, K., MacLean, D.A., Kiens, B., et al. (1996) Effects of glucose, glucose plus branched-chain amino acids, or placebo on bike performance over 100 km. *Journal of Applied Physiology* 81, 2644–2650.

Marino, F.E. (2004) Anticipatory regulation and avoidance of catastrophe during exercise-induced hyperthermia. *Comparative Biochemistry and Physiology: Part B Biochemistry and Molecular Biology* 139(4), 535–538.

McConell, G., Snow, R.J., Proietto, J., et al. (1999) Muscle metabolism during prolonged exercise in humans: influence of carbohydrate availability. *Journal of Applied Physiology* 87, 1083–1086.

Meeusen, R. (1999) Overtraining and the central nervous system—the missing link? In *Overload, Performance Incompetence, and Regeneration in*

Sport (eds. M. Lehmann, C. Foster, U. Gastmann, et al.), pp. 187–202. Kluwer Academic/Plenum Publishers, New York.

Meeusen, R., Thorre, K., Chaouloff, F., et al. (1996) Effects of tryptophan and/or acute running on extracellular 5-HT and 5-HIAA levels in the hippocampus of food-deprived rats. *Brain Research* 740(1–2), 245–252.

Meeusen, R., Watson, P., Hasegawa, H., et al. (2006) Central fatigue. The serotonin hypothesis and beyond. *Sports Medicine* 36(10), 881–909.

Mittleman, K.D., Ricci, M.R., & Bailey, S.P. (1998) Branched-chain amino acids prolong exercise during heat stress in men and women. *Medicine & Science in Sports & Exercise* 30, 83–91.

Newsholme, E.A., Acworth, I., & Blomstrand, E. (1987) Amino acids, brain neurotransmitters and a function link between muscle and brain that is important in sustained exercise. In *Advances in Myochemistry* (ed. G. Benzi), pp. 127–133. John Libbey Eurotext, London.

Nybo, L. (2003) CNS fatigue and prolonged exercise: effect of glucose supplementation. *Medicine & Science in Sports & Exercise* 35(4), 589–594.

Nybo, L., Nielsen, B., Blomstrand, E., et al. (2003) Neurohumoral responses during prolonged exercise in humans. *Journal of Applied Physiology* 95, 1125–1131.

Pottier, A., Bouckaert, J., Gilis, W., et al. (2010) Mouth rinse but not ingestion of a carbohydrate solution improves 1 h cycle time trial performance. *Scandinavian Journal of Medicine and Science in Sports* 20(1), 105–111.

Roelands, B. & Meeusen, R. (2010) Alterations in central fatigue by pharmacological manipulations of neurotransmitters in normal and high ambient temperature. *Sports Medicine* 40(3), 229–246.

Roelands, B., Hasegawa, H., Watson, P., et al. (2008) The effects of acute dopamine reuptake inhibition on performance. *Medicine & Science in Sports & Exercise* 40(5), 879–858.

Rollo, I., Williams, C., Gant, N., et al. (2008) The influence of carbohydrate mouth rinse on self-selected speeds during a 30-min treadmill run. *International Journal of Sports Nutrition and Exercise Metabolism* 18(6), 585–600.

St Clair Gibson, A., Baden, D.A., Lambert, M.I., et al. (2003). The conscious perception of the sensation of fatigue. *Sports Medicine* 33(3), 167–176.

Struder, H.K., Hollmann, W., Platen. P., et al. (1998) Influence of paroxetine, branched-chain amino acids and tyrosine on neuroendocrine system responses and fatigue in humans. *Hormone and Metabolic Research* 30, 188–194.

Taylor, J.L., Todd, G., & Gandevia, S.C. (2006) Evidence for a supraspinal contribution to human muscle fatigue. *Clinical and Experimental Pharmacology and Physiology* 33, 400–405.

Tumilty, L., Davison, G., Beckmann, M., et al. (2011) Oral tyrosine supplementation improves exercise capacity in the heat. *European Journal of Applied Physiology* 111, 2941–2950.

van Hall, G., Raaymakers, J.S., Saris, W.H., et al. (1995) Ingestion of branched-chain amino acids and tryptophan during sustained exercise in man: failure to affect performance. *Journal of Physiology* 486, 789–794.

Watson, P., Shirreffs, S.M., & Maughan, R.J. (2004) The effect of acute branched-chain amino acid supplementation on prolonged exercise capacity in a warm environment. *European Journal of Applied Physiology* 93(3), 306–314.

Watson, P., Hasegawa, H., Roelands, B., et al. (2005) Acute dopamine/noradrenaline reuptake inhibition enhances human exercise performance in warm, but not temperate conditions. *Journal of Physiology* 565, 873–883.

Whitham, M. & McKinney, J. (2007) Effect of a carbohydrate mouthwash on running time trial performance. *Journal of Sports Sciences* 25(12), 1385–1392.

Yeghiayan, S., Luo, S., Shukitt-Hale, B., et al. (2001) Tyrosine improves behavioral and neurochemical deficits caused by cold exposure. *Physiology and Behavior* 72, 311–316.

PART 3

MICRONUTRIENTS AND DIETARY SUPPLEMENTS

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Chapter 18

Vitamins, Minerals, and Sport Performance

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Introduction

Overall nutritional intake, including the proper balance between macronutrients and micronutrients, is important for optimum physical performance (Farajian et al., 2004; Patlar et al., 2010). Vitamins and minerals are required for hundreds of metabolic reactions in the body, including those involved in energy metabolism. Though research has not provided definitive evidence that athletes need to supplement with vitamins and minerals for optimum performance, many athletes use supplements even if they are obtaining adequate amounts of nutrients from food sources (Silva et al., 2010; Wienecke & Gruenwald, 2007).

Nutritional deficiencies will be detrimental to the health and performance of all athletes and might also be a factor in the female athlete triad (disordered eating, amenorrhoea, and osteoporosis) (Gabel, 2006). Men have been reported to have higher nutrient intakes than women, probably because of a higher overall energy intake (Aerenhouts et al., 2008; Farajian et al., 2004). Nonetheless, Garrido et al. (2007) noted that, even when a group of adolescent male soccer players had higher energy intakes than females, they still had low intakes of vitamin A, vitamin E, folate, and magnesium.

In this chapter, research on vitamins and minerals among various athletes from different countries will be reviewed. Due to the limitations of space, and lack of research in some areas, not all vitamins and minerals are discussed.

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Vitamin and Mineral Supplementation Use Among Athletes

Athletes consume vitamin and mineral supplements for a number of reasons, including improved athletic performance, to correct "deficient" intakes, and/or for recovery purposes. Though many of these reasons have not been well established, supplement use continues to remain high among athletes.

Hiekkenen et al. (2011) used questionnaires to assess the use of dietary supplements, as well as reasons for their use, among Finnish Olympic athletes in 2002 (n = 446) and 2009 (n = 372). They reported a high prevalence of supplement use during 2002 (81%) and 2009 (73%), with a slight decline from 2002 to 2009. Vitamin D supplementation was low in both years (0.7% and 2.0%, respectively), but use of omega-3 fatty acid increased from 11% in 2002 to 19% in 2009 (p = 0.002). The main reason given for the use of these supplements was to prevent nutritional deficiencies, but those who took other nutritional supplements did so for recovery from exercise. Only 27% of the athletes consulted with diet specialists in 2009, demonstrating the need for professionals in nutrition and dietetics to be more involved with nutritional counseling for elite athletes.

Aerenhouts et al. (2008) evaluated the dietary habits of Flemish adolescent track and field athletes (29 girls, 31 boys), who had a minimum of 2 years

of track and field training practice. They reported that all athletes consumed breakfast on a daily basis, that girls consumed more fruit and boys consumed more juices and sports drinks between meals. Total fiber intake was well below the recommended Belgian intakes. Most pertinent to this chapter, vitamin and mineral intakes were usually low, even though 38% of the athletes reported micronutrient supplementation.

Evaluation of the dietary intakes of the Greek national swimming and water polo teams using a 24-hour dietary recall and a food frequency questionnaire (Farajian et al., 2004) revealed that 71% of the males and 93% of the females did not meet the recommended intakes for at least one of the antioxidant vitamins (e.g., vitamins A, C, and E), which was a direct result of their low fruit and vegetable intake.

Vitamin and Mineral Supplementation and Athletic Performance

The effects of vitamin and mineral supplementation on athletic performance have been studied in many types of athletes and most researchers have not reported beneficial effects.

Knechtle et al. (2008) examined the effect of vitamin and mineral supplementation prior to a multistage, ultra-endurance run on race performance at the 2006 Deutschlandlauf in Germany. In this race, athletes ran across Germany from north to south, running more than 1200 km over 17 consecutive stages. They studied 20 male ultra-endurance runners (46 ± 10 years of age, 71.8 ± 5.2 kg of body weight, 179 ± 6-cm tall, with a body mass index (BMI) of 22.5 ± 1.9 kg/m^2) by asking them to first complete a questionnaire concerning their use of vitamin and mineral supplements 4 weeks prior to the race. Based on the questionnaire, the researchers reported that six runners consumed multivitamins, five consumed vitamin E, four consumed vitamin C, two consumed a vitamin B complex, 1 consumed folate, and 11 reported no vitamin supplement intake. Seven runners stated that they consumed a multi-mineral supplement, while nine consumed magnesium, five consumed zinc, three consumed iron, three consumed calcium, and eight reported no mineral supplementation use. Perhaps unsurprisingly, Knechtle et al. (2008) reported no differences in performance between athletes who consumed vitamin and/or mineral supplements and those who reported no intake.

There has been an increase in masters-trained athletes (defined in many different ways, but typically defined as individuals who are ≥30 years of age). In a study conducted in 16 endurance-trained older individuals (66 ± 6 years of age), Louis et al. (2010) examined the effect of a vitamin and mineral supplement on muscular activity and cycling efficiency during a heavy cycling trial. The participants cycled about 6 hours/week and ran about 1 hour/week. The supplement, Isoxan Senior, was reported to contain the following: 106.4 mg of vitamin C, 16 mg of vitamin E, 1.7 mg of vitamin B_1 (thiamin), 2 mg of vitamin B_2 (riboflavin), 20 mg of vitamin B_3 (niacin), 2.6 mg of vitamin B_6, 400 mg of vitamin B_9 (folate), 3.2 mg of vitamin B_{12}, 133.2 mg of calcium, 133.2 mg of magnesium, 16 mg of zinc, 2 mg of iron, 4.6 mg of manganese, 2.64 mg of copper, 93.2 mg of selenium, and 5.7 mg of β-carotene. One month prior to the supplementation, all participants were tested for dominant leg strength and their maximal oxygen consumption (VO_2 max) using a cycle ergometer. After this test, participants were randomly assigned to the supplement group (n = 8) or placebo group (n = 8). Following 21 days of supplementation (or the placebo), participants returned to the laboratory and performed a 10-minute controlled cycling test and a maximum voluntary contraction leg extension measure. Electromyographic (EMG) activity was measured in the vastus lateralis and vastus medialis during the knee extensor measures. Twenty-four to forty-eight hours after the second session, participants performed a third session that consisted of 10 sets of 10 repetitions on the horizontal leg press at 70% of their 1-repetition maximum (1-RM), with 90 seconds rest between sets. The main findings of this study were an interaction between fatigue and multi-vitamin/mineral supplementation during the 10-minute constant intensity cycling and an altered EMG activity during the dynamic and isometric leg strength exercises. Due to the small sample size and short length of time of supplementation, however, these results should be treated with caution.

Though many athletes do not like to hear that foods are their best source of energy and micronutrients, they need to be taught the importance of eating a variety of foods. In addition, because many athletes are randomly tested, they need to be aware that supplements may be tainted with prohibited substances. This is extremely important since many athletes do not know their supplements' active ingredients (Dascombe et al., 2010) or the potential hazards of supplementation (Braun et al., 2009). Even when athletes may believe supplements are associated with positive doping violations, they still continue to take them (Dascombe et al., 2010). Athletes need to be better educated on the consequences of supplement use (Tian et al., 2009). Finally, it must be stressed that the studies represented here had small sample sizes, were not long in duration, and one was based on self-reported vitamin–mineral intake, which decreased the control the researchers had on the participants' intake.

Vitamins

Vitamin A

Vitamin A is necessary for vision, maintaining structure of epithelial cells, and immune responses (Institute of Medicine [IOM], 2002). Vitamin A is important in growth and maintenance of bone tissue (Garrido et al., 2007). Low vitamin A intakes have been reported in some adolescent athletes (Garrido et al., 2007; Martinez et al., 2011). Good food sources of vitamin A include liver, carrots, and sweet potatoes (providing β-carotene, which needs to be converted to vitamin A) (Table 18.1).

There is an almost complete absence of studies on vitamin A supplementation and athletic performance. Patlar et al. (2010) evaluated the effects of vitamin A supplementation on trace element metabolism in seven healthy male national Turkish Taekwondo athletes. At the start of the study, these athletes were approximately 22 years of age, with an average body weight of 65 ± 3 kg, and averaged 10–12 years of training. All participants were supplemented with 100 mg of vitamin A as retinol for 6 weeks, while training 5 days/week. Blood samples were taken at baseline and end of supplementation before and after an exhausting bout of exercise. Following 6 weeks of vitamin A supplementation, boron and nickel blood levels significantly decreased ($p < 0.001$). Patlar et al. (2010) stated that the decreased blood concentrations of boron and nickel could be a result of the antioxidant effect of the vitamin A supplementation. Despite these findings, it is important to note that this was another study with a small sample size, and that there was no control group, greatly limiting the credibility of these observations.

Table 18.1 Example of food sources for vitamins presented

Vitamin	Examples of good food sources
Vitamin A	Liver and other organ meats Sweet potato with peel Carrots Spinach Fortified cereals
Vitamin D	Fish liver oils Fatty fish Fortified milk products Fortified orange juice Fortified cereals Non-food source: Formed naturally as a result of sunlight exposure
Vitamin E	Fortified cereals Sunflower seeds Almonds Peanut butter Wheat germ Vegetable oils
Vitamin B_1 (thiamin)	Whole grains Enriched/fortified breads Enriched/fortified cereals
Vitamin B_2 (riboflavin)	Milk Bread products Fortified cereals
Folate	Dark, leafy vegetables Enriched and whole grain breads Fortified cereals Strawberries
Vitamin C (ascorbic acid)	Red and green peppers Kiwis Oranges Strawberries Broccoli

Source: Adapted from http://www.webmd.com/a-to-z-guides/vitamins-and-minerals-good-food-sources (accessed June 17, 2012).

Vitamin D

Vitamin D is known as both a vitamin and a hormone, because it is synthesized in one part of the body, but elicits its effects in another part of the body. Vitamin D plays crucial roles in bone metabolism, calcium metabolism, cell proliferation and differentiation, and muscle function. Without vitamin D, only 10–15% of dietary calcium would be absorbed (Holick et al., 2006).

Vitamin D is acquired by food and sunlight (Table 18.1). When acquired by sunlight, vitamin D is synthesized in the epidermis by the action of solar ultraviolet B (UVB) photons on the skin (Bartoszewksa et al., 2010). Whether consumed by food or acquired by sunlight, vitamin D goes through several processes to be converted within the body to its most active form, 1,25 dihydroxyvitamin D_3, also known as calcitriol. Despite calcitriol being the most active form of vitamin D, its precursor, 25-hydroxyvitamin D (or calcidiol) is used to measure vitamin D status in the blood, because it has a longer half-life.

Assessment of vitamin D status has focused more in the elderly, because vitamin D status has been shown to decrease with age, especially in those individuals over 65 years of age, and has been associated with poor physical performance (Houston et al., 2011). Houston et al., (2011) evaluated the association between calcidiol and physical performance over a 1-year period in 368 community-dwelling older individuals (70–89 years of age) at-risk for disability participating in the Lifestyle Interventions and Independence for Elders Pilot (LIFE-P). Physical performance was examined by the short physical performance battery (SPPB) and a 400-m walk test at baseline, 6 months, and 12 months. Houston et al. (2011) reported that half of the participants were deficient in calcidiol (<50 nmol/l) at baseline. They also reported that low levels of vitamin D were significantly associated with lower SPPB scores and slower 400-m walk speeds. They also reported that individuals who were deficient in blood calcidiol levels at baseline, but who were not deficient at 12 months, showed significant improvements in SPPB scores compared to those who did not improve vitamin D status over 12 months. They concluded that improvements in calcidiol levels above 50 nmol/l were related to clinically significant improvements in physical performance in older individuals.

Vitamin D status has also been evaluated in the younger population. Lovell (2008) conducted a cross-sectional study in 18 female elite gymnasts (10–17 years of age) in the Australian Institute of Sport. These athletes were assessed for serum calcidiol levels and their dietary calcium intake. Of the 18 gymnasts assessed, 15 were found to have serum calcidiol levels <75 nmol/l, with six gymnasts having levels below 50 nmol/l. Furthermore, 13 of the athletes had dietary intakes of calcium below the daily recommended intakes. Lovell (2008) reported that gymnasts and other athletes, who spend most of their training indoors, should be evaluated for their vitamin D status and calcium intake.

Another factor in low vitamin D intakes and status may be a low energy intake, even though food normally makes only a small contribution to vitamin D status. Female adolescent runners, like gymnasts, try to keep their body weight low, leading to low bone mineral density (Barrack et al., 2010). Barrack et al. (2010) examined diet and menstrual histories, serum hormone concentrations and bone mineral density in 39 female cross-country runners (16 years of age). They collected 7-day dietary records, daily 24-hour dietary recalls, and measured serum levels of various hormones related to bone turnover (e.g., insulin-like growth factor-I, estradiol, parathyroid hormone, calcidiol). Bone was measured directly using dual-energy X-ray absorptiometry (DXA). They also assessed menstrual history via questionnaire. Runners who had an increased bone turnover (13 of the 39 measured), had lower levels of serum estradiol and calcidiol, bone mineral density, and fewer menstrual cycles compared to runners with normal bone turnovers. Though runners typically train outdoors, the lower energy intake can lead to lower nutrient intake (including calcium and vitamin D intake) and decreased bone mineral density. Although peak bone mineral density is not typically achieved until 30 years of age (depending on the skeletal site), energy intake at a younger age can determine whether or not a person achieves his or her peak bone mineral density. Achieving peak bone mineral density will decrease the risk of osteoporosis later in life. Osteoporosis has often been

defined as a pediatric disease that manifests itself in adulthood.

Most of the research on vitamin D status has been conducted in female athletes, but Bescos Garcia and Rodriguez Guisado (2011) examined serum calcidiol status concentrations in 21 elite male basketball players from the same professional Spanish team after their winter training (collected at the end of two seasons: April 2009 and March 2010). The athletes also completed 4-day dietary records. Twelve athletes had serum calcidiol levels <50 nmol/l and were considered deficient. Vitamin D and calcium intakes were 139 ± 78 IU/day and 948 ± 419 mg/day, respectively. They did report that high vitamin D and calcium intakes correlated with vitamin D status. Bescos Garcia and Rodriguez Guisado (2011) stated that basketball players are at a greater risk of vitamin D deficiency after the winter. This is similar to observations in gymnasts who train indoors, individuals who live in latitudes where vitamin D in the skin is not converted from November to March, and in older individuals who may not receive as much sunlight as younger individuals.

Vitamin E (α-Tocopherol)

Vitamin E's main role in the body is as an antioxidant and there has been much interest in its potential effects on oxidative damage and lipid peroxidation (Evans, 2000; IOM, 2000). Good food sources of vitamin E include nuts and oils (Table 18.1). Vitamin E is stored in the inner mitochondrial membrane of most cells at the electron transport chain (Evans, 2000). The only form of vitamin E that can be stored in blood and tissues of humans is α-tocopherol (IOM, 2000).

Oxidative stress occurs in the body on a daily basis; during maximal exercise, however, there are more reactive oxygen species (free radicals) produced as a result of greater rate of oxygen consumption (Carlsohn et al., 2010; Naziroglu et al., 2010). The increase in the production of reactive oxygen species might damage cell integrity and contractile roles of muscle cells, and thus, researchers have studied the effects of antioxidant supplementation in athletes to evaluate if they act to reduce free radical production. Vitamins C and E are both antioxidant vitamins, which can help reverse or decrease reactive oxygen species production. The effects of supplementation on blood lipid peroxidation and antioxidant levels after maximal training were evaluated in 14 basketball players (Naziroglu et al., 2010). After taking resting blood samples, the 14 players were separated into two groups: maximal training only for 35 days or maximal training plus vitamins C and E supplementation for 35 days. Vitamin E was given as DL-a-tocopheryl at 150 mg, while 500 mg of vitamin C was given for the entire 35 days.

Naziroglu et al. (2010) reported significantly greater levels of plasma vitamins A and E, as well as increased glutathione peroxidase (a vitamin E-dependent enzyme) in the supplemented group, compared to the non-supplemented group. Though supplementation improved the antioxidant defense system of these players, the researchers did not assess other health or performance measures.

The effect of free radical production has not been studied in adolescent athletes. Thus, Carlsohn et al. (2010) examined the effect of exercise on the antioxidant status in 90 male and female adolescent athletes compared to 18 controls (16 ± 2 years of age). This was not a supplementation study. The researchers measured antioxidant intake and blood levels of vitamin E (α-tocopherol). They reported that antioxidant intake was associated with energy intake and was within the recommended intake levels for vitamins C and E, as well as β-carotene. The antioxidant capacity was greater in athletes compared to controls. Carlsohn et al. (2010) stated that if adolescent athletes consume diets that provide the proper amount of energy and a variety of micronutrients, they will obtain the appropriate antioxidant levels (e.g., vitamins C, E, and β-carotene) to meet what is needed for the body to regulate free radical production. Furthermore, they reported that regular exercise increases blood antioxidant capacity in adolescent athletes, establishing that regular exercise and eating a varied diet can lead to better antioxidant capacity.

More recently Patlar et al. (2011) evaluated the relationship between exercise, vitamins, and mineral supplementation in seven elite male Turkish Taekwondo athletes (22 ± 1 years of age, 66.4 ± 2.4 kg body

weight), who had been practicing Taekwondo for at least 10 years. They were all supplemented with 300 mg/day of vitamin E for 6 weeks. The researchers reported significant increases ($p < 0.001$) in blood levels of cobalt, cadmium, chromium, nickel, manganese, copper, iron, zinc, phosphorus, sodium, potassium, and calcium. Supplementation resulted in significant increases of all elements relative to values before supplementation ($p < 0.001$), with the exception of boron and sulfur, which remained without change. Boron and sulfur were the only two minerals that were unchanged after supplementation. It appears that vitamin E supplementation in this group of young athletes influenced mineral levels in the blood, but once again the small sample size and the absence of a control group limit the interpretation of the findings. Furthermore, this is the same group of athletes presented earlier in the chapter, where vitamin A supplementation was studied. Thus, further caution must be taken, because it is not clear whether or not vitamins A and E were given together or over different periods of time. If the supplements were given at different times, no mention of a washout period was given in either publication (Patlar et al., 2010, 2011).

The B Vitamins

The eight B-complex vitamins are responsible for energy production, hemoglobin synthesis, immune function, and muscle development and repair (Woolf & Manore 2006). B vitamins that are potentially important for exercise include thiamin, riboflavin, niacin, vitamin B_6 (pyridoxine), pantothenic acid, and biotin. The other B vitamins, folate and B_{12} are important for erythrocyte and protein production and tissue repair (American Dietetic Association [ADA], 2009).

Thiamin (Vitamin B_1) Thiamin functions in the metabolism of carbohydrates, fats, and branched-chain amino acids (BCAA) (Woolf & Manore, 2006). For carbohydrate and fat metabolism, thiamine pyrophosphate (TPP), the active form of thiamin, is required in the pyruvate dehydrogenase complex to convert pyruvate to acetyl-CoA and in the α-ketoglutarate dehydrogenase complex in the Krebs cycle (Sato et al., 2011). Finally, thiamin is used to breakdown BCAA by the decarboxylase enzyme. Food sources of thiamin are listed in Table 18.1.

Because of the high demands of ultra-endurance training on vitamin and mineral loss through sweat loss, Knez and Peake (2010) examined the vitamin and mineral intake of 37 triathletes (24 men, 13 women) in Qatar via a 7-day dietary record. Based on this dietary record, they found that the men and women triathletes met or exceeded recommendations for all nutrients except vitamin D. Female athletes consumed somewhat less than the recommended intakes for folate and potassium. More than 60% of the triathletes reported vitamin and mineral supplementation (98% took vitamin C; 78% took vitamin E, 52% took multi-vitamins). Male triathletes reported higher thiamin intakes than women, but both men and women exceeded the recommended thiamin intake. Almost half of the athletes (48%) used supplements to decrease symptoms for colds. Only one athlete was taking a supplement on medical advice; the rest of the athletes consumed the supplements, despite no documented deficiency.

Sato et al. (2011) examined the blood concentrations of thiamin and riboflavin in 19 Japanese collegiate swimmers (6 men and 13 women) during low- and high-intensity training periods. Among other measures, they also evaluated 3-day dietary records during these times. As expected energy expenditure during the high-intensity training was significantly greater than during the low-intensity period. Blood thiamin levels decreased during the high-intensity training compared to the low-intensity training (41 ± 6 ng/ml to 36 ± 3 ng/ml in males ($p = 0.048$) and 38 ± 10 ng/ml to 31 ± 5 ng/ml in females ($p = 0.004$)). Blood levels of riboflavin remained the same. Thus, although dietary intakes may be within adequate intakes, blood levels may not reflect that during high-intensity training.

Riboflavin (Vitamin B_2) Riboflavin is a constituent of coenzymes flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD) (IOM, 2000). These coenzymes are involved in

reduction–oxidation reactions, the transferring of elections, for energy metabolism, amino acid metabolism, and hormone production (IOM, 2000). Riboflavin is important in exercise because the transfer of elections is needed to produce adenosine triphosphate (ATP) in the election transport chain (Woolf & Manore, 2006). Food sources of riboflavin are listed in Table 18.1.

Blood concentrations of riboflavin did not significantly decrease after intensive exercise in college swimmers (Sato et al., 2011). Riboflavin intake was within recommended levels in a group of elite Spanish adolescent soccer players (Garrido et al., 2007). Researchers have not reported riboflavin deficiency among athletes (Woolf & Manore, 2006). This may be a result of riboflavin reabsorption by the kidneys when riboflavin levels decrease in the blood (Sato et al., 2011).

Vitamin B_6 Vitamin B_6 is the generic term for six compounds (vitamers) with vitamin B_6 activity: pyridoxine, an alcohol; pyridoxal, an aldehyde; and pyridoxamine, which contains an amino group; and their respective 5′-phosphate esters. Pyridoxal-5′-phosphate (PLP) and pyridoxamine-5′-phosphate (PMP) are the active coenzyme forms of vitamin B_6 (IOM, 1998). Vitamin B_6 is involved in more than 100 enzyme reactions, with its primary function in protein metabolism, amino acid metabolism, and one-carbon transfers (IOM, 1998). Food sources of vitamin B_6 are listed in Table 18.1.

Choi and Cho (2009) examined the effect of vitamin B_6 deficiency on the antioxidant defense system in 48 rats that had exercised-induced oxidative stress. Rats were fed either a vitamin B_6-deficient diet or a control diet for 4 weeks. Lower plasma catalase and superoxide dismutase activity were found in the deficient group compared to the control group. They also reported higher plasma malondialdehyde concentrations and lower serum high density lipoprotein-cholesterol concentrations in the vitamin B_6-deficient group compared to the control group. Choi and Cho (2009) speculated that the lower antioxidant capacity may have been a result of the vitamin B_6 deficiency and further provoked by the stress of exercise (greater free radical production).

Folate Folate is a B-vitamin that occurs in foods, whereas folic acid is the synthetic form of folate found in supplements and is the form added to foods that have been fortified. Table 18.1 lists food sources of folate.

Folate plays many important roles in the body, including cell production and maintenance, especially important during pregnancy (fetus) and growth. Folate is required for deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) synthesis. Normal red blood cell production requires folate; if folate is not consumed at required levels, a megaloblastic anemia can result. Folate is also crucial for the metabolism and maintenance of normal levels of homocysteine: high levels of this amino acid have been linked with cardiovascular disease (Rousseau et al., 2005).

Because of evidence of acute cardiovascular events in marathon runners, Real et al. (2005) evaluated the changes in plasma homocysteine concentrations in 22 non-professional male runners the day before and 24 hours after finishing a 42.2-km marathon race. The men were 36 (range: 23–49) years of age. None of the athletes supplemented with vitamin B_6, vitamin B_{12}, or folate. There were no changes in plasma folate or plasma vitamin B_{12} concentrations post-marathon; however, there was a 19% increase in plasma homocysteine levels after the marathon. Prior to the race, only 20% of the participants had plasma homocysteine levels >10 μmol/l (which is the level for ischemic heart disease), but after the race, 50% of the runners had plasma homocysteine levels above 10 μmol/l. Though these runners may not have a greater risk of post-race cardiovascular events, further investigation is warranted to assess whether this poses a real danger to these athletes or if the effects are transient.

Woolf and Manore (2006) note that training volume may be the determinant factor in whether or not there is a significant increase or decrease in plasma homocysteine concentrations. Nonetheless, Joubert and Manore (2008) found that plasma homocysteine concentrations were not different between highly physically active individuals and low physically active individuals.

Okura et al. (2006) examined whether 20 weeks of aerobic exercise could affect total homocysteine

levels in 711 men and women, aged 17–65 years. They found that, in African Americans males, total homocysteine concentrations did not change significantly with exercise training, but were significantly increased in Caucasian men. The researchers also reported that individuals who began the study with high levels of plasma homocysteine showed a significant reduction with exercise; however, those who began the study in the normal range, showed a slight increase in plasma homocysteine concentrations.

Vitamin C (Ascorbic Acid)

Vitamin C is important in a number of physiological functions including: collagen synthesis, iron absorption, carnitine formation, neurotransmitter synthesis, production of serotonin, antioxidant function, and adrenal hormone release (e.g., cortisol, aldosterone). In its antioxidant role, vitamin C increases glutathione levels (Naziroglu et al., 2010) to prevent oxygen radicals from attacking cell structures (Gauche et al., 2006). See Table 18.1 for food sources of vitamin C.

Tian et al. (2009) assessed supplement use among 82 athletes from 16 different sports who attended a university in Singapore. They reported a supplement use of 77%, with 20 different products listed among the athletes surveyed. Each athlete consumed about three to four different products over a 1-year period. The most popular products included: sports drinks, vitamin C, multi-vitamins, and traditional/herbal preparations (e.g., ginseng). Prior to supplement use, the athletes did try to obtain more information; however, they did not go to nutrition professionals, but rather obtained their information from the media, the Internet, their coaches, and fellow athletes. Most athletes did not know where they could find reliable information and 86% were not aware that supplement use could have dangerous side effects.

Minerals

Chromium

Chromium in the form of the protein, chromodulin, can potentiate the effects of insulin (IOM, 2002; Lukaski, 2000). Food sources of chromium are listed in Table 18.2. Increases in lean body mass and decreases in fat mass were observed in a group of men who received chromium picolinate supplementation, with no improvement in athletic performance (Lukaski, 2000).

Volpe et al. (2001) examined the effect of chromium picolinate supplementation on body composition, resting metabolic rate (RMR), various biochemical factors, and blood iron and zinc concentrations in 44 moderately obese women, 27–51 years of age, participating in a 12-week supervised walking and weight training program. The women were randomly assigned, in a double-blind fashion, to either the chromium-supplemented group (400 μg/day of chromium) or an identical-looking placebo. Body composition and RMR were measured at baseline, 6 and 12 weeks, while the blood and urinary measures were conducted at baseline and 12 weeks only. There were no significant differences as a result of the chromium supplementation.

More recently, Yazaki et al. conducted a 24-week randomized, double-blind, placebo controlled study to evaluate the effects of 1000 μg/day of chromium picolinate supplementation in 80 healthy overweight adults. All participants received nutrition

Table 18.2 Example of food sources for minerals presented

Mineral	Examples of good food sources
Chromium	Meats Poultry Gish Some cereals
Iron	Beef Fish Eggs Lentils Beans Fortified cereals
Zinc	Oysters Red meats Fortified cereals

Source: Adapted from http://www.webmd.com/a-to-z-guides/vitamins-and-minerals-good-food-sources (accessed June 17, 2012).

education at 12 weeks. The researchers used computed tomography to assess central adiposity. Similar to Volpe et al. (2001), Yazaki et al. (2010) did not report significant differences between groups in weight loss or central adiposity measures.

Volek et al. (2006) assessed the effects of 600 μg/day of chromium picolinate in 16 overweight men (average BMI = 31 ± 3 kg/m^2) who were randomly assigned to either the chromium group or a placebo for 4 weeks. After the supplementation period, the participants performed a supramaximal bout of cycling exercise to exhaust their stores of muscle glycogen. Following a supramaximal exercise bout, participants were provided high glycemic index carbohydrates for the following 24 hours. Muscle biopsies were taken at rest, immediately post-exercise, and at 2 and 24 hours post-exercise. Volek et al. (2006) found no significant differences in glucose or insulin concentrations between the groups, concluding that 4 weeks of chromium supplementation did not improve glycogen synthesis during recovery after high-intensity exercise followed by a high glycemic index feeding. Based on the aforementioned studies, it appears that chromium is not effective as an ergogenic aid for weight loss or to promote muscle glycogen storage.

Iron

Iron plays a vital role in exercise because of its function in transporting oxygen to peripheral tissues. Iron is a structural component of with hemoglobin, myoglobin, and cytochromes and confers their functional properties to bind oxygen (Williams, 2005). Iron is dependent on copper to convert ferrous into ferric ions. Low copper levels have been related to iron deficiency anemia (Speich et al., 2001). Food sources of iron are listed in Table 18.2.

Although iron deficiency anemia is often discussed and researched, iron overload could potentially be a problem in male athletes, especially because men typically consume a significant larger amount of energy than women. Mettler and Zimmermann (2010) evaluated the iron status of 170 male and female recreational runners who ran the Zurich marathon. Iron deficiency was defined either as plasma ferritin concentrations <15 μg/l (iron depletion) or as the ratio of the concentration of transferrin receptor (sTfR) to plasma ferritin (sTfR:log(Plasma ferritin) index) of ≥4.5 (known as functional iron deficiency).

Mettler and Zimmermann (2010) reported iron depletion in 2 of 127 men studied and in 12 of 43 women studied; whereas functional iron deficiency was found in 5 of 127 men and 11 of 43 women. They reported that body iron stores were significantly greater ($p < 0.001$) in men than in women runners. The median plasma ferritin concentration in men was 104 μg/l, with an upper limit reported as 628 μg/l. Iron overload was found in 19 of 127 men and 2 of 43 women. Their finding of a greater number of male recreational runners with iron overload is important because so many athletes supplement, without regard to their vitamin or mineral status. Supplementation should be used only if individuals are tested for iron deficiency anemia.

Studies from 2002 and 2009 of Finnish athletes showed that iron supplements were the most commonly reported mineral supplement used (Heikkenen et al., 2011). Tian et al. (2009) noted that iron supplements were the second least used supplement in Singapore university athletes. Iron intake for males were reportedly higher than females in Greek aquatic athletes (Farajian et al., 2004) and Flemish adolescent sprint athletes (Aerenhouts et al., 2008); however, iron supplementation was more prevalent among female Finnish athletes (Heikkenen et al., 2011).

Zinc

Zinc is involved in over 200 enzymatic processes in the body, many involved in energy metabolism and antioxidant functions (Lukaski, 2000; Volpe, 2008). Good food sources of zinc are listed in Table 18.2.

Plasma zinc concentrations have been shown to decrease with acute stress. This decrease is thought to be related to an increased uptake of zinc by the liver and bone marrow for synthesis of acute phase proteins. Volpe et al. (2007) assessed short-term changes in zinc kinetics, using the stable isotope Zn^{70}, to define the kinetics of zinc metabolism following an acute, strenuous bout of exercise in sedentary men. They performed a cross-over design study in 12 healthy, sedentary men, 25–35 years of

age. Zn^{70} was infused 10 minutes after an exhaustive cycling exercise bout or at rest. Plasma zinc levels significantly decreased after exercise, with a mean decrease of 14% ± 4% observed at 70 minutes post-exercise. Volpe et al. (2007) reported increases in the size of the rapidly exchangeable plasma zinc pool and the liver zinc pool, signifying a shift of plasma zinc into the interstitial fluid and liver after exercise, possibly paralleling the acute stress response of strenuous exercise.

One of zinc's many functions includes its role in the conversion of thyroxine (T_4) to the more active triiodothyronine (T_3). T_3 is involved with the body's overall metabolism and therefore could affect exercise performance and health. In a case study, Maxwell and Volpe (2007) examined the effects of zinc supplementation on plasma zinc, serum ferritin, plasma T_3 and T_4, serum free T_3 and T_4, and thyroid-stimulating hormone (TSH) levels, and RMR in two zinc-deficient, physically active women, who were supplemented with 26.4 mg/day of zinc (as zinc gluconate) for 4 months. They found that the zinc deficiency was clinically corrected in both subjects, while serum ferritin levels declined, resulting in subjects being identified as borderline iron deficient. At 4 months, total T_3 concentrations increased in one participant, while all thyroid hormone levels increased in the other participant. RMR increased in both participants at 4 months. As this was a case study, statistical analyses could not be performed, but zinc supplementation appeared to be responsible for the increase in plasma zinc and decline in serum ferritin levels in both participants and appeared to have a positive effect on total T_3 concentrations and RMR.

de Oliveira et al. (2009) supplemented 21 Brazilian male football players with 22 mg/day of zinc as zinc gluconate over a 12-week period. These athletes were compared with 26 football players given a placebo for the same time period. After 12 weeks of supplementation, plasma zinc and erythrocyte iron increased in both groups ($p < 0.001$); however, urinary zinc increased significantly in the zinc-supplemented group, while erythrocyte zinc significantly decreased in the placebo group. Plasma iron and copper significantly decreased in the zinc-supplemented group. Antioxidant markers were improved in the zinc-supplemented group. Thus, short-term zinc supplementation in adolescent athletes impaired iron and copper status, but improved antioxidant capacity. More research needs to be conducted in this age group to assess the risk–benefit ratio of supplementation.

Summary

The research conducted on vitamins and minerals in athletes of all ages and from many different countries, has shown equivocal results, with the majority of researchers reporting no effect of vitamin and/or mineral supplementation performance. In general, if a positive effect was found, it typically was not directly related to performance. Furthermore, most of the studies conducted in vitamin and mineral supplementation had small sample sizes, and some did not have control groups. In addition, with varying study durations and vitamin and mineral supplement dosages, it is difficult to compare studies. Given the widespread use of vitamin and mineral supplements in most athletic populations, further research is needed, alongside an education program for athletes and those who advise them.

References

Aerenhouts, D., Hebbelinck, M., Poortmans, J.R., et al. (2008) Nutritional habits of Flemish adolescent sprint athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 509–523.

American Dietetic Association [ADA] (2009) Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *Journal of the American Dietetic Association* 109, 509–527.

Barrack, M.T., Van Loan, M.D., Rauh, M.J., et al. (2010) Physiologic and behavioral indicators of energy deficiency in female adolescent runners with elevated bone turnover. *American Journal of Clinical Nutrition* 92, 652–659.

Bartoszewska, M., Kamboj, M., & Patel, D.R. (2010) Vitamin D, muscle function, and exercise performance. *Pediatric Clinics of North America* 57, 849–861.

Bescos Garcia, R. & Rodriguez Guisado, F.A. (2011) Low levels of vitamin D in professional basketball players after

wintertime: relationship with dietary intake of vitamin D and calcium. *Nutricion Hospitalaria* 26, 945–951.

Braun, H., Koehler, K., Geyer, H., et al. (2009) Dietary supplement use among elite young German athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 97–109.

Carlsohn, A., Rohn, S., Mayer, F., et al. (2010) Physical activity, antioxidant status, and protein modification in adolescent athletes. *Medicine & Science in Sports & Exercise* 42, 1131–1139.

Choi, E.Y., Cho, Y.O. (2009) Effect of vitamin B(6) deficiency on antioxidative status in rats with exercise-induced oxidative stress. *Nutrition Research and Practice* 3(3), 208–211.

Dascombe, B.J., Karunaratna, M., Cartoon, J., et al. (2010) Nutritional supplementation habits and perceptions of elite athletes within a state-based sporting institution. *Journal of Science and Medicine in Sport* 13, 274–280.

de Oliveira, K.J.F., Donangelo, C.M., de Oliveira Jr., A.V., et al. (2009) Effect of zinc supplementation on the antioxidant, copper, and iron status of physically active adolescents. *Cell Biochemistry and Function* 27, 162–166.

Evans, W.J. (2000) Vitamin E, vitamin C, and exercise. *American Journal of Clinical Nutrition* 72, 647S–652S.

Farajian, P., Kavouras, S.A., Yannakoulia, M., et al. (2004) Dietary intake and nutritional practices of elite Greek aquatic athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 14, 574–585.

Gabel, K.A. (2006) Special nutritional concerns for the female athlete. *Current Sports Medicine Reports* 5, 187–191.

Garrido, G., Webster, A.L., & Chamorro, M. (2007) Nutritional adequacy of different menu settings in elite Spanish adolescent soccer players. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 421–432.

Gauche, E, Lepers, R., Rabita, G., et al. (2006) Vitamin and mineral supplementation and neuromuscular recovery after a running race. *Medicine & Science in Sports & Exercise* 38, 2110–2117.

Heikkinen, A., Alaranta, A., Helenius, I., et al. (2011) Dietary supplementation habits and perceptions of supplement use among elite Finnish athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 271–279.

Holick, M.F. (2006) Resurrection of vitamin D deficiency and rickets. *Journal of Clinical Investigation* 116, 2062–2072.

Houston, D.K., Tooze, J.A., Hausman, D.B., et al. (2011) Change in 25-hydroxyvitamin D and physical performance in older adults. *Journals of Gerontology. Series A, Biological Sciences and Medical Sciences* 66(4), 430–436.

Institute of Medicine [IOM]. Food and Nutrition Board (1998) *Dietary Reference Intakes: Thiamin, Riboflavin, Niacin, Vitamin B_6, Folate, Vitamin B_{12}, Pantothenic Acid, Biotin, and Choline*. National Academies Press, Washington, DC.

Institute of Medicine [IOM]. Food and Nutrition Board (2000) *Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and Carotenoids*. National Academies Press, Washington, DC.

Institute of Medicine [IOM]. Food and Nutrition Board (2002) *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc*. National Academies Press, Washington, DC.

Joubert, L.M. & Manore, M.M. (2008) The role of physical activity level and B-vitamin status on blood homocysteine levels. *Medicine & Science in Sports & Exercise* 40, 1923–1931.

Knechtle, B., Knechtle, P., Schulze, I., et al. (2008) Vitamins, minerals and race performance in ultra-endurance runners—Deutschlandlauf 2006. *Asia Pacific Journal of Clinical Nutrition* 17, 194–198.

Knez, W.L. & Peake, J.M. (2010) The prevalence of vitamin supplementation in ultraendurance triathletes. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 507–514.

Louis, J., Hausswirth, C., Bieuzen, F., et al. (2010) Vitamin and mineral supplementation effect on muscular activity and cycling efficiency in master athletes. *Applied Physiology, Nutrition and Metabolism* 35, 251–260.

Lovell, G. (2008) Vitamin D status of females in an elite gymnastics program. *Clinical Journal of Sports Medicine* 18, 159–161.

Lukaski, H.C. (2000) Magnesium, zinc, chromium nutriture and physical activity. *American Journal of Clinical Nutrition* 72, 585S–593S.

Martinez, S., Pasquarelli, B.N., Romaguera, D., et al. (2011) Anthropometric characteristics and nutritional profile of young amateur swimmers. *Journal of Strength and Conditioning Research* 25, 1126–1133.

Maxwell, C. & Volpe, S.L. (2007) Effect of zinc supplementation on thyroid hormone function. A case study of two college females. *Annals of Nutrition and Metabolism* 51(2), 188–194.

Mettler, S. & Zimmermann M.B. (2010) Iron excess in recreational marathon runners. *European Journal of Clinical Nutrition* 64, 490–494.

Naziroglu, M., Kilinc, F., Uguz, A.C., et al. (2010) Oral vitamin C and E combination modulates blood lipid peroxidation and antioxidant vitamin levels in maximal exercising basketball players. *Cell Biochemistry and Function* 28, 300–305.

Okura, T., Rankinen, T., Gagnon J., et al. (2006) Effects of regular exercise on homocysteine concentrations: the heritage family study. *European Journal of Applied Physiology* 98, 394–401.

Patlar, S., Boyali, E., Baltaci, A.K., et al. (2010) The effect of vitamin A supplementation on various elements in elite taekwondo players. *Biological Trace Element Research* 139, 296–300.

Patlar S, Boyali E, Baltaci AK, et al. (2011) Elements in sera of elite taekwondo athletes: effects of vitamin E supplementation. *Biological Trace Element Research* 139(2), 119–125.

Real, J.T., Merchante, A., Gomez, J.L., et al. (2005). Effects of marathon running on plasma total homocysteine concentrations. *Nutrition, Metabolism, and Cardiovascular Diseases* 15, 134–139.

Rousseau, A.S., Robin, S., Roussel, A.M., et al. (2005) Plasma homocysteine is related to folate intake but not training status. *Nutrition, Metabolism, and Cardiovascular Diseases* 15, 125–133.

Sato, A., Shimoyama, Y., Ishikawa, T., et al. (2011) Dietary thiamin and riboflavin intake and blood thiamin and riboflavin concentrations in college swimmers undergoing intensive training. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 195–204.

de Silva, A., Samarasinghe, Y., Senanayake, D., et al. (2010) Dietary supplement intake in national-level Sri Lankan athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 15–20.

Speich, M., Pineau, A., & Ballereau, F. (2001) Minerals, trace elements and related biological variables in athletes and during physical activity. *Clinica Chimica Acta* 312, 1–11.

Tian, H.H., Ong, W.S., & Tan, C.L. (2009) Nutritional supplement use among university athletes in Singapore. *Singapore Medical Journal* 50, 165–172.

Volek, J.S., Silvestre, R., Kirwan, J.P., et al. (2006) Effects of chromium supplementation on glycogen synthesis after high-intensity exercise. *Medicine & Science in Sports & Exercise* 38(12), 2102–2109.

Volpe, S.L. (2008) Minerals (chromium, magnesium, zinc, selenium) as an ergogenic aid. *Current Sports Medicine Reports*, 7(4), 224–229.

Volpe, S.L., Huang, H.W., Larpadisorn, K., et al. (2001) Effect of chromium supplementation and exercise on body composition, resting metabolic rate and selected biochemical parameters in moderately obese women following an exercise program. *Journal of the American College of Nutrition* 20(4), 293–306.

Volpe, S.L., Lowe N.M., Woodhouse, L.R., et al. (2007) Effect of maximal exercise on the short-term kinetics of zinc metabolism in sedentary men. *British Journal of Sports Medicine* 41(3), 156–161.

Wienecke, E. & Gruenwald, J. (2007). Nutritional supplementation: is it for everybody? *Advances in Natural Therapy* 24, 1126–1135.

Williams, M.H. (2005) Dietary supplements and sports performance: minerals. *Journal of the International Society of Sports Nutrition* 2, 43–49.

Woolf, K. & Manore, M.M. (2006) B-vitamins and exercise: does exercise alter requirements? *International Journal of Sport Nutrition and Exercise Metabolism* 16, 453–484.

Yazaki, Y., Faridi, Z., Ma, Y., et al. (2010) A pilot study of chromium picolinate for weight loss. *Journal of Alternative and Complementary Medicine* 16(3), 291–299.

Chapter 19

Iron Requirements and Iron Status of Athletes

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The Physiology of Iron

Virtually all eukaryotic cells use iron as a cofactor for fundamental biochemical activities, such as energy metabolism and DNA synthesis, and these reactions are all based on its flexible coordination chemistry, that allows it to associate with the effectors proteins, and its redox reactivity (Aisen et al., 2001). Under anaerobic conditions, however, iron is potentially toxic, since it catalyses the production of reactive oxygen species (ROS) which generate the highly reactive radicals that can damage cellular macromolecules and in turn induce tissue injury and disease (Galaris & Pantopoulos, 2008; Kell, 2009).

About half of the total body iron (i.e., at least 2.1 g in humans) forms part of the structure of the oxygen-transporting hemoglobin (Hb) of mature red blood cells (RBC) and developing erythroid cells. Significant amounts of iron are also present in macrophages (up to 600 mg), deriving from senescent RBC sequestration, and in the oxygen-transporting myoglobin of muscles (~300 mg), whereas excess body iron (about 1 g) is stored in the liver (Andrews, 1999; Olsson & Norrby, 2008). Other tissues contain lower, but not negligible, amounts of iron.

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Iron is the functional component of Hb and myoglobin, playing a pivotal role in oxygen delivery. This metal is also a key structural and functional component of the heme moiety of mitochondrial enzymes and cytochromes involved in the electron transport chain and in oxidative phosphorylation (Peeling et al., 2008). It is thus clear that a functional iron deficiency is associated with a decline in wellness and in athletic performance (Wilkinson et al., 2002). Accordingly, iron deficiency, which may result from many different causes, is a common concern of athletes especially for those involved in endurance sports (Beard & Tobin, 2000; Zoller & Vogel, 2004).

A number of exercise-generated mechanisms are proposed to influence the iron status of an athlete including hemolysis, hematuria, sweating, gastrointestinal bleeding, and injuries (Zoller & Vogel, 2004). Besides the well-known variation of iron status throughout the different phases of training and during the stressful competition season, the important effect of exercise-dependent fluctuations in the level of cytokines and hormones requires further scrutiny to better explain the molecular mechanism involved in this challenging issue (Peeling et al., 2008).

Iron deficiency is the most common single nutrient deficiency disease worldwide, with an estimated prevalence of ~15% of the population worldwide (Beard & Tobin, 2000).

The prevalence of iron-deficiency anemia in Europe and North America is reported to be very low if com-

pared to less developed parts of the world: in Europe only 1% of adult males and 14% of adult females had anemia, compared to 27% and 48% in males and females in Africa and 40% and 57%, respectively, in South Asia (Marx, 1997). More recently it was reported that the prevalence of iron-deficient anemia is estimated to be 3–5% in women and 1% in men in the United States, while iron deficiency without anemia is more common, affecting 12–16% of premenopausal adult women and 2% of adult men (Sinclair & Hinton, 2005).

Among athletes, the prevalence of iron depletion without anemia seems to be significantly higher than among the general population: in adolescent and adult elite female athletes, competing in various sport disciplines, the prevalence is 25–35%. Iron deficiency is less common among male young and adult elite athletes but varies widely with sport affiliation: 15% of male basketball players, 11% of males competing in a variety of sports, and 36% of elite gymnasts (Sinclair & Hinton, 2005).

Generally, iron deficiency can be defined as depletion of body's iron stores and restriction of iron supply to various tissues. Clearly, iron depletion leads to a reduction in the oxygen transport capacity, as well as in the oxidative capacity at the cellular level. The rate of depletion of iron stores mainly depends upon the fine balance between loss and intake (Beard & Tobin, 2000).

Iron deficiency is a common feature of adolescent girls, since the menstrual iron losses are combined with the rapid growth to increase the iron consumption rate. Each milliliter of blood lost contains nearly 0.5 mg of iron. Abundant menses (i.e., more than 80 ml in as much as 10% of females) can trigger or amplify iron deficiency. Other important risk factors for young women include pregnancy, high parity, intrauterine devices, and a vegetarian diet. The leading factors affecting iron status in the general population include gastrointestinal bleeding (e.g., intestinal parasites), gastric achlorhydria, celiac disease, and *Helicobacter pylori* infection (Zimmermann & Hurrell, 2007).

Intestinal Absorption, Body Iron Recycling, and Delivery to Target Tissues

Iron losses occur moderately, but continuously, as a result of mucosal and skin desquamation, but might generally increase with bleeding and mainly with the female menstrual cycle. No regulation of iron excretion seems to exist.

Iron balance is mainly regulated by duodenal absorption. In the intestinal lumen the dietary Fe^{3+} is reduced to Fe^{2+} by the activity of ferric reductases and is further transported across the apical membrane of the enterocyte through the aspecific transporter DMT1 (divalent metal transporter 1, also known as SCLA11A2). Dietary heme is also transported across the enterocytic brush border, by an unknown carrier, where it is metabolized by heme oxygenase-1 (HO-1) to free Fe^{2+}. The efflux of Fe^{2+} from the enterocyte toward the bloodstream, throughout the basolateral labyrinth, is mediated by the membrane-bound complex ferroportin (SCLA11A3)–hephaestin and is coupled to its reoxidation to Fe^{3+} mediated by the soluble Cu^{2+}-dependent ferroxidase ceruloplasmin (Yeh et al., 2009).

Fe^{3+} is present in plasma mainly bound to its specific transporter transferrin (Tf) and further delivered to the tissues. Only 0.1% (3 mg) of the total body iron is bound to Tf, and the Tf iron pool is mainly sustained by the recycling iron recovered from senescent RBC and by newly absorbed dietary iron to a lesser extent (Wang & Pantopoulos, 2011). Each Tf molecule transports two atoms of iron (Banfi, 2005).

Senescent RBC are cleared by the reticuloendothelial macrophages. After heme degradation, iron is released in plasma via ferroportin, coupled with the ceruloplasmin-mediated reoxidation of Fe^{2+} to Fe^{3+}, and bound to Tf (Wang & Pantopoulos, 2011).

All iron-exporting cells (i.e., enterocytes, macrophages, hepatocytes) express ferroportin, which is a necessary protein for iron release in the bloodstream, as well as in the iron transfer to the fetus. Deletion of ferroportin in mice embryos is lethal, supporting its essential role in life, whilst its conditional inactivation causes iron retention in iron-exporting cells (Donovan et al., 2005).

Rather understandably, iron must be present in the blood stream in a redox-inactive and nontoxic form, a condition satisfied by the binding of the metal with Tf, which maintains its solubility and thus prevents its participation in the Fenton and Haber–Weiss reactions. Diferric-Tf is captured by

specific membrane receptors (TfR) of the target cells. The type 1 is expressed by cells of the erythroid lineage, hepatocytes, monocytes, and blood–brain barrier, whereas the type 2 is liver-specific (Gomme et al., 2005). The complex $2Fe^{3+}$-Tf-TfR is endocytosed and the fall of pH in late endosomes induces iron release, so that apotransferrin loses the affinity for TfR and is degraded in lysosomes, while TfR is recycled in the plasma membrane (Banfi, 2005). Iron passes into the cytoplasm, through a process mediated by the endosomal DMT1, where it is incorporated into iron-containing proteins. Conversely, excess iron is incorporated in stored form, bound to ferritin (Peeling et al., 2008).

Regulation of Ferroportin-Mediated Iron Extrusion: The Role of Hepcidin

Ferroportin-mediated Fe^{2+} efflux is negatively regulated by hepcidin. Hepcidin, a peptide hormone produced by the hepatocytes, binds with ferroportin and promotes its phosphorylation, thereby allowing internalization and lysosomal degradation (Hentze et al., 2010; Nemeth & Ganz, 2009). After dietary iron intake or during inflammatory conditions, the accumulation of hepcidin in the bloodstream decreases iron absorption by the gut and reduces retention from macrophages. This protective mechanism is responsible for iron depletion during infections, with a resulting inhibition of bacterial growth.

In iron deficiency, hypoxia, or anemia, a drop of hepcidin levels promotes intestinal absorption and release of iron from macrophages. The disruption of this finely tuned hepcidin-mediated control is associated with metabolic iron overload and hemochromatosis (Lee & Beutler, 2009), whereas increased hepcidin levels are associated with conditions of iron deficiency (Weiss & Goodnough, 2005). High hepcidin levels due to genetic predisposition account for iron-refractory iron-deficiency anemia (IRIDA) development (Finberg, 2009).

Hepcidin expression is chiefly modulated at the transcriptional level. The basal expression is under control of the C/EBPα (CCAAT/enhancer binding protein α) transcription factor (Courselaud et al., 2002). Enhanced expression of hepcidin can conversely be induced by iron. In fact, iron induces the expression of BMP6 (bone morphogenetic protein 6) in both the liver and the intestine (Meynard et al., 2009), which further binds its specific receptor on the hepatocyte's surface. The triggered cascade induces the phosphorylation of SMAD1/5/8 and the translocation of SMAD4 into the nucleus where it associates with the hepcidin gene promoter (Andriopoulos et al., 2009; Meynard et al., 2009; Wang et al., 2005). HFE (hemochromatosis protein), TfR2 (transferrin receptor-2), and HJV (hemojuvelin, a BMP co-receptor) are involved in this signaling, but their role still needs to be elucidated, although evidence has been provided that mutations in these genes impair hepcidin expression and they are associated to hemochromatosis (Lee & Beutler, 2009). HJV is capable of inducing hepcidin, but it is degraded by the membrane-resident serine protease matriptase-6 (TMPRSS-6). Inactivating mutations in TMPRSS-6 are associated with IRIDA (Finberg, 2009).

A variety of pathways induce the hepcidin expression during inflammation. Interleukin-6 (IL-6) induces the phosphorylation of STAT3 (signal transducer and activator of transcription 3) that translocates in the nucleus where it binds to the hepcidin promoter inducing its transcription. Additional mechanisms involve IL-1β (which acts through C/EBPα and the BMP/SMAD signaling), endoplasmic reticulum stresses (which is mediated by CREBH (cAMP-responsive element-binding protein H)), and CHOP (C/EBPα homologous protein). Lipopolysaccharide (LPS) also participates in an autocrine fashion on macrophages through TLR4 (toll-like receptor 4), whereas specific pathogens act through other TLRs (Wang & Pantopoulos, 2011).

Hepcidin expression is instead inhibited in anemia. In thalassemia, the suppression is mediated by GDF15 (growth differentiation factor 15) (Wang & Pantopoulos, 2011), whilst erythropoietin (Epo) downregulates it through the induction of a decrease in C/EBPα binding to hepcidin promoter (Pinto et al., 2008).

The role of hypoxia and oxidative stresses in hepcidin suppression is also not well understood. In hypoxia a certain role is played by HIFs, whereas oxidative stress promotes the expression of the

histone deacetylases (HDACs) that inhibit the C/EBPα and STAT3 binding to the hepcidin promoter (Wang & Pantopoulos, 2011).

Regulation of Body Iron Stores

Along with the iron continuously exchanged between the different active stores, there is an amount of metabolically inactive iron stored with ferritin that might be used in case of functional needs. Ferritin is a complex globular protein composed of 24 amino acid chains, which is able to store up to 400,000 atoms of iron in its core. The amino acid chains form eight channels that allow the traffic of the iron ions. There are different isoforms of ferritin, mainly differing in the combination of the amino acid chains. The isoferritins enriched in L chains are typical of tissues characterized by long-term iron storage, whereas isoferritins enriched in H chains are represented more in tissues with rapid exchange, such as the bone marrow. The H chains are also essential, since they retain the catalytic activity responsible for the oxidation of Fe^{2+} to Fe^{3+}, which is the storage form (Banfi, 2005).

Ferritin biosynthesis is modulated by the intracellular iron concentration. There is a direct relationship between the free iron in the cell and the degree of translation of the mRNA encoding for the H and L chains of ferritin. When available within the cell at high concentrations, iron binds and inactivates the iron-regulation protein (IRP). However, when the iron concentration decreases, the metal is released and the IRP binds to the iron-responsive elements (IRE) on the ferritin chains, thereby inhibiting their translation. The mechanism IRP–IRE also regulates the translation of the TfR mRNA, but with an opposite activity, so that higher intracellular iron activates the IRP that binds the IRE on TfR mRNA, thus inhibiting its degradation. This system is able to effectively modulate iron homeostasis, preventing the intracellular iron concentration from reaching toxic levels, being also actively involved in the control of a variety of iron-dependent proteins (Banfi, 2005).

Several iron-independent stimuli are able to regulate the production of ferritin hormones, cytokines, and oxidative species (Peeling et al., 2008). Chronic and acute inflammation induce increases in ferritin levels, which is hence considered an acute phase protein. A decrease of its concentration in serum is typically associated with iron deficiency and thereby represents the biochemical gold standard for the diagnosis of iron-deficiency anemia (Banfi, 2005).

Iron in Sport

Iron deficiency induces clear morphological, biochemical, and physiological changes in several organs, mainly depending upon the turnover rate of the iron-containing proteins and often anticipating the clinical evidence of anemia. Indeed, iron is also involved in mitochondrial activities, synthesis of neurotransmitters and proteins, and organogenesis (Dallman, 1986).

Chronic deficiency is clinically characterized by glossitis, angular stomatitis, koilonychias, blue sclera, Plummer–Vinson syndrome (esophageal webbing), and anemia as well as by behavioral disorders for which a biological explanation is still lacking.

A decrease of Hb, especially when associated with low tissue iron stores, is detrimental for physical performance, especially considering that iron is lowered by chronic exercise and that women are more prone to exercise-related alterations in body iron status. Low iron is ultimately associated with a reduced delivery of oxygen to the muscles. VO_2 max is strongly influenced by the oxygen-carrying capacity of blood and, therefore, by the concentration of hemoglobin and the degree of anemia. However, when considering endurance exercise, performance is more related to the ability to maintain a prolonged activity of iron-dependent oxidases, and therefore more dependent upon the content of iron in the bodily tissues (Beard & Tobin, 2000).

The combination of a low RBC count, low Hb, and decreased tissue myoglobin always results in impaired oxygen diffusion except at rest and despite the increased cardiac output, low peripheral partial oxygen pressure (P_{O_2}), and the right-shift of the Hb–O_2 dissociation curve. The net result is a variable

decline of muscle aerobic capacity. Experimental models demonstrate that iron-deficient muscle have up to a 35% reduction in pyruvate and malate oxidases and a 50–90% decrease in Fe-S enzymes and heme-containing cytochromes (Dallman, 1986).

A decreased serum ferritin in female runners as compared with sedentary matched controls has been repeatedly highlighted, with one-third of athletes displaying levels lower than 12 mg/l in some surveys. The functional consequences of this ferritin drop, in the absence of a frank anemia, are, however, unclear (Beard & Tobin, 2000).

Sports Anemia

The need to supplement athletes with iron was first suggested by the evidence of changes in blood cell counts and iron-associated parameters during periods of intense training or, even more accentuated, at the start of the training. This condition is known as "sports anemia" and is common in endurance athletes. This is generally a transient phenomenon and only 8% of elite athletes show frank anemia (Hb concentrations below the reference limits, i.e., 135 g/l in males and 120 g/l in females). The main mechanism underlying this condition is represented by the early training-dependent increase in plasma volume that stimulates erythropoiesis through hormonal (e.g., Epo) and osmotic responses, thus inducing an increase of the red cell mass (Zoller & Vogel, 2004) including mature RBC as well as erythroid immature forms (e.g., reticulocytes) (Corsetti et al., 2012; Diaz et al., 2011; Lombardi et al., 2013). However, while the plasma volume may increase by more than 20%, the red cell mass increase is typically between 10% and 18%, resulting in a relative decrease in hematocrit. From a physiological perspective, plasma expansion decreases blood viscosity and improves blood flow in large vessels, whereas the greater deformability of newly formed erythrocytes is responsible for an increased capillary flow (Zoller & Vogel, 2004).

The exercise-induced variations in several hematological parameters cannot be attributed exclusively to the hemodilution, since important morphological variations occur in the cells of the erythroid lineage. As an example, the well-known microcytosis of the endurance athlete is secondary to reactive reticulocytosis due to intravascular hemolysis. The Hb released is then captured by haptoglobin (Hp) to be directed toward hepatic uptake and metabolism and generating hypohaptoglobinemia. The extent of the haptoglobin decrement cannot be entirely explained by foot-strike hemolysis (the RBC destruction induced by the reiterated impact with the ground) that is commonplace in runners (Telford et al., 2003).

The hypohaptoglobinemia observed in swimmers, weight lifters, and rowers is in fact unlikely to be due to foot-strike hemolysis, because of the lack of contact with the ground, but hemolysis might occur due to the breakdown of erythrocytes in the exercising muscles. Additional factors that might influence iron status metabolism in these circumstances involve gastrointestinal bleeding, sweating, and hematuria (Peeling et al., 2008).

When Hb is increased through blood transfusions or administration of recombinant human Epo (rhEpo), improved endurance performance has been clearly shown in endurance athletes (Adamson & Vapnek, 1991). It can hence be hypothesized that the production of Hb is limited by the iron availability during exercise and, as a consequence, iron supplementation might improve performance.

There is no evidence that iron supplementation increases athletic performance, except in individuals in whom iron deficiency was established. However, in athletes with low serum ferritin concentrations without anemia, iron supplementation might be useful (Zoller & Vogel, 2004).

Iron and Iron-Related Parameter in Response to Exercise

The hematological changes that occur during intense training are paralleled by the fall in ferritin concentration, mirroring a depletion of the body iron stores, even though ferritin levels also depend upon a number of other factors such as liver disease, alcohol consumption, infections, cardiovascular disease, and aging (Hallberg & Hulthen, 2003). Other parameters that can be used to monitor the iron status include sTfR, RBC protoporphyrin, and stainable bone marrow iron (hemosiderin) (Cook, 1999).

The half-life of iron is 2100 and 1300 days in sedentary males and females, respectively, while it has been calculated to be decreased to 1000 days in runners (Ehn et al., 1980). A 45-minute run at 70% VO_2 max was found to induce a reduction in ferritin but not in sTfR, whose levels decreased only following incremental exercise. Considering the extracellular fluid shifts (hemoconcentration during the exhaustive test, namely the incremental exercise, and hemodilution during the aerobic test), the investigators concluded that sTfR more reliably reflects exercise-induced changes in iron metabolism than does serum ferritin, which is also influenced by factors other than iron stores (Schumacher et al., 2002). Elite athletes usually display low ferritin levels, although real iron deficiency might not be really present. Indeed, gastrointestinal bleeding might be frequent in the endurance athletes during intense training period, with losses of up to 6.6 ml/day (Nachtigall et al., 1996). These bleedings are mainly consequences of the reduced blood flow to the gastrointestinal tract (up to 56%) at the favor of muscles and skin as a consequence of the increased sympathetic activity. The enterocytes, deprived of oxygen and nutritional factors, might undergo severe injury up to necrosis, leading to the onset of gastrointestinal hemorrhages. Trauma or preexistent gastrointestinal lesions can also be considered important causes of blood losses (Peeling et al., 2008). Finally, another important cause of gastrointestinal bleedings could be ascribed to the use of drugs, and particularly analgesics, that could exacerbate the blood losses that are already a feature of the endurance athlete and specifically of the marathon runner (Robertson et al., 1987).

Urinary iron losses are negligible, and any increase might be attributable to bladder trauma or traumatic intravascular hemolysis (Zoller & Vogel, 2004). The motion of the bladder during exercise may be the cause of bleeding in the presence of preexisting microlesions. The intensity of exercise, however, seems to be the most important cause of hematuria, since the blood flow has been shown to be proportionally decreased according to exercise intensity (Peeling et al., 2008). Significant loss of iron in sweat is limited to conditions of very high sweat rates (e.g., 0.08 $mg/m^2/h$), and it decreases over time, possibly due to the elimination of cellular debris and external contamination in the initial sweat (Peeling et al., 2008).

The estimated reduced hepatic iron concentration in endurance athletes is typically the result of the increased intestinal loss and the reduced absorption of non-heme iron that occurs during training. It has also been shown that the mean fractional absorption of an ingested dose of $FeSO_4$ is 16% in male runners and 30% in sedentary controls (Nachtigall et al., 1996).

Other than as a component of the functional core of Hb, iron enters in the constitution of a number of mitochondrial enzymes involved in the energy metabolism, whose functions are all modulated by exercise. Accordingly, tissue iron deficiency decreases performance in both animal and human studies, whereas long-term supplementation improves the response to exercise (Zoller & Vogel, 2004).

In a large study involving 873 athletes (514 men and 359 women) participating in disciplines characterized by different energy expenditures (aerobic, anaerobic, and mixed), Milic et al. (2011) showed that female athletes involved in mixed disciplines had lower value of serum ferritin and thus a higher risk of depletion of iron stores. Although elite athletes are prone to iron deficiency, the relative risk is lower than in young subjects involved in intensive training, probably due to the continuous monitoring and supplementation that athletes typically undergo (Deugnier et al., 2002).

Role of Inflammation in Iron Deficiency

New findings about the role of inflammation-related cytokines and hormones in inducing iron deficiency are arising alongside the traditional explanation of exercise-dependent iron losses. Inflammation is an acute phase response of the body to stressful stimuli and is characterized by a variety of systemic biochemical alterations whose final function is to help the organism to overcome a potentially harmful situation. Endurance exercise is a known inflammatory stimulus evoking an acute phase response and resulting in postexercise cytokine levels comparable to those seen during bacterial infection, surgery,

burns, and inflammatory disease. TNF-α and IL-1β increase up to 2–3 fold, whereas IL-6 can be increased up to 100-fold during exercise. This latter cytokine is released from the contracting muscle, especially when depleted of glycogen, at a rate closely related to the exercise intensity (Peeling et al., 2008).

Hepcidin, the inhibitor of iron absorption and recycling, has recently been found to be induced by IL-6 (Nemeth & Ganz, 2009). It is likely that the exercise-induced increase in IL-6 might upregulate hepcidin, and this mechanism might determine a reduction in postexercise iron absorption thereby providing another potential explanation for the high incidence of iron deficiency among athletes (Peeling et al., 2008).

Diagnosis of Iron Deficiency

Biological Variability and Critical Difference

Biological variability is due to the heterogeneity of physiological influences among individuals and in individuals over time and, at variance with other sources of variability (i.e., preanalytical, analytical, and postanalytical), its influence on the total variability of diagnostics is essentially unlimited. The two components of biological variability are interindividual (CV_g) and intraindividual (CV_i) variability. Interindividual variability consists of the differences in the magnitude of a measurement among individuals and is mainly derived from race, gender, and age. Intraindividual variability refers instead to the cyclical biological rhythm and the fluctuation beyond a homeostatic value in one single individual. The intraindividual variability is generally smaller than interindividual variability, so that the latter is the main determinant of the total biological variability (Noe, 2001).

The biological variability depends on a number of factors, roughly classifiable as "uncontrollable" (e.g., age, gender, menopause, and ethnicity) or controllable (circadian rhythm, menstrual cycle, exercise). The uncontrollable factors cannot be modified, so they require a careful calculation of age- and gender-related reference ranges. Conversely, the controllable factor effects might be minimized by a strict standardization of timing and condition of sampling, such as collection in the morning (from 8 AM to 10 PM) and limitation of strenuous or prolonged exercise 24 hours before sample collection (Banfi et al., 2010a).

The total variability (V_t) is the sum of analytical (V_a) and biological variability (V_b). In general, V_a is rather low in automated hematological analysis (<3%), so that V_t becomes highly dependent on V_b. The classical analytical goals for laboratory parameters are consistent with the foremost Fraser's equation $V_a = ½V_b$. Hematological parameters are characterized by a low V_a as well as a low V_b, so that the analytical goals are always reached for parameters such as Hb, RBC, and those derived from the former (Banfi et al., 2010b).

The critical difference (CD) corresponds to the statistically significant difference between two consecutive measurements in the same subject, which is considered to be unlikely to be due to casual oscillation of values. The 95% probability of a true change in a repeatedly measured parameter, not due to casual oscillations, is described as $CD^{95} = 2.77 (CV_g^2 + CV_i^2)^{1/2}$. The factor 2.77 is equal to $\sqrt{2}$ times the z score for the difference. In other terms, CD identifies whether an external factor (e.g., training or therapy) really modified the result of the parameter and whether the modification is independent of instrumental or biological variability (Banfi et al., 2010a).

Diagnostic Indices

After depletion of the iron stores, a decline in Hb, mean corpuscular Hb concentration (MCHC), size and volume of newly formed RBC, muscular myoglobin, and cellular content of Fe–S and heme-containing cytochromes is typically recorded.

Table 19.1 summarizes the reference values and the biological variability of iron status markers in adults.

From the diagnostic perspective, iron deficiency can be categorized into three degrees of severity:

- Iron depletion. Iron stores in the bone marrow, liver, and spleen are depleted (serum ferritin <35 μg/l; Hb >115 g/l; Tf saturation >16%).
- Iron-deficient erythropoiesis. Erythropoiesis diminishes as the iron supply to the erythroid marrow

Table 19.1 Reference ranges and biological variability of the main markers of iron status in adults

Parameter	Reference range		Biological variability	
	Male	Female	CV_g (%)	CV_i (%)
Fe^{3+} (μg/dl)	65–170	50–170	23	27
TIBC (μg/dl)	250–450	250–450	16	9
Tf (mg/dl)	220–400	220–400	2	3
Tf saturation (%)	20–60	20–60	20	9
TfR (mg/l)	0.3	1.75	30	11
Ferritin (ng/ml)	20–250	10–150	14	13
Hp (mg/dl)	30–185	30–180	36	23
Hb_{free} (g/l)	<0.1	<0.1	N.D.	N.D.

is reduced (serum ferritin < 20μg/l; Hb >115 g/l; Tf saturation <16%).

- Iron-deficient anemia. Hb production falls, up to anemia (serum ferritin <12 μg/l, Hb <115 g/l, Tf saturation <16%) (Peeling et al., 2008).

In the more severe cases of anemia, impairment of skeletal muscle working capacity is common (Schumacher et al., 2002), while the effect of iron deficiency without anemia, which seems more common in endurance athletes, is still undefined (Peeling et al., 2008).

A depletion of body iron stores is characterized by low serum ferritin and decreased hemosiderin, whereas inadequate delivery of iron to the peripheral tissues is characterized by low Tf saturation, a high erythrocyte protoporphyrin concentration, and an elevated sTfR (Beard & Tobin, 2000). The current indication for iron supplementation is serum ferritin <35 μg/l in male athletes and <22 μg/l in female athletes (Milić et al., 2010).

Serum Iron Serum iron concentration represents the amount of Fe^{3+} bound to Tf, but not the iron contained within free Hb. The routine assessment of serum iron is affected by a number of external factors and it is therefore an imprecise marker of iron status. Iron is subjected to a circadian rhythm with an acrophase between 8 AM and 10 AM, and a nadir at midnight. The range of circadian variation in the general population has been reported to span between 48 and 106 μg/dl (Haus, 1992). Diet also exerts a strong influence on this parameter, along with therapeutic iron loading, which together make it an unreliable marker for monitoring replacement therapy. Moreover, iron reaches the highest concentration in the premenstrual phase and the lowest during menstruation. Estroprogestinic oral contraceptive (OC) administration also increases serum iron (Banfi, 2005).

As such, serum iron concentration shows a high biological variability, with a CV_g of 23% and a CV_i of 27% (Sebastian-Gambaro et al., 1997).

Iron is measured preferably on serum, though heparin-plasma might also be suitable, but it cannot be assessed on EDTA- or citrate-plasma. The serum must be separated by centrifugation within 2 hours after blood collection. Samples are stable at room temperature for 7 days (Banfi, 2005). The reference ranges of iron in serum are 65–170 μg/dl in adult males, 50–170 μg/dl in adult females, and 50–120 μg/dl in teenagers (Burtis, 2006).

Total Iron Binding Capacity (TIBC) TIBC is measured in serum by adding Fe^{3+} to saturate the Tf. Excess Fe^{3+} is eliminated by adsorption. The iron measurement provides the final TIBC value (Banfi, 2005). The reference range is 250–450 μg/dl (Burtis, 2006), with a CV_g of 16% and a CV_i of 9% (Lacher et al., 2010).

Transferrin The mean half-life of Tf is 7 days and at least for the same period this parameter is reportedly stable in whole blood. It is assessed preferably in serum, but it can also be measured in EDTA- or heparin-plasma. Tf is a stable protein, being measurable in serum for at least 4 months after storage at room temperature. The best approaches entail immunoassays or electrophoresis, but it might also be derived from the TIBC by multiplying this last parameter by 0.7 (Banfi, 2005).

The reference range is 220–400 mg/dl (Burtis, 2006) and, characteristically, its biological variability is very low, CV_g being 2% and CV_i 3% (Sebastian-Gambaro et al., 1997).

Transferrin Saturation Transferrin saturation is typically derived by the following formula: [serum iron]100/TIBC (Banfi, 2005). The reference range is

between 20% and 60% (Burtis, 2006), with a CV_g of 20% and CV_i of 9% (Kroot et al., 2009).

Transferrin Soluble Receptor sTfR is an index of the erythropoiesis rate and of the erythroid mass. Its concentration increases as a consequence of iron deficiency as well as of hemolysis, both conditions being characterized by an increased turnover of RBCs. The sTfR therefore more accurately reflects the iron demand of bone marrow (Banfi, 2005; Zoller & Vogel, 2004). Day-to-day variations are greater for ferritin (13–75%) than for sTfR (4–16%). The sTfR, especially the sTfR/log(ferritin) index, is less variable and might more accurately reflect the iron status of an athlete. The influence of physical activity on sTfR expression has been the subject of a limited number of investigations (Zoller & Vogel, 2004).

sTfR should be assessed, by immunoassay, in serum separated within 8 hours from blood collection (Banfi, 2005). The normal values are between 0.30 and 1.75 mg/l (Burtis, 2006), although an international standard for harmonization of test results expression is lacking (Banfi, 2005). The CV_g is 30% and the CV_i is 11% (Raya et al., 2001).

Ferritin Serum ferritin concentrations decrease during training, since it reflects the onset of iron deficiency after physical activity. However, iron stores in the body can more reliably be assessed by measuring the amount of iron removed during repeated phlebotomies until anemia develops, then subtracting the amount of dietary iron absorbed during phlebotomy. This method is rather understandably unsuitable for the repeated determination of iron stores in athletes before and after training (Zoller & Vogel, 2004). Ferritin is preferably assessed in serum, while the use of plasma is possible depending on the analytical method (generally immunoassays). It is reportedly stable and can be measured after 7 days at room temperature without any significant variation (Banfi, 2005). Normal values in adult men are between 20 and 250 ng/ml and in women 10 and 150 ng/ml (Burtis, 2006). The CV_g is 14% and the CV_i is 13% (Sebastian-Gambaro et al., 1997).

Haptoglobins Hps are a group of globular multimeric proteins that bind covalently to free Hb, released from RBC, to avoid the possible negative effects on the acid–base balance. The irreversible binding Hp–Hb allows recognition and uptake by the reticuloendothelial system, a mechanism that allows the recovery of up to 3 g of Hb in the presence of massive hemolysis. Hp is also an acute phase protein, and its levels are increased substantially in inflammation. Several polymorphisms exist, which explain the wide variation of the reference ranges across different ethnic groups. Hp is generally measured by immunometrical assays in serum, but heparin- or EDTA-plasma is also suitable. It is stable in serum for up to 3 months at room temperature (Banfi, 2005). As previously described, its levels in serum are highly variable, and the normal range might span from 30 to 185 ng/ml (Burtis, 2006). The CV_g is 36% and the CV_i is 23% (Sebastian-Gambaro et al., 1997), probably as a consequence of the involvement of this protein in inflammation.

Free Hemoglobin The presence of free Hb in plasma is the result of its release from the breakdown of RBC. In plasma, Hb_{free} is buffered by Hp to avoid any change in the acid–base balance and prevent its precipitation in the form of cylinders in the renal tubules. Free Hb is measurable in EDTA- or heparin-plasma (separated within 8 hours from blood collection) by spectrophotometric techniques; its quantification is performed through comparison with a known hemolytic sample; the limit is 0.1 g/l (Banfi, 2005); in athletes, Hb_{free} concentrations higher than 0.1 g/l represent a sign of sports anemia.

Summary In summary, due to its high intra- and interindividual variability and the susceptibility to a number of external factors (diet) the measurement of serum iron is unlikely to reflect the real iron status of the body. Ferritin, being a marker of the iron store status and virtually unaffected by environmental factors on a short-term basis, seems to be more useful in this setting. As an acute phase protein, ferritin increases independently of iron status as a consequence of inflammation, but decreased levels are specifically diagnostic of iron deficiency.

Tf saturation is a useful parameter in differentiating iron-deficiency anemia from the anemia of

chronic disease, but it is affected by diurnal variation and the coexistence of other clinical conditions. As a marker of erythroid mass, sTfR represents a good index of bone marrow iron, which is not affected by the acute phase response. Quantitatively, the total body iron is well represented by the ratio: log(sTfR/ferritin). The result is directly proportional to the amount of stored iron in iron-replete subjects and tissue iron deficit in iron deficiency (Zimmermann & Hurrell, 2007).

Iron Supplementation

Iron Bioavailability

Despite its abundance, the oxidized iron form, Fe^{3+}, is poorly bioavailable due to limited solubility. Nutritional iron deficiency arises when physiological requirements cannot be met by iron absorption from the diet. Dietary iron bioavailability is low in subjects with low meat consumption. In meat from 30% to 70% of the iron is contained in heme of which 15–35% is absorbed; in vegetarian diet less than 10% of the iron, in non-heme form, is absorbed. The absorption of non-heme iron is increased by meat and ascorbic acid, defined as enhancers, while the list of iron absorption inhibitors is very much longer, including cellulose and hemicelluloses, pectin, phytates, polyphenols, bran, and calcium (Beard & Tobin, 2000; Hurrell, 2002; Zimmermann et al., 2005). On the other hand, heme iron absorption is enhanced by the coadministration of animal proteins and inhibited by calcium (Beard & Tobin, 2000).

Iron is widely present in foods so that its intake is directly related to the energy intake (6 mg per 4120 kJ) (Beard & Tobin, 2000): when the iron requirement is higher than the energy need the risk of deficiency increases; this is the case of growth in infants, young children, and adolescents, bleeding during menstruation and pregnancy (Zimmermann & Hurrell, 2007). As already reported, there is a delicate equilibrium between dietary intake and iron losses. The recommended daily intakes (RDIs)

Table 19.2 RDI for iron, during life according to different official international organizations (Schumann et al., 2007)

	WHO/FAO			DACH		US	
Life stage	Age (years)	Fe (mg/day) Bioav. 15%	Fe (mg/day) Bioav. 5%	Age (years)	Fe (mg/day)	Age (years)	Fe (mg/day)
Infants	0.5–1	6.2	18.6	0.5–1	0.5	0–0.5	0.27
	1–3	3.9	11.6	1–4	8	0.5–1	11
	4–6	4.2	12.6	4–7	8	1–3	7
	7–10	5.6	17.8	7–10	10	4–8	10
Males	11–14	9.7	29.2	10–13	12	9–13	8
	15–17	12.7	37.6	13–15	12	14–18	11
	18+	9.1	27.4	15–19	12	18+	8
				19+	10		
Females	11–14 no mens.	9.3	28	10–13	15	9–13	8
	11–14 with mens.	12.5	37.6	13–19	15	14–18	15
	15–17	20.7	62	19+	15	18+	18
	18+	19.6	58.8				
Postmenopausal		7.5	22.6		10		8
Pregnancy		Supplements			30		27
Lactation		10	30		20		9 (10 in younger)

WHO, World Health Organization; FAO, Food and Agriculture Organization; DACH, Germany, Austria, and Switzerland; US, United States; Bioav., bioavailability; mens., menstruation. EU (European Union) recommendations are identical with WHO/FAO at 15% bioavailability.

during life, according to different official international organizations, are reported in Table 19.2.

Specific Iron Needs and Iron Supplementation in Athletes

Generally, erythroblasts need 20 mg of iron daily for heme synthesis, even if it is mainly sustained by the recycling of senescent RBC (Banfi, 2005). In aerobic dancers the daily consumption of meat has been demonstrated to be sufficient to keep the ferritin level. On the other hand, moderate doses of $FeSO_4$ (125 mg equivalent to 39 mg of elemental iron) were able to prevent the ferritin decrease in college swimmers (Beard & Tobin, 2000).

It is now clear that there are three groups of athletes with a greater risk for iron deficiency: females, distance runners, and vegetarian; the coexistence of more conditions adds increased risk. Especially in these cases, iron intake monitoring and daily supplementation may prevent deficiency; on the other hand pharmacologic interventions should be limited to encourage development of good dietary habits.

The absorption of supplemental iron depends on the type of preparation used. Paradoxically complex multi-mineral preparation may provide less iron than believed, due to reduced bioavailability because of the presence of agents that inhibit iron absorption (e.g., calcium salts). Simpler preparations ($FeSO_4$, ferrous gluconate, or ferrous fumarate, containing 37–106 mg of elemental iron), however, need to be carefully controlled since high doses can cause gastrointestinal distress and constipation and also the possibility of increased oxidant potential (Beard & Tobin, 2000) or, more importantly, the possible development of hemochromatosis in susceptible subjects (Jenkinson & Harbert, 2008). Because young female athletes are often instructed to consume daily iron supplements in doses of >50 mg, noncompliance due to gastrointestinal symptoms can be a significant issue. Reports of iron supplementation on a weekly or biweekly basis in Third World populations showed the strong possibility that less frequent use of iron supplements can still provide positive effects without gastrointestinal distress. However, no known systematic studies have used this approach with athletes (Beard & Tobin, 2000).

It is important to consider that no performance benefit has been reported as a consequence of iron supplementation in athletes who were not iron deficient while improvements in muscular fatigability and VO_2 max have been reported in treated deficient athletes (Jenkinson & Harbert, 2008; Zoller & Vogel, 2004). Iron deficiency without anemia in athletes is still a controversial indication for supplementation (Zoller & Vogel, 2004).

If iron-deficiency anemia is confirmed by laboratory analysis, supplementation is necessary, but only after appropriate dietary adjustment (Jenkinson & Harbert, 2008).

In summary:

- The preferable iron source for professional and nonprofessional athletes is represented by a regular and balanced diet.
- Only when the diet has proved to be insufficient to satisfy the needed amount of iron, supplementation should be allowed.
- Supplementation should be performed through oral preparations and should be discontinued when the ferritin level is repleted.
- Supplementation regimens should be chosen to avoid any adverse effects.
- Parenteral administration of iron must be avoided because the supplementation by this way overcomes the physiological gut regulation of absorption.
- The quantity of iron to be administrated and the administration route might be differently chosen in case of frank pathology.

Final Remarks

Iron is an essential element, responsible for the efficient delivery of oxygen to the body and to the working muscle and for production of energy in the mitochondria. It is thus clear that iron status is an important determinant of athletic performance, at least in the case of severe deficiency, when the iron-dependent reactions cannot meet the high energy demand. However, it is common, for athletes, to be iron deficient as a result of iron losses occurring

during training from hemolysis, hematuria, sweating, and gastrointestinal bleeding, but also as a consequence of exercise-induced inflammation through the activity of hepcidin (Peeling et al., 2008). This situation can be more severe if other susceptibility factors coexist: female gender, distance running, and vegetarian diet (Jenkinson & Harbert, 2008).

Diagnosis of iron deficiency must be based on ferritin serum levels along with sTfR and the derived parameter log(sTfR/ferritin) and eventually accompanied by the routine hematological parameters useful to diagnose the eventual anemia (Zimmermann & Hurrell, 2007).

Currently no evidence exists to support the popular idea that iron supplementation increases athletic performance, except in individuals in whom iron deficiency is established. In athletes with low serum ferritin concentrations without anemia, iron supplementation might be useful. Serum ferritin concentrations should be monitored in athletes during periods of hard training, and physiological decreases in serum ferritin during the early stages of training should be taken into account before any decision to give iron is made. Some athletes take iron supplements to optimize the effect of rhEPO, a practice that remains unethical and dangerous (Zoller & Vogel, 2004).

$FeSO_4$, ferrous gluconate, and ferrous fumarate seem to be the most useful supplements in correcting an established iron deficiency; however, supplementation must be considered only after nutritional consultation and dietary modification (Jenkinson & Harbert, 2008).

References

Adamson, J.W. & Vapnek, D. (1991) Recombinant erythropoietin to improve athletic performance. *New England Journal of Medicine* 324, 698–699.

Aisen, P., Enns, C., & Wessling-Resnick, M. (2001) Chemistry and biology of eukaryotic iron metabolism. *International Journal of Biochemistry and Cell Biology* 33, 940–959.

Andrews, N.C. (1999) Disorders of iron metabolism. *New England Journal of Medicine* 341, 1986–1995.

Andriopoulos Jr, B., Corradini, E., Xia, Y., et al. (2009) BMP6 is a key endogenous regulator of hepcidin expression and iron metabolism. *Nature Genetics* 41, 482–487.

Banfi, G. (2005) *Ematologia dello Sport*. Edi Ermes, Milan, Italy.

Banfi, G., Lombardi, G., Colombini, A., et al. (2010a) Bone metabolism markers in sports medicine. *Sports Medicine* 40, 697–714.

Banfi, G., Lombardi, G., Colombini, A., et al. (2010b) Whole-body cryotherapy in athletes. *Sports Medicine* 40, 509–517.

Beard, J. & Tobin, B. (2000) Iron status and exercise. *American Journal of Clinical Nutrition* 72, 594S–597S.

Burtis, C.A., Ashwood, E.R., & Bruns, D.E. (2006) Hemoglobin, iron and bilirubin. In *Tietz. Textbook of Clinical Chemistry and Molecular Diagnostics*, 4th edn (eds. C.A. Burtis, E.R. Ashwood, & D.E. Bruns), pp. 1974–2072. Elsevier Saunders, St. Louis, MO.

Cook, J.D. (1999) Defining optimal body iron. *Proceedings of the Nutrition Society* 58, 489–495.

Corsetti, R., Lombardi, G., Lanteri, P., et al. (2012) Haematological and iron metabolism parameters in professional cyclists during the Giro d'Italia 3-weeks stage race. *Clinical Chemistry and Laboratory Medicine* 50, 949–956.

Courselaud, B., Pigeon, C., Inoue, Y., et al. (2002) C/EBPalpha regulates hepatic transcription of hepcidin, an antimicrobial peptide and regulator of iron metabolism. Cross-talk between C/EBP pathway and iron metabolism. *Journal of Biological Chemistry* 277, 41163–41170.

Dallman, P.R. (1986) Biochemical basis for the manifestations of iron deficiency. *Annual Review of Nutrition* 6, 13–40.

Deugnier, Y., Loreal, O., Carre, F., et al. (2002) Increased body iron stores in elite road cyclists. *Medicine & Science in Sports & Exercise* 34, 876–880.

Diaz, V., Lombardi, G., Ricci, C., et al. (2011) Reticulocyte and haemoglobin profiles in elite triathletes over four consecutive seasons. *International Journal of Laboratory Hematology* 33, 638–644.

Donovan, A., Lima, C.A., Pinkus, J.L., et al. (2005) The iron exporter ferroportin/Slc40a1 is essential for iron homeostasis. *Cell Metabolism* 1, 191–200.

Ehn, L., Carlmark, B., & Hoglund, S. (1980) Iron status in athletes involved in intense physical activity. *Medicine & Science in Sports & Exercise* 12, 61–64.

Finberg, K.E. (2009) Iron-refractory iron deficiency anemia. *Seminars in Hematology* 46, 378–386.

Galaris, D. & Pantopoulos, K. (2008) Oxidative stress and iron homeostasis: mechanistic and health aspects. *Critical Reviews in Clinical Laboratory Sciences* 45, 1–23.

Gomme, P.T., McCann, K.B., & Bertolini, J. (2005) Transferrin: structure, function and potential therapeutic actions. *Drug Discovery Today* 10, 267–273.

Hallberg, L. & Hulthen, L. (2003) High serum ferritin is not identical to high iron stores. *American Journal of Clinical Nutrition* 78, 1225–1226.

Haus, E., Touitou, Y. (1992) Chronobiology in laboratory medicine. In *Biological Rhythms in Clinical and Laboratory Medicine* (eds. Y. Touitou & E. Haus), pp. 673–708. Springer, Berlin.

Hentze, M.W., Muckenthaler, M.U., Galy, B., et al. (2010) Two to tango: regulation of mammalian iron metabolism. *Cell* 142, 24–38.

Hurrell, R. (2002) How to ensure adequate iron absorption from iron-fortified food. *Nutrition Reviews* 60, S7–S15; discussion S43.

Jenkinson, D.M. & Harbert, A.J. (2008) Supplements and sports. *American Family Physician* 78, 1039–1046.

Kell, D.B. (2009) Iron behaving badly: inappropriate iron chelation as a major contributor to the aetiology of vascular and other progressive inflammatory

and degenerative diseases. *BMC Medical Genomics* 2, 2.

Kroot, J.J., Hendriks, J.C., Laarakkers, C.M., et al. (2009) (Pre)analytical imprecision, between-subject variability, and daily variations in serum and urine hepcidin: implications for clinical studies. *Analytical Biochemistry* 389, 124–129.

Lacher, D.A., Hughes, J.P., & Carroll, M.D. (2010) Biological variation of laboratory analytes based on the 1999–2002 National Health and Nutrition Examination Survey. *National Health Statistics Reports*, 1–7.

Lee, P.L. & Beutler, E. (2009) Regulation of hepcidin and iron-overload disease. *Annual Review of Pathology* 4, 489–515.

Lombardi, G., Lanteri, P., Fiorella, P.L., et al. (2013) Comparison of the hematological profile of elite road cyclists during the 2010 and 2012 GiroBio ten-day stage races and relationships with final ranking. *PLoS ONE* 8, e63092.

Marx, J.J. (1997) Iron deficiency in developed countries: prevalence, influence of lifestyle factors and hazards of prevention. *European Journal of Clinical Nutrition* 51, 491–494.

Meynard, D., Kautz, L., Darnaud, V., et al. (2009) Lack of the bone morphogenetic protein BMP6 induces massive iron overload. *Nature Genetics* 41, 478–481.

Milić, R., Banfi, G., Del Fabbro, M., et al. (2010) Serum creatinine concentrations in male and female elite swimmers. Correlation with body mass index and evaluation of estimated glomerular filtration rate. *Clinical Chemistry and Laboratory Medicine* 49, 285–289.

Milic, R., Martinovic, J., Dopsaj, M., et al. (2011) Haematological and iron-related parameters in male and female athletes according to different metabolic energy demands. *European Journal of Applied Physiology* 111, 449–458.

Nachtigall, D., Nielsen, P., Fischer, R., et al. (1996) Iron deficiency in distance runners. A reinvestigation using Fe-labelling and non-invasive liver iron quantification. *International Journal of Sports Medicine* 17, 473–479.

Nemeth, E. & Ganz, T. (2009) The role of hepcidin in iron metabolism. *Acta Haematologica* 122, 78–86.

Noe, D. (2001) Biologic variability. In *The Logic of Laboratory Medicine*, 2nd edn. Available at http://users.rcn.com/dennisanoe (accessed on May 2011).

Olsson, K.S. & Norrby, A. (2008) Comment to: Hepcidin: from discovery to differential diagnosis. Haematologica 2008; 93:90–97. *Haematologica* 93, e51; discussion e52.

Peeling, P., Dawson, B., Goodman, C., et al. (2008) Athletic induced iron deficiency: new insights into the role of inflammation, cytokines and hormones. *European Journal of Applied Physiology* 103, 381–391.

Pinto, J.P., Ribeiro, S., Pontes, H., et al. (2008) Erythropoietin mediates hepcidin expression in hepatocytes through EPOR signaling and regulation of C/EBPalpha. *Blood* 111, 5727–5733.

Raya, G., Henny, J., Steinmetz, J., et al. (2001) Soluble transferrin receptor (sTfR): biological variations and reference limits. *Clinical Chemistry and Laboratory Medicine* 39, 1162–1168.

Robertson, J.D., Maughan, R.J., & Davidson, R.J. (1987) Faecal blood loss in response to exercise. *British Medical Journal (Clinical Research Edition)* 295, 303–305.

Schumacher, Y.O., Schmid, A., Konig, D., et al. (2002) Effects of exercise on soluble transferrin receptor and other variables of the iron status. *British Journal of Sports Medicine* 36, 195–199.

Schumann, K., Ettle, T., Szegner, B., et al. (2007) On risks and benefits of iron supplementation recommendations for iron intake revisited. *Journal of Trace Element in Medicine & Biology* 21, 147–168.

Sebastian-Gambaro, M.A., Liron-Hernandez, F.J., & Fuentes-Arderiu, X. (1997) Intra- and inter-individual biological variability data bank. *European Journal of Clinical Chemistry & Clinical Biochemistry* 35, 845–852.

Sinclair, L.M. & Hinton, P.S. (2005) Prevalence of iron deficiency with and without anemia in recreationally active men and women. *Journal of the American Dietetic Association* 105, 975–978.

Telford, R.D., Sly, G.J., Hahn, A.G., et al. (2003) Footstrike is the major cause of hemolysis during running. *Journal of Applied Physiology* 94, 38–42.

Wang, J. & Pantopoulos, K. (2011) Regulation of cellular iron metabolism. *Biochemical Journal* 434, 365–381.

Wang, R.H., Li, C., Xu, X., et al. (2005) A role of SMAD4 in iron metabolism through the positive regulation of hepcidin expression. *Cell Metabolism* 2, 399–409.

Weiss, G. & Goodnough, L.T. (2005) Anemia of chronic disease. *New England Journal of Medicine* 352, 1011–1023.

Wilkinson, J.G., Martin, D.T., Adams, A.A., et al. (2002) Iron status in cyclists during high-intensity interval training and recovery. *International Journal of Sports Medicine* 23, 544–548.

Yeh, K.Y., Yeh, M., Mims, L., et al. (2009) Iron feeding induces ferroportin 1 and hephaestin migration and interaction in rat duodenal epithelium. *American Journal of Physiology—Gastrointestinal and Liver Physiology* 296, G55–G65.

Zimmermann, M.B. & Hurrell, R.F. (2007) Nutritional iron deficiency. *Lancet* 370, 511–520.

Zimmermann, M.B., Chaouki, N., & Hurrell, R.F. (2005) Iron deficiency due to consumption of a habitual diet low in bioavailable iron: a longitudinal cohort study in Moroccan children. *American Journal of Clinical Nutrition* 81, 115–121.

Zoller, H. & Vogel, W. (2004) Iron supplementation in athletes-first do no harm. *Nutrition* 20, 615–619.

Chapter 20

Calcium and Vitamin D

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Introduction

Calcium and vitamin D play an important role in an athlete's health, training, and performance. While it has long been recognized that adequate calcium and vitamin D status is necessary for bone health, newer research is highlighting nonskeletal benefits—particularly for vitamin D—which include immune modulation, muscle function, and even athletic performance. In addition, low calcium intake and vitamin D deficiency are linked to increased risk for a number of chronic diseases—including hypertension, cardiovascular disease, and certain types of cancer—which can occur even in the trained athlete. This chapter reviews the importance of calcium and vitamin D in the health and performance of athletes and provides tips for assessing and treating calcium and vitamin D insufficiency.

Calcium

Calcium Absorption and Dietary Sources

Calcium is an integral component of the skeleton which provides both structure and a reservoir of calcium for other essential calcium-dependent functions. These functions include skeletal and smooth muscle contraction, nerve conduction, and intracellular signaling. Maintaining circulating ionized calcium concentration within the narrow physiological range of 1.16–1.32 mmol/l (Fischbach & Dunning, 2009) is critical for normal body functioning. If serum-ionized calcium concentration deviates even slightly, calcium-sensitive receptors within the parathyroid gland signal the release of parathyroid hormone (PTH) which induces production of calcitriol ($1,25(OH)D_2$), the hormonally active form of vitamin D. Calcitriol and PTH act to increase serum calcium concentration by increasing intestinal calcium absorption, activating bone resorption, and reducing urinary calcium excretion. As serum calcium concentration rises, feedback mechanisms reduce PTH secretion. If there is rise or overshoot in serum calcium, calcitonin, secreted by the C cells of the thyroid, blocks calcium resorption (Ross et al., 2010).

Calcium is absorbed by both active transport (transcellular) and passive diffusion across intestinal mucosa (Ross et al., 2010). The transcellular mechanism occurs primarily in the duodenum and accounts for the majority of calcium absorption at low to moderate dietary intake. This process is dependent on both $1,25(OH)D_2$ and intestinal vitamin D-binding protein. On the other hand, passive diffusion of calcium between mucosal cells occurs throughout the intestines and more readily with higher intake of calcium.

On average, fractional calcium absorption (percent of given dose absorbed) is ~25% of intake (Ross et al., 2010). Mean urinary losses average 22% of total calcium intake and fecal loss averages 75% of intake with minor losses (at least in the nonathlete) in sweat, skin, and hair. Fractional calcium

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absorption rises adaptively as intake is lowered, but this rise is not sufficient to offset low calcium intakes. Fractional calcium absorption is thought to decline with aging and after menopause (Heaney et al., 1989a).

The most recognized source of dietary calcium is dairy products including milk, yogurt, and cheese. Many green vegetables are also rich sources. In the United States, an average 72% of calcium intake comes from milk, cheese, yogurt, and foods to which dairy is added, including pizza, lasagna, and dairy desserts (Ross et al., 2010). The remaining calcium comes from vegetables, grains, legumes, fruits, eggs, meat, and other foods. Calcium fortification of an increasing number of foods (that do not naturally contain calcium)—such as orange juice and ready-to-eat cereals—is also becoming common in the United States, Canada, and other countries.

Indices of Status

Unlike vitamin D and minerals such as iron, there are no clear biochemical indices or serum markers of acute calcium intake or status. Although calcium concentration is easily measured in serum, ionized and total concentrations of calcium are tightly regulated (as discussed above) and have no correlation with calcium intake unless intake is severely restricted. In addition, total serum calcium concentration is dependent on factors including serum albumin and total serum protein concentrations, magnesium status, phosphate intake, and laxative use (Fischbach & Dunning, 2009). Over the long term, inadequate calcium intake may result in reduced bone mineral density, increasing risk for osteoporosis. Reductions in bone mineral or bone density are not good status markers of calcium intake because of their dependence on other factors including genetics, weight-bearing exercise, and intake of other nutrients (Ross et al., 2010; World Health Organization and Food and Agricultural Organization of the United Nations, 2004), including magnesium and vitamins A, C, D, and K. While calcium balance in the research setting is determined by the relationship between calcium intake and calcium absorption and excretion (World Health Organization and Food and Agricultural Organization of the United Nations, 2004), the only way to assess status in the sports medicine environment is by dietary assessment. Accurate dietary assessment, however, is difficult due to variability of calcium content of food, inadequacy of nutrient databases for calcium content of certain foods, and ability of individual athletes to recall or estimate food sources (Ross et al., 2010).

Calcium Intake and Status of Athletes

Available evidence suggests that calcium intake commonly falls short of that recommended. This is particularly true among women. According to a 2005–2006 US national survey, only about one-third of US citizens 1 year or older met the suggested target for calcium, and young women were even less likely to meet their requirements (Moshfegh et al., 2009). There, however, is a wide variation in calcium intake between countries, which generally follows animal protein intake and is dependent on intake of dairy products. According to the World Health Organization (World Health Organization and Food and Agricultural Organization of the United Nations, 2004), the lowest calcium intake occurs in developing countries with the highest in developed countries, particularly in North America and Europe. For example, despite a large number of US citizens who reportedly do not meet calcium recommendations, calcium intake in the United States averages 1031 mg/day compared to 896 mg in Europe, 368 mg in Africa, and 305 mg in the Far East. Intake may be even higher in European countries whose populations consume a lot of dairy including cheese.

Limited recent research in athletic groups has observed similar findings. Male athletes on average consume more calcium than female athletes but the results are variable (Table 20.1). Some athletes, particularly men, consume well above the recommended amount. Others who are trying to obtain a low body weight for competition, including figure skaters and distance runners, may have substandard intakes. Little published data are available on intake of athletes worldwide.

Table 20.1 Calcium intake of athletes

Study	Subjects	Assessment index	Calcium intake (mg/day) (mean and SD)
Bennell et al. (1996)	53 female and 58 male track and field athletes with (sf) or without (nsf) stress fractures Age: 21 years (17–26) Country: Australia	Food frequency questionnaire plus two 4-day food diaries	Females: 1075 (sf) and 985 (nsf) Males: 1325 (sf) and 1252 (nsf)
Ziegler et al. (1999)	20 female national figure skaters Age: 14.1 years (11–18) 21 male national figure skaters Age: 17 years (11–18) Country: United States	4-day diet records (2 nonconsecutive weekdays, both weekend days)	Females: 743 ± 634 Males: 1254 ± 694
Leachman Slawson et al. (2001)	22 African American and 37 Euro-American NCAA Division I-A women athletes (basketball, golf, tennis, track/cross-country, volleyball) Age: 20 years (18–23) 29 African American and 57 Euro-American NCAA Division I-A male athletes (basketball, football, golf, soccer, track/cross-country) Age: 20 years (18–24) Country: United States	Calcium checklist over past 7 days	All Females: 898 ± 467 Euro-American: 878 ± 434 African American: 930 ± 527 All Males: 1354 ± 904 Euro-American: 1472 ± 962 African American: 1120 ± 740
Beshgetoor and Nichols (2003)	25 female master cyclists and runners —16 supplementing, 9 not supplementing Age: 50 years (50–59) Country: United States	4-day diet records (3 weekdays, 1 weekend day)	1708 ± 127 (supplementing) 791 ± 174 (not supplementing)
Clark et al. (2003)	13 female NCAA Division I soccer players Age: 20 ± 1 year Country: United States	3-day diet (records 2 weekdays, 1 weekend day) during pre- and post-season and modified 24-h recall	Pre-season: 931 ± 223 Post-season: 695 ± 289
Ward et al. (2004)	57 female collegiate athletes (22 cross-country, 15 soccer, 3 basketball, 13 volleyball, 23 field hockey players) Age: 17–21 years Country: United States	Rapid assessment method (RAM) and 6-day diet records	1143 ± 514
Juzwiak et al. (2008)	44 male tennis players Age: 10–19 years Country: Brazil	4-day nonconsecutive diet (3 weekdays, 1 weekend day)	926 ± 353

Source: Data compiled by Keri Shaw as part of an undergraduate independent study project at the University of Wyoming.

Functions of Calcium

Calcium is most commonly associated with the formation and metabolism of bone. It also plays a major role in the metabolic function of every tissue primarily through action as an intracellular signal. As such, the concentration of ionized calcium is tightly regulated in the circulation through buffering from bone tissue, and within the cell.

Bone Health Calcium salts provide rigidity to bone structure, which in the vertebrate skeleton is in a form that approximates hydroxyapatite [$Ca_{10}(OH)_2(PO_4)_6$] (World Health Organization and

Food and Agricultural Organization of the United Nations, 2004). This form of calcium is embedded in the collagen fibrils and is critical for not only rigidity, but also strength and elasticity (Ross et al., 2010). This allows for normal movement during exercise and sports performance. While the formation of endochondral bone in the axial skeleton and long bones is beyond the concept of this text, it is important to mention that both calcium and phosphorus are required for adequate mineralization. In the initial stages of mineralization, phosphorus is laid down as phosphate, which subsequently drives the binding of positively charged calcium ions. Too much or too little mineral can disrupt the chemical structure of the hydroxyapatite and impair bone structure, the former making it too brittle and the latter making it too ductile and weak. While calcium is critical to bone formation, genetics is thought to be the chief determinant of bone mass, which is in turn the major determinant of bone structure. Mechanical loading induced by sports training and regular physical activity is critical for skeletal homeostatis. Specifically, mechanical loading of the skeleton (i.e., by weight-bearing exercise such as running, jumping) promotes bone formation, particularly in childhood and adolescence, whereas unloading the skeleton as during bed rest following injury or illness profoundly uncouples homeostasis and promotes bone resorption and suppresses bone formation (Ross et al., 2010). In the general population, evidence suggests that calcium intake correlates with bone mineral density (Ross et al., 2010), but in young athletes and military personnel, intakes greater than 1500 mg/day may be needed to prevent stress fractures (Tenforde et al., 2010).

Calcium Signaling and Other Functions Other functions of calcium critical to sports and exercise performance include intracellular signaling, neuromuscular activation, enzyme activation, blood coagulation, and hormone secretion. Fluctuations in intracellular calcium concentration serve as a major pathway for intracellular signaling, which mediates all types of muscle contraction, including that of the heart, skeletal muscle, and smooth muscles found in blood vessels such as the veins and arteries. Calcium also activates a number of enzymes, which include those involved in the synthesis and breakdown of muscle and liver glycogen, and is critical for blood clotting. Calcium's function as a mediator of smooth and skeletal muscles explains why regular calcium intake is involved in lowering blood pressure when consumed as part of a healthy diet (Nowson et al., 2004) and is often anecdotally implicated as a cause of/or treatment for muscle cramps. Research supporting its use in the treatment of sports-related muscle cramps, however, is not available.

Calcium Requirements for Athletes

The dietary recommendation for calcium varies between countries. Higher recommendations are made by the United States and Canada and the 2004 Expert Consultation recommendations of the WHO compared to other countries (Table 20.2). While the different recommendations may be partially related to genetic (ethnic), cultural, and dietary differences by country, the different assumptions used to establish calcium recommendations are also important. For example, the recommendations of Australia and the United Kingdom do not account for insensible calcium losses, and the European Union assumes a 30% rather than 25% fractional absorption of dietary calcium (World Health Organization and Food and Agricultural Organization of the United Nations, 2004). Dietary factors of importance include vitamin D status and animal protein and sodium intakes. Higher intake of the latter increases urinary calcium excretion and is presumed to increase calcium requirement. The higher requirements by some countries for adolescents and postmenopausal women account for the higher rate of skeletal calcium accretion during puberty (from ages 10 to 17), and either extra urinary obligatory calcium losses (World Health Organization and Food and Agricultural Organization of the United Nations, 2004) or loss of the estrogen-protective effect on bone (Ross et al., 2010) following menopause.

Regular exercise or sports training has not been shown to increase calcium requirements. There is a suggestion, however, that exercise may increase calcium loss in sweat and urine. Several studies have found high concentrations (average ~45 mg/l) of calcium in sweat (Baker et al., 2011). This suggests

Table 20.2 Recommended intake for calcium and vitamin D

Group	Australia and New Zealand[a]	Nordic Countries[b]	United Kingdom[c]	United States and Canada[d]	World Health Organization[c]
Calcium (mg/day)					
Children	500–1000	900	350–550	700–1000	500–700
Adolescents	1300	900	800 girls 1000 boys	1300	1300
Adults >18 or 19 years	1000	800	700	1000	1000
Older adults	1300 women pm 1300 men >70 years	800	700	1200 women pm 1200 men >70 years	1200 women pm 1200 men >65 years
Vitamin D (IU/day) (mg/day in parentheses)					
Children	200 (5)	300 (7.5)	280 (7) <4 years	600 (15)	200 (5)
Adolescents	200 (5)	300 (7.5)	400 (10)	600 (15)	200 (5)
Adults >18 or 19 years	200 (5)	300 (7.5)	400 (10)	600 (15)	200 (5)
Older Adults	400 (10)—50–70 years 600 (15) >70 years	400 (10 >60 years)	400 (10)	800 (20) >70 years	400 (10)—51–65 years 600 (15) >65 years

Source: Compiled using the following references: Anonymous (2004), Scientific Advisory Committee on Nutrition (SACN) (2007), The Australian National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (2006), Ross et al. (2010), and World Health Organization and Food and Agricultural Organization of the United Nations (2004).

Recommended daily intake for children >1 year and adults defined as >18 or 19 years. Nutrient requirements are often higher after menopause (pm, postmenopause).

[a]Recommended dietary intake (calcium); adequate intake (vitamin D).
[b]Nutrition recommendation.
[c]Recommended nutrient intake.
[d]Dietary reference intake.

that athletes who sweat heavily during prolonged training may have increased calcium loss and elevated requirements. In addition, limited evidence also suggests that amenorrheic athletes may require an additional 500 mg/day of calcium to maintain calcium balance, presumably because of the absence of adequate estrogen (Heaney et al., 1978).

Eumenorrheic athletes can meet calcium requirements by including several servings of dairy products or five to eight servings of calcium-containing plant foods daily (Table 20.3). Plant foods that are rich in well-absorbable calcium include low-oxalate green leafy vegetables, calcium-set tofu, fortified juice, soy milk, rice milk, textured vegetable protein, and certain legumes (Craig & Mangels, 2009). Sardines are also an excellent nondairy source of absorbable calcium. Laboratory studies have determined that the calcium bioavailability of most plant foods is as good as or better than cow's milk, which has a fractional absorption of 32% (Weaver et al., 1999; Zhao et al., 2005) (Figure 20.1). The exceptions include soymilk fortified with tricalcium phosphate, most legumes, nuts, and seeds which have a fractional absorption between 17% and 24% (Craig & Mangels, 2009; Zhao et al., 2005). Spinach, Swiss chard, beet greens, and rhubarb are not well-absorbed sources of calcium due to their high oxalate or phytate content which reduces the bioavailability of dietary calcium (Ross et al., 2010). In support of the efficiency of calcium-rich plant foods, a clinical trial observed that young vegetarians maintained positive calcium balance and appropriate bone resorption when calcium was provided either from dairy products or exclusively from plant foods, despite lower calcium intake on the plant-based (843 ± 140 mg) compared to the dairy-containing (1322 ± 303 mg) diet (Kohlenberg-Mueller & Raschka, 2003). Another trial in postmenopausal women found significantly lower urinary calcium excretion in those following a vegetarian diet compared to an omnivorous diet (3.2 ± 1.2 vs. 3.9 ± 1.3 mmol/24 hours), despite similar dietary calcium intake (Ball & Maughan, 1997).

Table 20.3 Calcium, magnesium and vitamin D content of selected foods

Food	Portion	Calcium (mg)	Magnesium (mg)	Vitamin D (IU)
Grain equivalents				
Bread, most enriched	1 slice (30 g)	43	7	0
Bread, whole wheat	1 slice (30 g)	20	24	0
Cereal, fortified	1 serving (30 g)	125–1000[a]	Varies	40–100[a]
Vegetables and legumes (including soy)				
Beet greens	1 cup[b] cooked	164	98	–
Black beans	1 cup[b] cooked	46	120	–
Broccoli	1 cup[b] cooked	62	32	–
Cabbage, pak-choi	1 cup[b] cooked	158	19	–
Cabbage, head	1 cup[b] cooked	46	12	–
Collard greens	1 cup[b] cooked	266	38	–
Chickpeas (garbanzo beans)	1 cup[b] cooked	80	79	–
Kale	1 cup[b] cooked	94	23	–
Lentils	1 cup[b] cooked	38	71	–
Mushrooms, sun-dried	1/2 cup[b] raw	2	5	192
Mushroom powder, Portobello	5 g	tr	tr	600
Mustard greens	1 cup[b] cooked	104	21	–
Okra	1 cup[b] cooked, sliced	62	58	–
Pinto beans	1 cup[b] cooked	79	86	–
Red kidney beans	1 cup[b] cooked	50	80	–
Soybeans	1 cup[b] cooked	175	148	–
Sweet potato	1 medium, baked	43	31	–
Tofu, firm (calcium-set)	1/2 cup[b]	861[a]	73	–
Tofu, regular (calcium-set)	1/2 cup[b]	434	37[a]	–
Turnip greens	1 cup[b] cooked	197	32	–
Fruits				
Grapefruit juice, fortified	1 cup[b]	350	30	0–100
Orange juice, fortified	1 cup[b]	350	27	100
Nuts and seeds				
Almonds	30 g	70	78	–
Cashews, peanuts, pecans, and pine nuts				
Nuts, sunflower seeds	30 g	5–20	22–71	–
Pumpkin seeds	30 g	12	151	–
Tahini	15 g	64	14	–
Walnuts	30 g	28	45	–
Milk, soy milk, cheese, and eggs				
Soy milk, fortified	1 cup[b]	368[a]	39[a]	100[a]
Cow's milk, skim	1 cup[b]	306	27	98
Cow's milk, 2%	1 cup[b]	285	27	98
Cheddar cheese	30 g	204	8	0
Mozzarella	30 g	207	7	0
Swiss	30 g	224	11	0
Egg yolk	1 each	1	22	20–40
Fish				
Cod liver oil	15 g	0	0	1360
Cod, Pacific	100 g	10	24	24
Cod, Atlantic	100 g	14	42	46
Salmon, farm-raised	100 g	15	30	250[c]
Salmon, wild	100 g	15	37	980[c]
Sardines, canned in oil	100 g	382	39	193
Tuna, yellow fin steak	100 g	4	42	82

(continued)

Table 20.3 *(Continued)*

Food	Portion	Calcium (mg)	Magnesium (mg)	Vitamin D (IU)
Tuna, light, canned in water	100 g	11	27	181
Tuna, white, canned in water	100 g	14	33	80
Others				
Margarine, fortified	15 g	tr[a]	0	8–80[a]
Molasses	15 g	41	48	–
Blackstrap molasses	15 g	200	–	–

Source: The USDA National Nutrient Database for Standard Reference http://www.ars.usda.gov/main/site_main.htm?modecode=12354500.

Values for most vegetables and legumes are for cooked, boiled, and drained without salt. Values for most nuts are for dry roasted without added oil or salt. Values for fish are for cooked, dry heat. Values for blackstrap molasses, mushroom powder, and calcium- and vitamin D-fortified food products obtained from the food label in the United States.

tr, trace.

[a]Varies by brand.

[b]1 cup = ~1/4 liter.

[c]From Lu et al. (2007).

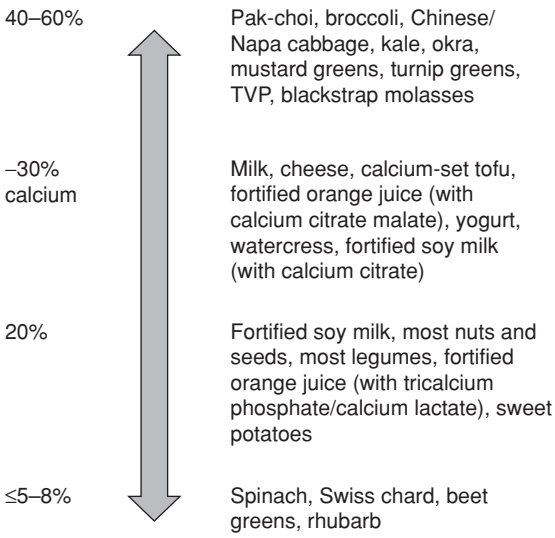

Figure 20.1 Fractional absorption of calcium-containing plant foods. Developed using references of Weaver et al. (1999), Zhao et al. (2005), and Craig and Mangels (2009).

Athletes not meeting their calcium intake may consider calcium supplementation (plus vitamin D, if appropriate) as a short-term option but should work toward consumption of calcium-rich foods daily as part of a well-balanced diet that is adequate in phosphorus, protein, magnesium, and vitamins A and K. Supplements may be particularly helpful when requirements are elevated due to amenorrhea or menopause. Calcium carbonate and calcium citrate are well-absorbed sources used in supplements (Ross et al., 2010); calcium carbonate is generally less expensive but requires gastric acid for optimal absorption. As was once thought, long-term supplementation with calcium carbonate (Minihane & Fairweather-Tait, 1998) or other forms including calcium chloride (Gaitan et al., 2011) does not inhibit absorption of nonheme iron or compromise iron status in iron-replete adults. Calcium supplements are better absorbed if taken with food (Heaney et al., 1989b) and in doses of 500 mg or less.

Calcium Toxicity and Adverse Effects

Excess calcium intake at the level required to induce toxicity is almost impossible to achieve from food alone, but excess calcium intake may occur as a result of calcium supplementation (including calcium-fortified foods) (Ross et al., 2010). While the classic "toxicity" state of hypercalcemia (total calcium >2.63 mmol/l) is observed with calcium or vitamin D excess, malignancy and primary hyperparathyroidism (Moe, 2008) are more common causes. Clinical signs and symptoms of hypercalcemia include anorexia, weight loss, polyuria, heart arrhythmias, fatigue, and soft tissue calcifications. Symptoms vary depending on the magnitude of the hypercalcemia and how rapidly it develops (Jones, 2008). Hypercalciuria typically occurs with severely

elevated hypercalcemia (>12 ng/dl). Although calcium supplements are generally considered safe, research has identified an association between calcium supplementation and increased risk for cardiovascular events (Bolland et al., 2010), kidney stones (nephrolithiasis) (Jackson et al., 2006), and prostate cancer (Raimondi et al., 2010). Thus, while it is important for athletes to meet calcium requirements, they should avoid oversupplementation.

Vitamin D

Vitamin D Synthesis and Sources

Although vitamin D is thought of as a "vitamin," physiological requirements can be met entirely through endogenous synthesis (Holick, 2007; Zittermann, 2003). When the skin is exposed to ultraviolet-B radiation (UVB, wavelength of 290–315 nm), 7-dehydrocholesterol—present in the plasma membrane of epidermal and dermal cells—is converted to pre-vitamin D_3 (precholecalciferol). Pre-vitamin D_3 then undergoes thermal isomerization to a more thermodynamically stable vitamin D_3 (cholecalciferol) over a period of several days. Vitamin D_3 moves into the dermal capillary bed and into circulation with the assistance of vitamin D-binding protein (VDBP) and undergoes obligate hydroxylation to 25(OH)D in the liver by enzymes of the cytochrome P-450 system. Further hydroxylation in kidney tubules to the hormonally active form, $1,25(OH)_2D$, by the cytochrome enzyme CYP27B1 is driven by PTH when serum calcium and phosphate concentrations drop below the physiological range. In addition, many extra-renal cells (and tissues), including macrophages, brain, colon, breast, and others, have the enzymatic machinery (1-α-hydroxylase) to locally produce $1,25(OH)_2D$ (Cannell et al., 2008a). Because sunlight is necessary to activate this process, any factor that limits the amount or quality of sun exposure can compromise vitamin D status (Box 1). This includes the winter season at locations distant from the equator (latitude >~35°) (Cannell et al., 2008a; Holick, 2007) because the solar zenith angle prevents sufficient UVB photon from reaching the earth's surface.

Vitamin D can also be obtained in the diet from limited natural and fortified sources including fatty fish, sun-dried mushrooms, fortified milk and other fortified foods (Table 20.3). Dietary vitamin D includes both D_3 (cholecalciferol) and D_2 (ergocalciferol, derived from UVB exposure of fungi and yeast ergosterols). Both forms are absorbed into intestinal mucosal cells from micelles in association with lipid (and the aid of bile salts) where they are incorporated into chylomicrons and enter circulation via the lymphatic system. As such vitamin D is thought to be most efficiently absorbed when consumed with meals containing fat (Mulligan & Licata, 2010; Ross et al., 2010). The fractional absorption of vitamin D is ~50% except in individuals with malabsorption syndromes (Basu & Donaldson, 2003) including pancreatic insufficiency and impaired bile secretion. Following absorption, dietary vitamin D is transported to the liver by VDBP and metabolized similar to endogenous vitamin D_3.

Indices of Vitamin D Status

There is general agreement that circulating 25(OH)D concentration is the best indicator of vitamin D status (Cannell et al., 2008a; Hollis, 2005, 2007, 2009; Zittermann, 2003). The circulating concentration of $1,25(OH)_2D$, the active hormone, is sensitive to serum PTH concentration (as reviewed earlier) and is not reflective of vitamin stores. 25(OH)D also has a longer half-life (2–3 weeks) (Zittermann, 2003) than $1,25(OH)_2D$ (<4 h) and is better reflective of intestinal calcium absorption (Heaney et al., 2003; Zittermann, 2003) than $1,25(OH)_2D$. Extra-renal tissues are dependent on adequate serum 25(OH)D for local (autocrine/paracrine) synthesis of $1,25(OH)_2D$.

Definitive thresholds for vitamin D status have not yet been scientifically established. Most vitamin D researchers currently define vitamin D deficiency as a serum 25(OH)D concentration <50 nmol/l (Holick, 2007; Holick et al., 2011), insufficiency as a concentration <75 (Holick, 2007; Holick et al., 2011) to <80 nmol/l (Hollis, 2005), and toxicity as a concentration >375 nmol/l when coupled with an elevated serum calcium concentration (Cannell et al., 2008a; Hollis, 2005, 2007). While the cutoff for deficiency is the approximate concentration at which PTH rises abruptly and the cutoff for

insufficiency is the concentration where PTH plateaus and calcium absorption is maximized (Craig & Mangels, 2009; Holick, 2009; Hollis, 2005; Zittermann, 2003), Heaney (2011) recently argued that the distinction between deficiency and insufficiency is not useful or necessary. Heaney strongly believes that serum 25(OH)D concentrations below 120 nmol/l are associated with preventable disease and are therefore indicative of deficiency. In contrast, the recently revised dietary guidelines of the United States and Canada were established using 50 nmol/l as adequate using the justification that serum concentrations above 75 nmol/l have not consistently been associated with increased benefit. Other experts have defined concentrations between 100 to 125 and 250 nmol/l as optimal, which are thought to be the concentration where the human genome evolved (Cannell et al., 2008a; Hollis, 2005). Increasing evidence also suggests that risk for chronic and acute illness may be reduced when vitamin D is maintained in the presumed optimal range (Cannell et al., 2008a; Heaney, 2011).

Vitamin D Intake and Status of Athletes

It is well recognized that suboptimal vitamin D status is widespread among the general population worldwide. Among athletes, the prevalence of deficiency varies by sport, training location, and skin color (Larson-Meyer & Willis, 2010) and appears to be lower in the winter and among athletes who train indoors versus outdoors. Studies to date have found the highest prevalence of vitamin D deficiency in athletes training in both Finland (Lehtonen-Veromaa et al., 1999) and the Middle East (Hamilton et al., 2010) and documented the best status in some US college athletes in the fall season (Table 20.4).

Although the most probable reason for suboptimal vitamin D status is insufficient UVB exposure (Table 20.4), poor vitamin D intake may contribute to the problem. Dietary assessment studies over the last 15 years have found that athletes do not come close to meeting the dietary recommendations of most countries (Figure 20.2), including the newly revised US recommended daily allowance (RDA) of 600 IU (Larson-Meyer & Willis, 2010). Studies, in fact, have reported average vitamin D intakes of 100 IU (Rankinen et al., 1998) to slightly more than 250 IU (Halliday et al., 2011) among athletic groups across the globe. A recent study found that only 5% of college athletes consumed the US RDA from food alone (Halliday et al., 2011).

Concerning dietary practices, vitamin D intake is found to be low in some vegetarians (Davey et al., 2003) and vegans (Outila et al., 2000) and in those who consume few dairy products or fortified foods. The high phytate and fiber content of vegetarian diets may also decrease vitamin D absorption and/or increase vitamin D loss by impairing intrahepatic vitamin D circulation (Batchelor & Compston, 1983). In contrast, consumption of fish at least four times per week (Nakamura et al., 2000)—particularly wild fatty fish—helps prevent vitamin D deficiency.

It is important to mention, nevertheless, that assessment of intake is difficult due to the variability of vitamin D in natural sources, including fish and fish products, and the failure of the nutrient databases to keep up with food fortification (Ross et al., 2010). One study found that only 20% of milk samples in the United States and 27% of those in Canada contained 81–120% of the amount of vitamin D stated on the label (Chen et al., 1993), while a high percentage contained either less than 5% or more than 120% of the labeled content. This contributes to the probable inaccuracy of estimates of vitamin D intake in athletes and the general population.

In addition to dietary sources, intake of vitamin D-containing supplements, including a multivitamin, may result in higher but not necessarily optimum serum 25(OH)D concentration (Lehtonen-Veromaa et al., 1999). A study from my lab found that ~33% of college athletes take a multivitamin at least 4 days/week and that vitamin D status correlates with multivitamin intake in the winter (Halliday et al., 2011). In contrast, 75% never take larger supplemental doses (>1000 IU). Although multivitamins in the United States typically contain 400–600 IU of vitamin D, preparations with higher levels are available and may be an effective way to improve status in athletes living in certain countries.

Increasing evidence has also suggested that excess adiposity increases risk for low vitamin D status. In

Table 20.4 Vitamin D status in athletes living in various geographic locations

Reference	Athlete population	Location	Season	25(OH)D (nmol/l)	Vitamin D status (38 nmol/l)
Bannert et al. (1991)	85 male and female competitive gymnasts (8–27 years)	Magdeburg, East Germany (52.5°N)	nr	~50–60 (est) (0–200)	37% <25
Bescos Garcia and Rodriguez Guisado (2011)	21 male elite basketball players (24 ± 4 years)	Barcelona, Spain (41.2°N)	Competitive season	Blacks 23.7 ± 7.3 Whites 55.3 ± 16.5	90.5% <75 57.1% <50
Constantini et al. (2010)	52 male and 46 female athletes and dancers (10–30 years)	Jerusalem, Israel (31.5°N)	Random sample	63.3 ± 20.8	73% <75
Hamilton et al. (2010)	93 sportsmen (21 years)	Doha, Qatar (25.4°N)	nr	27.5 (est) (17.5–70)	91% <50 58% <25
Halliday et al. (2011)	18 male and 23 female college athletes (18–24 years)	Laramie, WY 73 (41.3°N)	Fall Spring Winter	122.5 ± 41.5 76.3 ± 23.5 104.8 ± 36.5	12.2% <80 2.4% <50 63.6% <80 3.0% >50 20% <80 4% <50
Helle and Bjerkan (2011)	78 male and 55 female elite athletes (24 ± 4 years)	Oslo, Norway (59.6°N)	Fall	82 ± 30	35%<80 13<50
Jonvik et al. (2011)	48 female elite athletes (23 ± 4 years)	Oslo, Norway (59.6°N)	Fall	99 ± 32	25% <80 2% <50
Lehtonen-Veromaa et al. (1999)	66 female gymnasts, 65 runners, 60 controls (9–15 years)	Turku, Finland (60.4°N)	Winter Summer	34 ± 14 63 ± 15	68% <38 13% <10 11.6% <38
Lovell (2008)	18 female gymnasts (10–17 years)	Canberra, Australia (35.3°S)	Fall (early May)	56 (9–84.2)	83% <75 33% <50
Maimoun et al. (2006)	7 male competitive road cyclists (20–39 years)	Montpellier, France (43.6°N)	Competitive season	81.3 ± 16	nr
Shindle et al. (2011)	31 white and 58 black American football players (21–32 years)	Various locations	Spring	Blacks 51 Whites 75.8	80.9% <80 30.3% <50
Storlie et al. (2011)	39 male collegiate outdoor sport athletes (18–33 years)	Ellenburg, WA (46.9°N)	Fall	127.7 ± 46.6	25% <80 2.5% <50
Willis et al. (2008)	9 male and 10 female distance runners (19–45 years)	Baton Rouge, LA (30.5°N)	Random sample	96.8 ± 43 (39–202.5)	42% <80 0.5% <50

Data are presented as means or, if available, means ± SD. The range, if available, is given in parenthesis.
est, estimated; nr, not reported.

non-obese individuals, a certain fraction of both D_3 and D_2 is stored in subcutaneous fat and is released during the winter (or other periods of reduced UVB exposure) (Holick, 2007). In overweight and obese individuals, however, vitamin D uptake and clearance by adipose tissue are enhanced (Liel et al., 1988; Sabetta et al., 2010), and this tissue appears to sequester vitamin D deep in subcutaneous fat (Wortsman et al., 2000). This is consistent with a higher volume of distribution in those with higher adiposity which is thought to decrease release of D_3 from adipose storage (Sabetta et al., 2010).

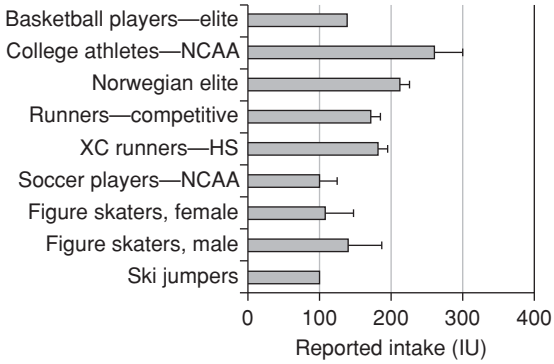

Figure 20.2 Vitamin D intake in athletes from a variety of sports. NCAA, National Collegiate Athletic Association; XC, cross-country; HS, high school. Developed using references of Rankinen et al. (1998), Lehtonen-Veromaa et al. (1999), Ziegler et al. (2001), Halliday et al. (2011), Clark et al. (2003), Helle and Bjerkan (2011), and Bescos Garcia and Guisade (2011).

In nonathletes, serum 25(OH)D concentration is lower in obese compared to non-obese individuals (Hypponen & Power, 2007; Snijder et al., 2005) and inversely associated with body fat percentage (Snijder et al., 2005). Our studies have found that 25(OH)D concentration correlates negatively with body fat percentage within the body fat range of mixed sport college athletes (Figure 20.3), but this relation is not apparent in the winter, when 25(OH)D concentration drops precipitously (Halliday et al., 2011). Additional studies are needed to confirm these findings and address whether athletes with high adiposity are at increased risk of deficiency.

Functions of Vitamin D

Recent research has found that vitamin D functions as a modulator of several hundred genes involved

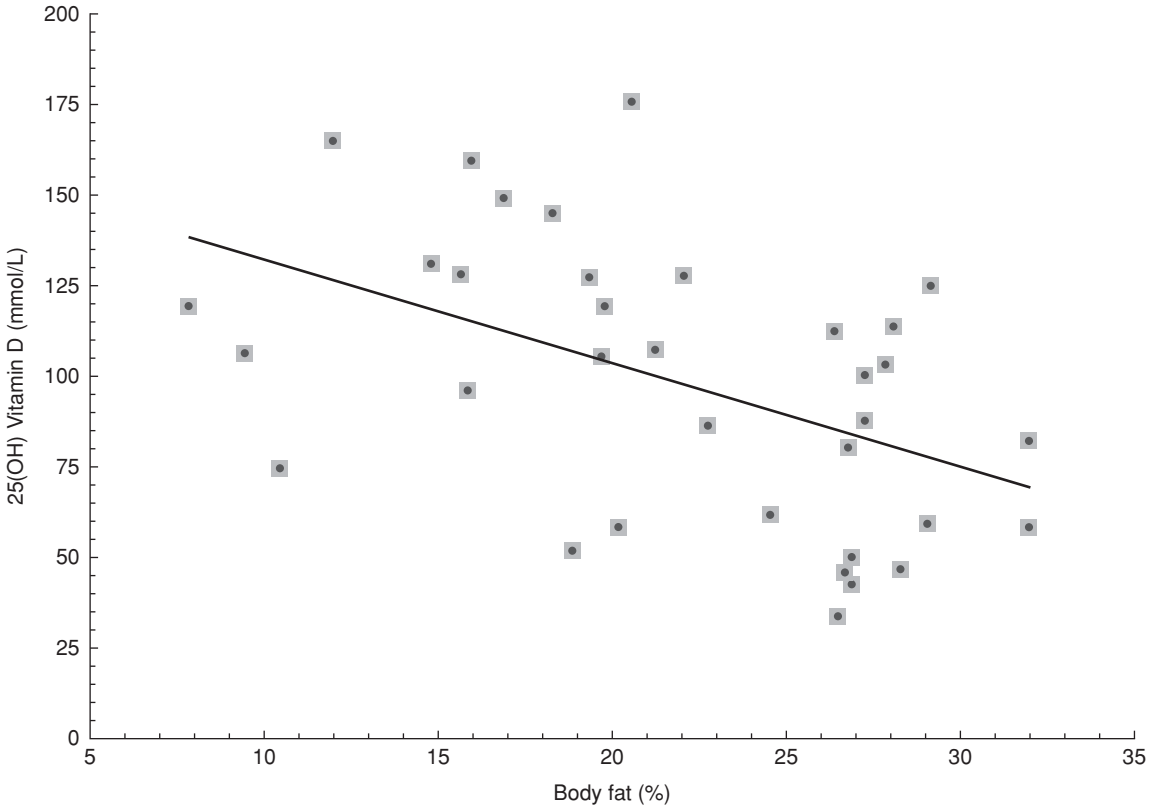

Figure 20.3 Relation between body fat and serum 25(OH)D concentration in the fall in 35 collegiate athletes, $r = -0.49$, $p = 0.003$. Unpublished data from Larson-Meyer.

in cellular growth, immune function, and protein synthesis (Cannell et al., 2009; Holick et al., 2011), which is estimated to be ~5% of the human genome (Zella et al., 2008). In this role, cellular-derived $1,25(OH)_2D$ interacts with its nuclear vitamin D receptor (VDR), which is present in most tissues and cells including intestine, bone, immune cells (Holick, 2007), and skeletal muscle (Bischoff-Ferrari et al., 2004a). The vitamin D–VDR complex then binds with the retinoic acid X receptor, which is recognized by specific vitamin D-response elements on the gene sequence, to regulate expression of specific genes (Holick, 2007). As reviewed earlier, a higher serum 25(OH)D concentration increases the substrate for paracrine/autocrine 1-α-hydroxylase, thereby promoting adequate intracellular $1,25(OH)_2D$ concentration for gene expression modulation. Higher 25(OH)D concentration in circulation is imperative because 25(OH)D is used directly by most tissues, containing the 1-a-hydroxylase, to make $1,25(OH)_2D$.

Bone Health Vitamin D influences bone health by upregulating expression of genes that enhance intestinal calcium absorption, renal tubular resorption (in association with elevated PTH), and osteoclastic activity (Holick, 2007). A powerful example is found with fractional calcium absorption, which is ~10–15% in the vitamin D-deficient state and greater than 30% when serum 25(OH)D concentration is at least 75 nmol/l (Heaney et al., 2003). Much of this effect is due to vitamin D-enhanced expression of intestinal calcium binding and epithelial calcium channel proteins, which include calbindin. Serum 25(OH)D concentration is also found to correlate with bone mineral density and/or bone mineral content in the hip and lumbar spine in both younger and older women (Bischoff-Ferrari et al., 2004b).

Several studies have provided evidence that sufficient vitamin D status is important for bone health and prevention of bone injury in athletic populations. One study in young Finnish military recruits found a 3.6 times higher stress fracture risk in recruits with serum 25(OH)D concentration below 75 nmol/l (Ruohola et al., 2006). In another, stress fractures were associated with higher PTH concentration (which are directly influenced by calcium intake and vitamin D status) even though 25(OH)D concentration was not directly associated (Valimaki et al., 2005). A randomized double-blind trial in US female naval recruits found that stress fracture incidence was reduced by 20% following 8 weeks of supplementation with 800 IU of vitamin D plus 2000 mg calcium (Lappe et al., 2008). Stress fractures can be a common problem for many athletes and represent a serious obstacle to training.

Skeletal Muscle Function Vitamin D, specifically $1,25(OH)_2D$ is thought to modulate skeletal muscle function through genomic and nongenomic events (Barker et al., 2011; Hamilton, 2010). The genomic events are currently understood to occur through modulation of the nuclear receptor which, upon binding to vitamin D, induces gene transcription and protein synthesis, ultimately influencing muscle cell proliferation and differentiation, calcium uptake, and phosphate transport across the sarcolemma. The nongenomic responses involve modulation of calcium uptake across the sarcolemma and the activation of mitogen-activated protein kinase signaling pathways. In agreement, animal studies have found that vitamin D deficiency induces atrophy of fast-twitch muscle fibers, impairs sarcoplasmic calcium uptake, and prolongs time to peak contractile tension and relaxation (Hamilton, 2010).

Skeletal Muscle Pain and Weakness. Musculoskeletal pain and weakness are well-established but often forgotten symptoms of vitamin D deficiency that resolve with repletion (Cannell et al., 2008a; Holick, 2007; Zittermann, 2003). A study conducted at an inner city clinic in the northern United States found, for example, that 93% of those with persistent nonspecific musculoskeletal pain had 25(OH)D concentrations <50 nmol/l and 28% had concentrations <20 nmol/l (Plotnikoff & Quigley, 2003). Another in the North West and Midlands areas of England found that low 25-OH vitamin D concentrations of <25 nmol/l were 3.5 times more common among women with widespread musculoskeletal pain, which was more prevalent in South Asian than European women (Macfarlane et al., 2005). Furthermore, several open (i.e., not randomized) trials in nonathletes have found improved muscle pain with

vitamin D_3 supplementation (Al Faraj & Al Mutairi, 2003; de Torrente de la Jara et al., 2004). In one of these trials, 3 months of treatment with 5000–10,000 IU/day resulted in clinically improved reductions in back pain in all patients with initially low vitamin D stores (Al Faraj & Al Mutairi, 2003).

Skeletal Muscle Performance. Currently published studies have not yet evaluated whether low vitamin D status directly impairs muscle strength or performance in athletes. Although provocative evidence from the Russian and German literature at the turn of the twentieth century suggests that UVB irradiation positively affects athletic performance (Cannell et al., 2009) , these studies were not conducted using the rigorous scientific standards used today and also did not assess vitamin D status following irradiation. Two recent studies involving nonathletes found positive associations between serum 25(OH)D concentration and aerobic fitness (Mowry et al., 2009), and jump height, velocity, and power (Ward et al., 2009) in young American women and British school girls, respectively, with severely deficient status. In the British study, 1 year of vitamin D supplementation, which increased 25(OH D concentrations from 18 to 56 nmol/l, was found to improve skeletal muscle movement efficiency but not jumping performance (Ward et al., 2010). Although it is not yet established whether there is a threshold above which skeletal muscle functions optimally, a study in 4100 older individuals—who were both active and sedentary—observed steady increases in lower-extremity neuromuscular performance with increasing 25(OH)D concentrations that appeared to plateau around 94 nmol/l(Bischoff-Ferrari et al., 2004c).

Rehabilitation. The importance of vitamin D status in musculoskeletal rehabilitation following injury or surgery is an emerging area of interest. A recent study of patients in a general rehabilitation unit found that vitamin D deficiency delayed inpatient rehabilitation (Kiebzak et al., 2007). More specific to orthopedic injury, another found that low vitamin D influenced strength and recovery in younger, recreationally active individuals after anterior cruciate ligament repair (Barker et al., 2011). In this study, those with 25(OH)D concentration below 75 nmol/l recovered more slowly than those with concentrations above 75 nmol/l and had significantly dampened increases in peak isometric force. Studies in vitamin D-deficient hospitalized patients have observed that treatment with at least 1000 IU/day of vitamin D normalizes muscle strength and improves muscle function in 1–2 months (Zittermann, 2003). A randomized trial in older female stroke patients found that supplementation with 1000 IU/day vitamin D increased the relative number and size of type II muscle fibers and improved muscle strength. Certainly these results are of interest because muscle weakness challenges physical rehabilitation in the sports medicine setting.

Immunity and Inflammation Vitamin D is also recognized as an important regulator of inflammation and immunity. Vitamin D upregulates gene expression of broad spectrum antimicrobial peptides (AMP)—important regulators in innate immunity—and also downregulates expression of inflammatory cytokines (Liu et al., 2006; Wang et al., 2004). AMP is secreted by cells of the innate immune system, including macrophages, monocytes, natural killer cells, and epithelial cells in the respiratory tract (Gombart et al., 2005), and exerts its effect by compromising the integrity of the cell membrane of invading pathogens. In addition, vitamin D has an immunomodulatory effect on T and B lymphocytes (Zittermann, 2003) thereby playing an important role in acquired immunity. Vitamin D's influence on acquired immunity, however, is beyond the scope of this chapter.

Innate Immunity. The synthesis and release of AMPs—such as cathelicidin—is triggered when invading pathogens are recognized by *toll-like receptors* (TLRs). Liu et al. (2006) demonstrated that TLR stimulation triggers conversion of 25(OH)D to $1,25(OH)_2D$, and induces expression of the VDR, which in turn promotes the synthesis of cathelicidin. African Americans with reduced 25(OH) D concentration exhibit decreased ability to synthesize cathelicidin, which may explain why they have historically been more susceptible to tuberculosis. Additional studies suggests that vitamin D_3 acts to trigger the oxidative burst in activated macrophages (Sly et al., 2001) and that a single dose

of vitamin D_3 (100,000 IU) can enhance the innate immune response and restrict growth of mycobacteria (Martineau et al., 2007). Cannell et al. (2006) suggested that individual susceptibility to the influenza virus may be tied to vitamin D status and the ability of vitamin D to regulate AMP expression in epithelial cells of the respiratory tract. In this manner, seasonal fluctuations in vitamin D status could explain, in part, the seasonality of viral respiratory infections.

These findings may be directly applicable to athletes. Some research suggests that prolonged intense training has a suppressive effect on innate immune function and increases risk of upper respiratory tract infection (Nieman, 2000; West et al., 2006). In a study in college athletes, vitamin D status in the winter and spring was negatively associated with documented frequency of acute upper respiratory illness. The breakpoint for contracting single illness appeared to occur at ~95 nmol/l, such that all athletes with circulating stores that were lower than the breakpoint had one or more episodes of illness, whereas those with higher stores had one or fewer episodes. Another study in 800 Finnish military soldiers found that those with serum 25(OH)D concentrations less than 40 nmol/l had 63% more absences from duty due to respiratory illness than those with higher stores (Laaksi et al., 2007). These results are in agreement with a study in the general population which found that maintaining a serum 25(OH)D of at least 96 nmol/l reduced the risk of developing an acute viral respiratory tract infection by twofold and also markedly reduced days missed from work (Sabetta et al., 2010). Certainly, reducing the burden of illness would be a benefit to athletic training and performance.

Inflammation. Vitamin D may also work through the immune system to control inflammation, which results from the accumulation of fluid and immune cells in injured tissue. Vitamin D has been shown to increase the production of several anti-inflammatory cytokines including transforming growth factor and interleukin-4 (Cantorna et al., 1998) and reduce the production of several proinflammatory cytokines such as interleukin-6 (IL-6) , interferon-γ, interleukin-2, and tumor necrosis factor (TNF-α) (Muller et al., 1992; Rigby et al., 1987; Zhu et al., 2005)

Although there is limited evidence to date which directly links vitamin D insufficiency to sports-related inflammation, several of the proinflammatory cytokines, particularly IL-6, are elevated following a single bout of exercise, which unexplainably occurs to a greater degree in some well-trained athletes compared to others (Edwards et al., 2006). Elevated concentrations of the proinflammatory cytokines are hypothesized to be involved in overtraining syndrome (Smith, 2000). In this cascade of events, musculoskeletal trauma induces the release of IL-6, which stimulates the conversion of monocytes to macrophages in circulation. Macrophages in turn produce large quantities of other proinflammatory cytokines including TNF-α and additional IL-6 which coordinate the whole-body (or systemic) inflammatory response via the brain/CNS. While it is not completely known if and how vitamin D influences the inflammatory cascade, animal studies have found that vitamin D supplementation has the potential to reduce the inflammatory cycle (Cantorna et al., 2000).

Currently, limited evidence directly links compromised vitamin D status with increased risk or severity of sports-related inflammation or injury or with overtraining syndrome. The earliest recognition of such a link was alluded to in a 1950s German report which observed that athletes experienced a significant reduction in chronic pain due to sports injuries following an extensive 6-week program of irradiation with a "central sun lamp." Two not yet published studies are also in support. One in distance runners found that the proinflammatory marker TNF-α is correlated with vitamin D status (assessed at least 36 hours after training) which becomes abruptly elevated in circulation as concentration drops below 80–85 nmol/l (Willis, 2008). Another in American professional football athletes found that players who sustained a muscle injury had significant lower 25(OH)D concentration than those without muscle injury (50 vs. 63 nmol/l) (Shindle et al., 2011). In contrast, a link between vitamin D status and overtraining injuries was not observed in a small group of NCAA Division I college athletes, who had few injuries over the course of the year (Halliday et al., 2011).

Vitamin D Requirements for Athletes

According to the newly revised US and Canadian Dietary Guidelines, the RDA for vitamin D is 600 IU for children and adults up to 70 years of age; and 800 IU for adults over 70 (Ross et al., 2010). While these requirements are higher than those of most countries (Table 20.2), many vitamin D experts believe that the RDA—which was established based exclusively on bone health (Ross et al., 2010)—is still not high enough to support nonskeletal health benefits (Heaney & Holick, 2011; Holick et al., 2011) and may also not be high enough to optimize athletic performance. In fact, as discussed below, the Endocrine Society suggests that individuals with limited sun exposure require two to three times the US RDA (1500–2000 IU/day) to keep 25(OH)D concentrations in the sufficient range (Holick et al., 2011). Interestingly, in the United Kingdom, The DRI defined by the 1991 report did not set a Reference Nutrient Intake for Vitamin D for those 4 to 65 years old who receive adequate sunlight exposure. There is no evidence, however, to suggest that the vitamin D needs of athletes differ from those of the general population.

Because there are limited foods which contain vitamin D, most athletes will need to meet requirements through regular supplementation, sensible sun exposure, or a combination of dietary intake, sun exposure, and supplementation. Regular consumption of vitamin D-fortified foods or a daily multivitamin alone is not likely to maintain sufficient status (>75–80 nmol/l) in the absence of UVB exposure.

Vitamin D Intoxication

Although intoxication from excess intake or supplementation is extremely rare, it can be caused by ingestion (intentional or unintentional) of extremely high supplemental doses (Araki et al., 2011; Jacobsen et al., 2011). Doses of 150,000 to 2,604,000 IU/day for 2 years, for example, are found to raise 25(OH)D concentrations to 1217 nmol/l and induce both hypercalcemia and hyperphosphatemia (Koutkia et al., 2001a). Doses of 10,000 IU/day for up to 5 months, on the other hand, do not cause toxicity (Vieth, 2004) and appear to be safe. A recent report described a case of vitamin D intoxication that was caused by manufacturer error in which 188,640 IU of vitamin D_3 had been added to a vitamin supplement instead of the intended 400 IU (Klontz & Acheson, 2007). In this case, the patient was admitted after 2 months of supplementation with severe hypercalcemia (>3.75 mmol/l) and elevated serum 25(OH)D (1171 nmol/l) concentration, which presented with classic signs and symptoms of toxicity including fatigue, constipation, back pain, forgetfulness, nausea, and vomiting. Prolonged hypercalcemia is known to produce soft tissue calcification and resultant renal and cardiovascular damage, hypertension, and heart rhythm abnormalities as previously discussed.

Knowledge concerning vitamin D toxicity is important to consider in the athletic population because some athletes, coaches, and trainers believe that "if a little is good, more is better." Vitamin D intoxication from UVB exposure, on the other hand, is not possible because excess previtamin D_3 photoisomerizes to biologically inert photoproducts, including lumisterol and tachysterol, with prolonged UV exposure (Ross et al., 2010). Vitamin D_3 can also be converted to inactive forms.

Clinical Assessment, Evaluation, and Treatment

Routine screening for adequacy of calcium intake and vitamin D deficiency may ensure that poor status does not compromise the health or performance of competitive athletes. Bi- or triannual screening in association with training periodization, season, and probable peak (late summer/early fall) and nadir (late winter) 25(OH)D concentration may be most useful to the sports medicine team. If routine screening is not possible, athletes with a history of stress fracture, frequent illness, bone and joint injury, skeletal weakness or pain, or signs of overtraining should be targeted. Careful attention should also be given to athletes with restrained eating patterns that spend the majority of time indoors (gymnasts, dancers, figure skaters, and wrestlers) as they may be at

increased risk for both vitamin D deficiency and poor calcium intake. Vegan athletes may also warrant screening considering the potential for reduced bioavailability of vitamin D_2 (vegan vitamin D) and limited intake of natural and fortified calcium and vitamin D-rich foods.

Steps for assessing vitamin D status are outlined in Table 20.5. Although serum 25(OH)D concentration using a reliable assay is the most important parameter, PTH along with other biochemical indices may provide additional information when bone density is low, stress fracture (or reaction) is evident, and/or vitamin D and calcium intakes are severely restricted. PTH concentration typically increases drastically as 25(OH)D concentration falls below 25–50 nmol/l (Holick, 2009) and is independently related to bone density (Halliday et al., 2011) and stress fracture risk (Ruohola et al., 2006). The history should address bone health, idiopathic muscle pain and weakness, overtraining injury, and frequency of illnesses (including respiratory tract infections). Although the physical exam may be unremarkable, assessment of muscle weakness and bone pain may be useful along with a bone density scan, typically of the hips and/or lumbar spine. Documentation of current and recent medications is important because many medications interfere with vitamin D absorption or metabolism (Cannell et al., 2008a; Holick, 2007).

Dietary assessment should focus on estimating intake of both calcium and vitamin D. This information can be obtained by evaluating consumption frequency of natural and fortified sources of both nutrients along with supplements. In many cases, dietary assessment should also include assessment of magnesium and other nutrients important to muscle function and bone health (including caffeine and vitamins A, C, and K). Suboptimal magnesium intake is common in the western diet, unless the athlete consumes ample nuts, seeds, legumes, whole grains, and green leafy vegetables, and may influence bone and muscle function (Nielsen & Lukaski, 2006) (see Table 20.3 for selected sources).

Following a detailed assessment, recommendations for achieving/maintaining optimal vitamin D status can be individualized to each athlete's current 25(OH)D concentration, clinical symptoms, diet, lifestyle habits, and belief system. The recommendation to obtain 5 (in very fair skinned) to 30 (in darker skinned) minutes of sunlight exposure to arms, legs, and back several times a week at close to solar noon without sunscreen (Cannell et al., 2008a; Holick, 2007) usually leads to sufficient vitamin D synthesis. Fair-skinned individuals sunbathing in a bathing suit, for example, produce 10,000 to 20,000 IU of vitamin D in less than 30 minutes (Hollis, 2005).

Athletes with insufficient status require supplementation with at least 1500–2000 IU/day vitamin D to keep 25(OH)D concentrations in the sufficient range (Holick et al., 2011). A rule of thumb—based on a meta-regression analysis of 16 supplementation trials (Cranney et al., 2008)—is to increase supplemental vitamin D by 1000 IU for every 25–50 nmol/l elevation of 25(OH)D desired. For example, a "normal-weight" athlete with a serum 25(OH)D concentration of 50 nmol/l would require an additional 2000 IU daily to increase stores to 100 nmol/l over 3–4 months. Higher doses may be required in athletes who have darker skin, and/or excess adiposity, malabsorption syndromes, or who take medications affecting vitamin D metabolism. Athletes who live or train at latitudes above 35°N or 35°S should supplement during winter, even if they maintain adequate stores during non-winter seasons. A recent study found that common genetic variants in vitamin D-binding protein (the serum protein that binds vitamin D) also predict differences in response of serum 25(OH)D concentration to D_3 supplementation (Fu et al., 2009) and may help explain why some athletes do not respond as well to oral supplementation.

To more rapidly replenish stores, athletes with deficient status may benefit from short-term, high-dose "loading" regimens under supervision of a physician. The Clinical Practice Guidelines of the Endocrine Society suggest that adults who are deficient be treated with 50,000 IU of vitamin D_2 or D_3 for at least 8 weeks (or its equivalent of 6000 IU daily) to achieve a serum concentration of 25(OH)D above 75 nmol/l followed by maintenance therapy of 1500–2000 IU/day (Holick et al., 2011). High-dose treatment with cod liver oil is not recommended because it contains high amounts of vitamin A,

Table 20.5 Clinical assessment of vitamin D status

Anthropometrics and biological factors
- Age
- Body fat percentage and body mass index
- Weight history

Biochemical (laboratory) data
- 25(OH)D concentration
- Others (particularly if 25(OH)D is low or high: parathyroid hormone, alkaline phosphatase, serum calcium, serum phosphorus, serum magnesium, thyroid stimulating hormone)

Clinical

History
- Stress and other bone fractures
- Bone pain
- Muscle pain, weakness, "heaviness in legs"
- Chronic injury
- Frequent viral and bacterial illness
- Photosensitivity
- Skin cancer/melanoma
- Family history skin CA

Medications
- Anticonvulsants, corticosteroids, cimetidine, theophylline, orlistat, antituberculosis agents (may decrease vitamin D status)
- Thiazide diuretics, atorvastatin, other statins (may increase circulating vitamin D)
- Sulfonamides, phenothiazines, tetracyclines, psoralens (increase) photosensitivity; may signal sun avoidance

Physical exam
- Idiopathic musculoskeletal pain
- Muscle weakness (proximal limbs)
- Undue pain on sternal or anterior tibial pressure
- Lower limb deformities (knock knees, bowed legs)
- Bowel function (steatorrhea)
- Skin pigmentation (or type)/hair color
- Contraindications to sunlight (albinism, porphyrias, xeroderma pigmentosum)

Dietary intake
- Vitamin D
- Vitamin D-containing supplements (including multivitamin)
- Calcium
- Phosphate (nutritional supplements and soda consumption; may elevate PTH) (Zittermann, 2003)
- Magnesium (commonly low in Western diet; important for muscle and bone health; deficiency may mask elevated PTH)
- Other nutrients: vitamins A, C and K, omega-3/omega-6 fatty acids

Lifestyle and environmental
- Training regimen
- Training environment in relation to sunlight exposure
- Training latitude, altitude, climate
- Sunscreen use (sun protection factor of 15 ↓ synthesis capacity by >98%)
- Uniform or athletic clothing worn and SPF of clothing
- Leisure sun exposure (frequency, duration, and time of day)
- Tanning bed use (must emit UVB)
- Bathing habits after sun exposure (may rinse off skin cells containing newly synthesized vitamin D)
- Belief system (concerning sunlight exposure)

Source: From Larson-Meyer and Willis (2010), with permission from *Current Sports Medicine Reports*.

which can antagonize the action of vitamin D (Cannell et al., 2008b). Although controversial, tanning beds which emit UVB radiation have been used to maintain 25(OH)D concentration within the normal range and resolve musculoskeletal pain and weakness associated with deficiency (Koutkia et al., 2001b). Tanning bed use has also been shown to help healthy individuals (Tangpricha et al., 2004) and athletes maintain adequate vitamin D stores (Halliday et al., 2011).

Conclusion

Given the established role of vitamin D in bone health and the more recently recognized role in immunity, inflammation, and chronic disease prevention, sports nutritionists and physicians should routinely assess vitamin D status. Recent research has provided evidence to suggest that maintaining adequate vitamin D status may reduce risk for stress fracture, acute infection, inflammation, and impaired muscle function. Additional research is needed to determine whether insufficient vitamin D status increases risk for injury and whether vitamin D supplementation to correct deficiency and insufficiency can affect overall health, training, and performance in athletes.

References

Al Faraj, S. & Al Mutairi, K. (2003) Vitamin D deficiency and chronic low back pain in Saudi Arabia. *Spine* 28, 177–179.

Anonymous (2004). *Nordic Nutrition Recommendations 2004: Integrating Nutrition and Physical Activity*, 4th edn. Nordic Council of Ministers, Copenhagen, Denmark.

Araki, T., Holick, M.F., Alfonso, B.D., et al. (2011) Vitamin D intoxication with severe hypercalcemia due to manufacturing and labeling errors of two dietary supplements made in the united states. *Journal of Clinical Endocrinology and Metabolism* 96, 3603–3608.

Baker, L.B., Stofan, J.R., Lukaski, H.C., et al. (2011) Exercise-induced trace mineral element concentration in regional versus whole-body wash-down sweat. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 233–239.

Ball, D. & Maughan, R.J. (1997) Blood and urine acid–base status of premenopausal omnivorous and vegetarian women. *British Journal of Nutrition* 78, 683–693.

Bannert, N., Starke, I., Mohnike, K., et al. (1991) Parameters of mineral metabolism in children and adolescents in athletic training. *Kinderarztliche Praxis* 59, 153–156.

Barker, T., Martins, T.B., Hill, H.R., et al. (2011) Low vitamin D impairs strength recovery after anterior cruciate ligament surgery. *Journal of Evidence-Based Complementary & Alternative Medicine* 16, 201–209.

Basu, T.K. & Donaldson, D. (2003) Intestinal absorption in health and disease: micronutrients. Best Practice & Research: Clinical Gastroenterology 17, 957–979.

Batchelor, A.J. & Compston, J.E. (1983) Reduced plasma half-life of radiolabelled 25-hydroxyvitamin D3 in subjects receiving a high-fibre diet. *British Journal of Nutrition* 49, 213–216.

Bennell, K.L., Malcolm, S.A., Thomas, S.A., et al. (1996) Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *American Journal of Sports Medicine* 24, 810–818.

Bescos Garcia, R. & Rodriguez Guisado, F.A. (2011) Low levels of vitamin D in professional basketball players after wintertime: relationship with dietary intake of vitamin D and calcium. *Nutrición Hospitalaria: Organo Oficial de la Sociedad Española de Nutrición Parenteral y Enteral* 26, 945–951.

Beshgetoor, D. & Nichols, J.F. (2003) Dietary intake and supplement use in female master cyclists and runners. *International Journal of Sport Nutrition and Exercise Metabolism* 13, 166–172.

Bischoff-Ferrari, H.A., Borchers, M., Gudat, F., et al. (2004a) Vitamin D receptor expression in human muscle tissue decreases with age. *Journal of Bone and Mineral Research* 19, 265–269.

Bischoff-Ferrari, H.A., Dietrich, T., Orav, E.J., et al. (2004b) Positive association between 25-hydroxy vitamin D levels and bone mineral density: a population-based study of younger and older adults. *American Journal of Medicine* 116, 634–639.

Bischoff-Ferrari, H.A., Dietrich, T., Orav, E.J., et al. (2004c) Higher 25-hydroxyvitamin D concentrations are associated with better lower-extremity function in both active and inactive persons aged > or =60 y. *American Journal of Clinical Nutrition* 80, 752–758.

Bolland, M.J., Avenell, A., Baron, J.A., et al. (2010) Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *British Medical Journal* 341, c3691.

Cannell, J.J., Vieth, R., Umhau, J.C., et al. (2006) Epidemic influenza and vitamin D. *Epidemiology and Infection* 134, 1129–1140.

Cannell, J.J., Hollis, B.W., Zasloff, M., et al. (2008a) Diagnosis and treatment of vitamin D deficiency. *Expert Opinion on Pharmacotherapy* 9, 107–118.

Cannell, J.J., Vieth, R., Willett, W., et al. (2008b) Cod liver oil, vitamin A toxicity, frequent respiratory infections, and the vitamin D deficiency epidemic. *Annals of Otology, Rhinology and Laryngology* 117, 864–870.

Cannell, J.J., Hollis, B.W., Sorenson, M.B., et al. (2009) Athletic performance and vitamin D. *Medicine & Science in Sports & Exercise* 41, 1102–1110.

Cantorna, M.T., Woodward, W.D., Hayes, C.E., et al. (1998) 1,25-Dihydroxyvitamin D3 is a positive regulator for the two anti-encephalitogenic cytokines TGF-beta 1 and IL-4. *Journal of Immunology* 160, 5314–5319.

Cantorna, M.T., Munsick, C., Bemiss, C., et al. (2000) 1,25-Dihydroxycholecalciferol

prevents and ameliorates symptoms of experimental murine inflammatory bowel disease. *Journal of Nutrition* 130, 2648–2652.

Chen, T.C., Shao, A., Heath, H., et al. (1993) An update on the vitamin D content of fortified milk from the United States and Canada. *New England Journal of Medicine* 329, 1507.

Clark, M., Reed, D.B., Crouse, S.F., et al. (2003) Pre- and post-season dietary intake, body composition, and performance indices of NCAA Division I female soccer players. *International Journal of Sport Nutrition and Exercise Metabolism* 13, 303–319.

Constantini, N.W., Arieli, R., Chodick, G., et al. (2010) High prevalence of vitamin D insufficiency in athletes and dancers. *Clinical Journal of Sport Medicine* 20, 368–371.

Craig, W.J. & Mangels, A.R. (2009) Position of the American Dietetic Association: vegetarian diets. *Journal of the American Dietetic Association* 109, 1266–1282.

Cranney, A., Weiler, H.A., O'Donnell, S., et al. (2008) Summary of evidence-based review on vitamin D efficacy and safety in relation to bone health. *American Journal of Clinical Nutrition* 88, 513S–519S.

Davey, G.K., Spencer, E.A., Appleby, P.N., et al. (2003) EPIC-Oxford: lifestyle characteristics and nutrient intakes in a cohort of 33 883 meat-eaters and 31 546 non meat-eaters in the UK. *Public Health Nutrition* 6, 259–269.

de Torrente de la Jara, G., Pecoud, A., & Favrat, B. (2004) Musculoskeletal pain in female asylum seekers and hypovitaminosis D3. *British Medical Journal* 329, 156–157.

Edwards, K.M., Burns, V.E., Ring, C., et al. (2006) Individual differences in the interleukin-6 response to maximal and submaximal exercise tasks. *Journal of Sports Sciences* 24, 855–862.

Fischbach, F. & Dunning, M.B. (2009). *A Manual of Laboratory and Diagnostic Tests*. Wolters Kluwer/Lippincott Williams & Wilkins, Baltimore, MD.

Fu, L., Yun, F., Oczak, M., et al. (2009) Common genetic variants of the vitamin D binding protein (DBP) predict differences in response of serum 25-hydroxyvitamin D [25(OH)D] to vitamin D supplementation. *Clinical Biochemistry* 42, 1174–1177.

Gaitán, D., Flores, S., Saavedra, P., et al. (2011) Calcium does not inhibit the absorption of 5 milligrams of nonheme or heme iron at doses less than 800 milligrams in nonpregnant women. *Journal of Nutrition* 141, 1652–1656.

Gombart, A.F., Borregaard, N., & Koeffler, H.P. (2005) Human cathelicidin antimicrobial peptide (CAMP) gene is a direct target of the vitamin D receptor and is strongly up-regulated in myeloid cells by 1,25-dihydroxyvitamin D3. *FASEB Journal* 19, 1067–1077.

Halliday, T., Peterson, N., Thomas, J., et al. (2011) Vitamin D status relative to diet, lifestyle, injury and illness in college athletes. *Medicine & Science in Sports & Exercise* 42, 335–343.

Hamilton, B. (2010) Vitamin D and human skeletal muscle. *Scandinavian Journal of Medicine & Science in Sports* 20, 182–190.

Hamilton, B., Grantham, J., Racinais, S., et al. (2010) Vitamin D deficiency is endemic in Middle Eastern sportsmen. *Public Health Nutrition* 13, 1–7.

Heaney, R.P. (2011) Assessing vitamin D status. *Current Opinion in Clinical Nutrition and Metabolic Care* 14, 440–444.

Heaney, R.P. & Holick, M.F. (2011) Why the IOM recommendations for vitamin D are deficient. *Journal of Bone and Mineral Research* 26, 455–457.

Heaney, R., Recker, R., & Saville, P. (1978) Menopausal changes in calcium balance performance. *Journal of Laboratory and Clinical Medicine* 92, 953–962.

Heaney, R.P., Recker, R.R., Stegman, M.R., et al. (1989a) Calcium absorption in women: relationships to calcium intake, estrogen status, and age. *Journal of Bone and Mineral Research* 4, 469–475.

Heaney, R.P., Smith, K.T., Recker, R.R., et al. (1989b) Meal effects on calcium absorption. *American Journal of Clinical Nutrition* 49, 372–376.

Heaney, R.P., Dowell, M.S., Hale, C.A., et al. (2003) Calcium absorption varies within the reference range for serum 25-hydroxyvitamin D. *Journal of the American College of Nutrition* 22, 142–146.

Helle, C. & Bjerkan, K. (2011) *Vitamin D Status blant Norske Toppidrettsutøvere-og Faktorer av Betydning for Vitamin D-Status*. Idrettsmedisinsk Høstkongress, 38 p, November 3–6, 2011, Oslo, Norway.

Holick, M.F. (2007) Vitamin D deficiency. *New England Journal of Medicine* 357, 266–281.

Holick, M.F. (2009) Vitamin D status: measurement, interpretation, and clinical application. *Annals of Epidemiology* 19, 73–78.

Holick, M.F., Binkley, N.C., Bischoff-Ferrari, H.A., et al. (2011) Evaluation, treatment, and prevention of vitamin D deficiency: an Endocrine Society clinical practice guideline. *Journal of Clinical Endocrinology and Metabolism* 96, 1911–1930.

Hollis, B.W. (2005) Circulating 25-hydroxyvitamin D levels indicative of vitamin D sufficiency: implications for establishing a new effective dietary intake recommendations for vitamin D. *Journal of Nutrition* 135, 317–322.

Hypponen, E. & Power, C. (2007) Hypovitaminosis D in British adults at age 45 y: nationwide cohort study of dietary and lifestyle predictors. *American Journal of Clinical Nutrition* 85, 860–868.

Jackson, R.D., LaCroix, A.Z., Gass, M., et al. (2006) Calcium plus vitamin D supplementation and the risk of fractures. *New England Journal of Medicine* 354, 669–683.

Jacobsen, R.B., Hronek, B.W., Schmidt, G.A., et al. (2011) Hypervitaminosis D associated with a vitamin D dispensing error. *Annals of Pharmacotherapy* 45, e52.

Jones, G. (2008) Pharmacokinetics of vitamin D toxicity. *American Journal of Clinical Nutrition* 88, 582S–586S.

Jonvik, K.L., Anderson, L.F., & Helle, C. (2011) *Vitamin D-status hos Kvinnelige Toppidrettsutøvere i Handball og Football i Norge ved Breddegrad 60 degree North*. Idrettsmedisinsk Høstkongress, 42 p, November 3–6, 2011, Oslo, Norway.

Juzwiak, C.R., Amancio, O.M., Vitalle, M.S., et al. (2008) Effect of calcium intake, tennis playing, and body composition on bone-mineral density of Brazilian male adolescents. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 524–538.

Kiebzak, G.M., Moore, N.L., Margolis, S., et al. (2007) Vitamin D status of patients admitted to a hospital rehabilitation unit: relationship to function and progress. *American Journal of Physical Medicine and Rehabilitation* 86, 435–445.

Klontz, K.C. & Acheson, D.W. (2007) Dietary supplement-induced vitamin D intoxication. *New England Journal of Medicine* 357, 308–309.

Kohlenberg-Mueller, K., & Raschka, L. (2003) Calcium balance in young adults on a vegan and lactovegetarian diet. *Journal of Bone and Mineral Metabolism* 21, 28–33.

Koutkia, P., Chen, T.C., & Holick, M.F. (2001a) Vitamin D intoxication associated with an over-the-counter supplement. *New England Journal of Medicine* 345, 66–67.

Koutkia, P., Lu, Z., Chen, T.C., et al. (2001b) Treatment of vitamin D deficiency due to Crohn's disease with tanning bed ultraviolet B radiation. *Gastroenterology* 121, 1485–1488.

Laaksi, I., Ruohola, J.P., Tuohimaa, P., et al. (2007) An association of serum vitamin D concentrations <40 nmol/l with acute

respiratory tract infection in young Finnish men. *American Journal of Clinical Nutrition* 86, 714–717.

Lappe, J., Cullen, D., Haynatzki, G., et al. (2008) Calcium and vitamin D supplementation decreases incidence of stress fractures in female navy recruits. *Journal of Bone and Mineral Research* 23, 741–749.

Larson-Meyer, D.E. & Willis, K.S. (2010) Vitamin D and athletes. *Current Sports Medicine Reports* 9, 220–226.

Leachman Slawson, D., Mcclanahan, B.S., Clemens, L.H., et al. (2001) Food sources of calcium in a sample of African-American an Euro-American collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 11, 199–208.

Lehtonen-Veromaa, M., Mottonen, T., Irjala, K., et al. (1999) Vitamin D intake is low and hypovitaminosis D common in healthy 9- to 15-year-old Finnish girls. *European Journal of Clinical Nutrition* 53, 746–751.

Liel, Y., Ulmer, E., Shary, J., et al. (1988) Low circulating vitamin D in obesity. *Calcified Tissue International* 43, 199–201.

Liu, P.T., Stenger, S., Li, H., et al. (2006) Toll-like receptor triggering of a vitamin D-mediated human antimicrobial response. *Science* 311, 1770–1773.

Lovell, G. (2008) Vitamin D status of females in an elite gymnastics program. *Clinical Journal of Sport Medicine* 18, 159–161.

Lu, Z., Chen, T.C., Zhang, A., et al. (2007) An evaluation of the vitamin D3 content in fish: is the vitamin D content adequate to satisfy the dietary requirement for vitamin D? *Journal of Steroid Biochemistry and Molecular Biology* 103, 642–644.

Macfarlane, G.J., Palmer, B., Roy, D., et al. (2005) An excess of widespread pain among South Asians: are low levels of vitamin D implicated? *Annals of the Rheumatic Diseases* 64, 1217–1219.

Maimoun, L., Manetta, J., Couret, I., et al. (2006) The intensity level of physical exercise and the bone metabolism response. *International Journal of Sports Medicine* 27, 105–111.

Martineau, A.R., Wilkinson, R.J., Wilkinson, K.A., et al. (2007) A single dose of vitamin D enhances immunity to mycobacteria. *American Journal of Respiratory and Critical Care Medicine* 176, 208–213.

Minihane, A. & Fairweather-Tait, S. (1998) Effect of calcium supplementation on daily nonheme-iron absorption and long-term iron status. *American Journal of Clinical Nutrition* 68, 96–102.

Moe, S.M. (2008) Disorders involving calcium, phosphorus, and magnesium. *Primary Care: Clinics in Office Practice* 35, 215–237, v–vi.

Moshfegh, A., Goldman, J., Ahuja, J., et al. (2009) *What We Eat in America, NHANES 2005–2006: Usual Nutrient Intakes from Food and Water Compared to 1997 Dietary Reference Intakes for Vitamin D, Calcium, Phosphorus, and Magnesium.* Available at http://www.ars.usda.gov/SP2UserFiles/Place/12355000/pdf/0506/usual_nutrient_intake_vitD_ca_phos_mg_2005-06.pdf (accessed May 22, 2013).

Mowry, D.A., Costello, M.M., & Heelan, K.A. (2009) Association among cardiorespiratory fitness, body fat, and bone marker measurements in healthy young females. *Journal of the American Osteopathic Association* 109, 534–539.

Muller, K., Haahr, P.M., Diamant, M., et al. (1992) 1,25-Dihydroxyvitamin D3 inhibits cytokine production by human blood monocytes at the post-transcriptional level. *Cytokine* 4, 506–512.

Mulligan, G.B. & Licata, A. (2010) Taking vitamin D with the largest meal improves absorption and results in higher serum levels of 25-hydroxyvitamin D. *Journal of Bone and Mineral Research* 25, 928–930.

Nakamura, K., Nashimoto, M., Hori, Y., et al. (2000) Serum 25-hydroxyvitamin D concentrations and related dietary factors in peri- and postmenopausal Japanese women. *American Journal of Clinical Nutrition* 71, 1161–1165.

Nielsen, F.H. & Lukaski, H.C. (2006) Update on the relationship between magnesium and exercise. *Magnesium Research* 19, 180–189.

Nieman, D.C. (2000) Special feature for the Olympics: effects of exercise on the immune system: exercise effects on systemic immunity. *Immunology and Cell Biology* 78, 496–501.

Nowson, C.A., Worsley, A., Margerison, C., et al. (2004) Blood pressure response to dietary modifications in free-living individuals. *Journal of Nutrition* 134, 2322–2329.

Outila, T.A., Karkkainen, M.U., Seppanen, R.H., et al. (2000) Dietary intake of vitamin D in premenopausal, healthy vegans was insufficient to maintain concentrations of serum 25-hydroxyvitamin D and intact parathyroid hormone within normal ranges during the winter in Finland. *Journal of the American Dietetic Association* 100, 434–441.

Plotnikoff, G.A. & Quigley, J.M. (2003) Prevalence of severe hypovitaminosis D in patients with persistent, nonspecific musculoskeletal pain. *Mayo Clinic Proceedings* 78, 1463–1470.

Raimondi, S., Mabrouk, J.B., Shatenstein, B., et al. (2010) Diet and prostate cancer risk with specific focus on dairy products and dietary calcium: a case-control study. *Prostate* 70, 1054–1065.

Rankinen, T., Lyytikainen, S., Vanninen, E., et al. (1998) Nutritional status of the Finnish elite ski jumpers. *Medicine & Science in Sports & Exercise* 30, 1592–1597.

Rigby, W.F., Denome, S., & Fanger, M.W. (1987) Regulation of lymphokine production and human T lymphocyte activation by 1,25-dihydroxyvitamin D3. Specific inhibition at the level of messenger RNA. *Journal of Clinical Investigation* 79, 1659–1664.

Ross, A.C., Taylor, C.L., Yaktine, A.L., et al. (2010). *Dietary Reference Intakes for Calcium and Vitamin D*. National Academies Press, Washington, DC.

Ruohola, J.P., Laaksi, I., Ylikomi, T., et al. (2006) Association between serum 25(OH)D concentrations and bone stress fractures in Finnish young men. *Journal of Bone and Mineral Research* 21, 1483–1488.

Sabetta, J.R., Depetrillo, P., Cipriani, R.J., et al. (2010) Serum 25-hydroxyvitamin D and the incidence of acute viral respiratory tract infections in healthy adults. *PLoS One* 5, e11088.

Scientific Advisory Committee on Nutrition (SACN) (2007). Update on Vitamin D. Position Statement by the Scientific Advisory Committee on Nutrition www. TSHOSHOP.co.uk.

Shindle, M., Voos, J., Gulotta, L., et al. (2011) Vitamin D status in a professional American football team. *Medicine & Science in Sports & Exercise* 43, S340–S341.

Sly, L.M., Lopez, M., Nauseef, W.M., et al. (2001) 1alpha, 25-Dihydroxyvitamin D3-induced monocyte antimycobacterial activity is regulated by phosphatidylinositol 3-kinase and mediated by the NADPH-dependent phagocyte oxidase. *Journal of Biological Chemistry* 276, 35482–35493.

Smith, L.L. (2000) Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? *Medicine & Science in Sports & Exercise* 32, 317–331.

Snijder, M.B., Van Dam, R.M., Visser, M., et al. (2005) Adiposity in relation to vitamin D status and parathyroid hormone levels: a population-based study in older men and women. *Journal*

of Clinical Endocrinology and Metabolism 90, 4119–4123.

Storlie, D.M., Pritchett, K., Pritchett, R., et al. (2011) 12-Week vitamin D supplementation trial does not significantly influence seasonal 25(OH)D concentrations in male collegiate athletes. *International Journal of Health and Nutrition* 2(1), 28–33.

Tangpricha, V., Turner, A., Spina, C., et al. (2004) Tanning is associated with optimal vitamin D status (serum 25-hydroxyvitamin D concentration) and higher bone mineral density. *American Journal of Clinical Nutrition* 80, 1645–1649.

Tenforde, A.S., Sayres, L.C., Sainani, K.L., et al. (2010) Evaluating the relationship of calcium and vitamin D in the prevention of stress fracture injuries in the young athlete: a review of the literature. *PM & R: The Journal of Injury, Function, and Rehabilitation* 2, 945–949.

The Australian National Health and Medical Research Council (NHMRC) and the New Zealand Ministry of Health (2006) *Nutrient Reference Values for Australia and New Zealand*. Canberra, Commonwealth of Australia.

Valimaki, V.V., Alfthan, H., Lehmuskallio, E., et al. (2005) Risk factors for clinical stress fractures in male military recruits: a prospective cohort study. *Bone* 37, 267–273.

Vieth, R. (2004) Why the optimal requirement for vitamin D3 is probably much higher than what is officially recommended for adults. *Journal of Steroid Biochemistry and Molecular Biology* 89–90, 575–579.

Wang, T.T., Nestel, F.P., Bourdeau, V., et al. (2004) Cutting edge: 1,25-dihydroxyvitamin D3 is a direct inducer of antimicrobial peptide gene expression. *Journal of Immunology* 173, 2909–2912.

Ward, K.D., Hunt, K.M., Berg, M.B., et al. (2004) Reliability and validity of a brief questionnaire to assess calcium intake in female collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 14, 209–221.

Ward, K.A., Das, G., Berry, J.L., et al. (2009) Vitamin D status and muscle function in post-menarchal adolescent girls. *Journal of Clinical Endocrinology and Metabolism* 94, 559–563.

Ward, K.A., Das, G., Roberts, S.A., et al. (2010) A randomized, controlled trial of vitamin D supplementation upon musculoskeletal health in postmenarchal females. *Journal of Clinical Endocrinology and Metabolism* 95, 4643–4651.

Weaver, C.M., Proulx, W.R., & Heaney, R. (1999) Choices for achieving adequate dietary calcium with a vegetarian diet. *American Journal of Clinical Nutrition* 70, 543S–548S.

West, N.P., Pyne, D.B., Renshaw, G., et al. (2006) Antimicrobial peptides and proteins, exercise and innate mucosal immunity. *FEMS Immunology and Medical Microbiology* 48, 293–304.

Willis, K.S. (2008) *Vitamin D Status & Immune System Biomarkers in Athletes*. Masters of Science thesis. University of Wyoming.

World Health Organization & Food and Agricultural Organization of the United Nations (2004) *Vitamin and Mineral Requirements in Human Nutrition*, 2nd edn. WHO Publications, Geneva, Switzerland.

Wortsman, J., Matsuoka, L.Y., et al. (2000) Decreased bioavailability of vitamin D in obesity. *American Journal of Clinical Nutrition* 72, 690–693.

Zella, L.A., Shevde, N.K., Hollis, B.W., et al. (2008) Vitamin D-binding protein influences total circulating levels of 1,25-dihydroxyvitamin D3 but does not directly modulate the bioactive levels of the hormone in vivo. *Endocrinology* 149, 3656–3667.

Zhao, Y., Martin, B.R., & Weaver, C.M. (2005) Calcium bioavailability of calcium carbonate fortified soymilk is equivalent to cow's milk in young women. *Journal of Nutrition* 135, 2379–2382.

Zhu, Y., Mahon, B.D., Froicu, M., et al. (2005) Calcium and 1 alpha,25-dihydroxyvitamin D3 target the TNF-alpha pathway to suppress experimental inflammatory bowel disease. *European Journal of Immunology* 35, 217–224.

Ziegler, P.J., Nelson, J.A., & Jonnalagadda, S.S. (1999) Nutritional and physiological status of U.S. National figure skaters. *International Journal of Sport Nutrition* 9, 345–360.

Ziegler, P., Nelson, J.A., Barratt-Fornell, A., et al. (2001) Energy and macronutrient intakes of elite figure skaters. *Journal of the American Dietetic Association* 101, 319–325.

Zittermann, A. (2003) Vitamin D in preventive medicine: are we ignoring the evidence? *British Journal of Nutrition* 89, 552–572.

Chapter 21

Exercise-Induced Oxidative Stress: Are Supplemental Antioxidants Warranted?

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Introduction

Free radicals are highly reactive, unstable molecules with unpaired electrons. Because many of these molecules are oxygen-based, the term *reactive oxygen species* (ROS) is preferred when describing free radicals within physiologic contexts. ROS participate in spontaneous and enzymatically driven oxidation–reduction ("redox") reactions and are fundamental to every facet of biochemistry. Due to their unstable chemical nature, however, unabated ROS production may also promote damaging redox alterations to cellular proteins, lipids, and DNA. Damage occurs when ROS initiate a chemical reaction cascade, where unpaired electrons are exchanged between various molecules within a cell. A systematic imbalance in ROS production is called *oxidative stress*, where *oxidative damage* indicates redox modifications to cellular lipids, proteins, and DNA. The reader is directed to several authoritative text on the topic ROS, redox reactions, and oxidative stress as they pertain to the topic of exercise and work physiology (Halliwell & Gutteridge, 1999).

While exercise promotes myriad health benefits, acute participation in muscular exercise results in increased production of ROS (Davies et al., 1982; Reid et al., 1992). Indeed, abundant scientific evidence indicates that ROS are produced within contracting skeletal muscle and are responsible for disturbances in muscle performance including oxidative stress and muscle fatigue (Reid et al., 1992). Given that ROS production is inevitable during exercise, cellular *antioxidant* defenses exist as a means of quenching ROS overproduction. Antioxidants are molecules that detoxify oxidants. For example, within the lipid and aqueous phases of cellular and extracellular environments, enzymatic and nonenzymatic antioxidants work in concert to quench ROS. While the human genome codes for many endogenous antioxidants, numerous other antioxidants are derived from the diet. Dietary and endogenous antioxidants cooperate as a network to counteract ROS and oxidative damage. Some of the dietary antioxidants are held to be "expendable" in that they both quench ROS and reduce (in this context, "reduce" indicates chemical reduction) other components of the antioxidant network (Halliwell & Gutteridge, 1999). Collective understanding of ROS production in contracting skeletal muscle and dietary fortification of antioxidant defenses has led to decades of research into dietary countermeasures against exercise-induced oxidative stress. This research emphasis is predicated on the idea that supplementation of exogenous antioxidants will attenuate exercise-induced oxidative stress with the intent of improved exercise performance and recovery.

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The objective of this chapter is to highlight the current understanding of exercise-induced oxidative stress with the purpose of addressing a perceived need to consume dietary antioxidants by many athletes and regular exercisers. In order to understand whether dietary fortification of antioxidants is advantageous to exercise training and performance, several fundamental facets of ROS biology must be appreciated. The following sections will briefly discuss production of ROS during exercise, in addition to considerations of endogenous and exogenous antioxidant defenses. Additional discussion will detail current understanding regarding the short-term impact of redox balance on muscle contractions in addition to related exercise training adaptations. Finally, we will also discuss dietary antioxidant supplementation strategies to improve exercise performance and address the important question of whether there is an established need for athletes to consume antioxidant supplements to improve exercise performance.

Exercise-Induced ROS Production

Three decades have passed since Davies et al (1982) first demonstrated that ROS are produced by contracting skeletal muscles (Davies et al., 1982). Since this time, a wealth of research has uncovered which ROS are produced during exercise and where they are formed. While there are many potential intracellular sources of ROS, a few appear to be most responsible for the oxidative stress experienced during exercise. Within exercising skeletal muscle, ROS production is somewhat compartmentalized and occurs primarily within the mitochondria and cytosol (Halliwell & Gutteridge, 1999).

Mitochondria as a Primary Source of ROS in Contracting Muscle

Conventional thinking among exercise and oxidative stress experts holds that mitochondria are the most important source of ROS generation during exercise. This notion of mitochondrial ROS production during exercise is a logical extension of the fact that small amounts of the free radical superoxide are produced during the formation of ATP. A certain percentage of oxygen is incompletely reduced by the electron transport chain. Complex I (NADH dehydrogenase) and complex III (cytochrome bc1) have been verified as the sites of ROS formation within the mitochondria. Instead of being fully reduced to form water, oxygen is incompletely reduced at complexes I and III to produce the free radical superoxide. Independent of exercise, superoxide is produced as a stoichiometric function of total oxygen flux within the mitochondria. Early estimates gauged that 2–5% of mitochondrial respiration results in ROS formation (Boveris & Chance, 1973). Given the 15- to 20-fold rise in metabolic rate experienced in humans during moderate- to high-intensity exercise, it was assumed that ROS formation within respiring mitochondria was directly proportional (Halliwell & Gutteridge, 1999).

This classic understanding of ROS formation in proportion to mitochondrial metabolic rate, while not supported by empirical evidence, has been central to many widely held assumptions about exercise-induced oxidative stress. If correct, one would expect the magnitude of an exercise-induced oxidative stress response to be proportional to the metabolic costs of the exercise performed. This relationship was tested indirectly by Alessio et al. (2000) who found that despite large differences in oxygen costs, the rise in circulating oxidative damage markers was virtually identical following either maximal treadmill exercise or isometric grip strength exercise sessions. We furthered this understanding by demonstrating that a post-exercise rise in blood oxidative damage markers was related to exercise intensity but not to exercise energy expenditure. In this study, the oxidative damage markers were highest following maximal intensity treadmill exercise, while extended duration treadmill exercise at intensities above and below lactate threshold produced little change in circulating oxidative damage markers (Quindry et al., 2003). Evidence from these studies provides a compelling, albeit indirect, challenge to the belief that ROS are derived largely from the mitochondria.

More recent findings from basic science investigations reveal several important lines of direct evidence which conclusively debunk the notion that mitochondria are the major source of ROS during

exercise. First, the rate of mitochondrial superoxide production is much lower than first believed, being closer to 0.15% of the total oxygen consumed by respiring mitochondria. Second, exercise appears to "tighten" electron transport, meaning that the rate of ROS production during exercise does not rise in proportion to the increase in metabolic rate (Anderson et al., 2007). In aggregate, the wealth of modern studies no longer supports the long-held notion that exercise-induced oxidative stress is largely dependent on mitochondrial-derived ROS.

Acute exercise also induces ROS from cytosolic sources. Cytosolic ROS production is largely catalyzed by enzymes, including xanthine oxidase (XO), NAD(P)H oxidase, and nitric oxide synthase (NOS) (Davies et al., 1982; Jackson, 1998; Reid et al., 1992). NAD(P)H oxidase and other membrane-bound/membrane-associated enzymes also implicate extracellular spaces as targets of ROS-induced damage. Other extracellular sources of ROS include autoxidation of catecholamines and free radical burst from phagocytes recruited to sites of inflammation during and following exercise (Halliwell & Gutteridge, 1999). This information summarizes the major forms of ROS likely produced during exercise.

Within the context of exercise, several additional facets of ROS and redox biology should be considered. First, the reactive nature of ROS means they are short-lived; possessing an average half-life on the order of milliseconds. Superoxide, in addition to the slightly longer-lived nitric oxide, hydrogen peroxide, hydroxyl radical, and other biologically important ROS, reacts rapidly to extinction with antioxidants or other cellular constituents. The speed with which ROS react and disappear presents a difficult problem for scientists attempting to measure the real-time rate of ROS production in living cells. While there are some analytic means to identify ROS directly, modern science often relies on the "chemical finger prints" and physiologic outcomes of ROS to assess their impact during exercise. The short-lived nature of ROS also means that they typically do not travel far from their site of production. This rapid disappearance of ROS underpins the compartmentalized nature of oxidative stress described earlier (Halliwell & Gutteridge, 1999). Hydrogen peroxide and NO are notable exceptions to ROS compartmentalization in that they can readily diffuse through membrane phospholipid bilayers. Collectively, this basic understanding of ROS production is important because countermeasures against unwanted oxidative damage may require strategic targeting to the local environment where ROS are produced.

Exercise and Antioxidants

Oxidative stress results from an imbalance between ROS production and ROS removal. Redox balance between oxidants and antioxidants is illustrated conceptually in Figure 21.1. Overproduction of ROS in addition to the loss of, or deficiency in, antioxidants can yield an imbalance that favors oxidative stress. Similar to oxidant production, antioxidant defenses are also compartmentalized within cells. Compartmentalization of antioxidants includes both enzymatic and nonenzymatic components, in addition to aqueous and lipid-phase antioxidants. The cellular distribution of the antioxidant defense network corresponds to all recognized ROS sources. Biologically relevant antioxidant defenses are summarized in Table 21.1. Categorization is based on enzymatic/nonenzymatic function, location, and the potential for dietary contribution. A brief discussion of enzymatic, nonenzymatic, and dietary antioxidants follows with emphasis to exercise and sport performance. In preparation for an integrated discussion of antioxidants and exercise performance, a preliminary overview of antioxidant supplementation research is also provided.

Enzymatic Antioxidants

The key enzymatic antioxidants within skeletal muscle include superoxide dismutase (SOD), catalase (CAT), and glutathione peroxidase (GPx). The SOD enzyme converts superoxide to the less reactive ROS, hydrogen peroxide. Cellular variations of SOD exist based upon cellular location and associated mineral cofactors. Mitochondria contain a manganese-containing isoform of SOD (MnSOD), while copper–zinc SOD (CuZnSOD) is located in the cytosol. The enzymes CAT and GPx are responsible for converting hydrogen peroxide to water.

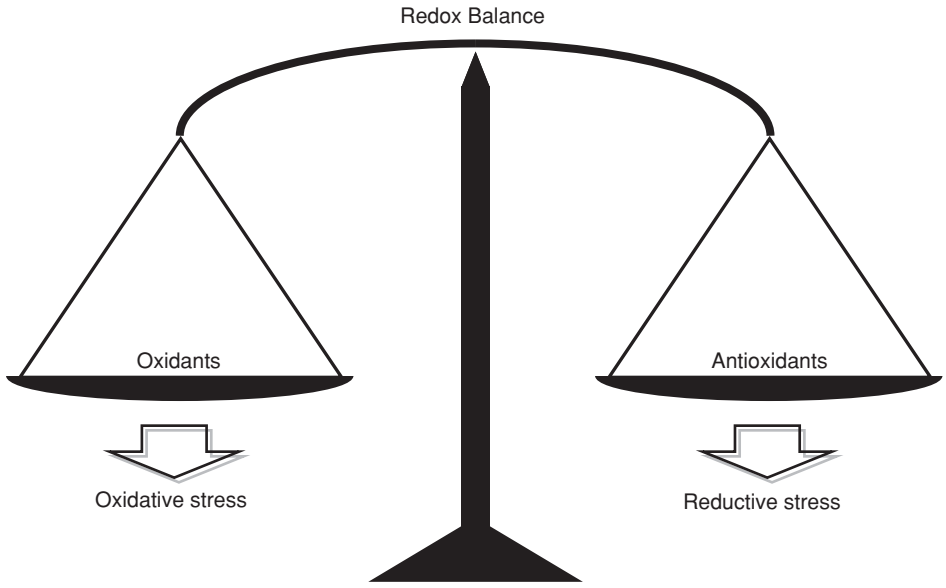

Figure 21.1 The redox balance between oxidative stress and reductive stress. The relationship between oxidant and antioxidants determine redox balance. An overabundance of oxidants and/or deficiency in antioxidants results in oxidative stress. Likewise, the often overlooked reductive stress will occur when antioxidant levels overwhelm oxidant production.

Table 21.1 Cellular and extracellular antioxidant defenses

Antioxidant	Form: E, endogenous; D, dietary	Type: A, aqueous; L, lipid phase	Location: C, cellular; E, extracellular; M, mitochondrial
Enzymatic antioxidants			
Superoxide dismutase (SOD)	E	A	C, E, M
Glutathione peroxidase (GPx)	E	A	C, M
Catalase (CAT)	E	A	C, M
Nonenzymatic antioxidants			
Glutathione (GSH)	E	A	C
Vitamin E	D	L	C, E
Vitamin C	D	A	C, E
Carotenoids	D	L	C, E
Uric acid	E	A	C, E
Flavonoids	D	A, L	C, E, M
Ubiquinones	D, E	L	C, M
α-Lipoic acid	E	A	C, E

The reader may have already recognized evidence of the antioxidant network in the current discussion in that hydrogen peroxide produced by SOD is further converted to redox stable products by CAT and GPx. Thus, antioxidant enzymes work in series to fully detoxify ROS. This network is further emphasized by the fact that the nonenzymatic antioxidant glutathione is a substrate for the enzymatic antioxidant GPx (Ji, 1995). The antioxidant network, therefore, includes an interface between enzymatic and nonenzymatic antioxidant defenses.

Nonenzymatic Antioxidants

Glutathione (GSH) is an important aqueous-phase cellular antioxidant. In chemical terms, GSH is the most abundant nonprotein antioxidant thiol found in cells and can scavenge several physiologically important ROS. In redox reactions, two GSH molecules donate their hydrogen molecules, forming the oxidized disulphide glutathione (GSSG). Oxidized glutathione is "recycled" by the enzyme glutathione reductase. Further recycling of GSSG occurs through interactions with water-soluble

antioxidants, such as vitamin C in addition to lipid-soluble antioxidants including vitamin E. The collective utility of GSH as a cellular reducing agent resides in the broad interactions within the antioxidant network, including enzymatic, lipid, and water-soluble antioxidants. GSH is found in high concentrations in skeletal muscles, with preferential distribution in the more oxidative slow twitch muscle fibers (Ji, 1995). GSH, and in particular the GSH/GSSG, is an important factor in mediating the adaptive response to metabolic challenges like exercise (Jones, 2006). Because GSH is interconnected with the antioxidant network and bioenergetic control, dramatic and sustained fluctuations in GSH/GSSG are thought to be detrimental to cellular health. This point of caution likely extends to supplemental GSH as an intended countermeasure to exercise-induced oxidative stress. In this regard, supplemental delivery of GSH is complicated because it is broken down in the small intestine. In response to this fact, however, alternative approaches to support cellular GSH have been investigated. Research over the last two decades reveals *N*-acetylcysteine (NAC), a nonspecific antioxidant, actively supports GSH resynthesis within the antioxidant network (Ruffmann & Wendel, 1991). Early nonhuman studies demonstrated that moderate dosages of NAC administration are effective in improving muscular contractions, an outcome that was supported in human exercise studies. Early optimism that NAC could be an effective ergogenic agent has been tempered by the fact that NAC use in humans is also marked by severe nausea and gastrointestinal side effects (Reid, 2008).

Uric acid is an important aqueous-phase antioxidant in cells and extracellular environments including blood plasma. Uric acid, which is present in micromolar concentrations, is the major water-soluble antioxidant in blood plasma (Cao & Prior, 1998). While the role of uric acid is well established, compartmental shifts of this antioxidant occur during exercise, such that high-intensity exercise can produce acute elevations in circulating uric acid (Quindry et al., 2003). While some authors have described a post-exercise elevation in uric acid as "a compensatory protective response" designed to fortify plasma antioxidant levels, the response is recognized to simply be the biochemical outcome of accelerated purine metabolism during exercise. Ironically, this metabolically driven production of uric acid in exercising skeletal muscle is also marked by enzymatic production of ROS. To determine if circulating uric acid plays an important antioxidant role during exercise, we recently tested the postulate that diminished uric acid levels will increase exercise-induced oxidative stress in the blood. Using a pharmacological approach to reduce circulating uric acid, our results indicate that a decline in circulating uric acid does not exacerbate exercise-induced oxidative markers of damage in the blood (McAnulty et al., 2007).

Ascorbic acid, or vitamin C, is a potent aqueous phase antioxidant. In addition to direct ROS scavenging, vitamin C fortifies the antioxidant network by "recycling" aqueous phase antioxidants and lipid phase antioxidants alike (Packer et al., 1986). During redox reactions, vitamin C becomes oxidized to the ascorbate radical. In the presence of transition metals, including the physiologically plentiful iron and copper, high doses of vitamin C can actually exert pro-oxidant effects. Understanding the pro-oxidant potential of vitamin C has tempered historic enthusiasm for vitamin C megadoses. More recent evidence in blood plasma, however, reveals that while the rate of lipid oxidative damage is promoted by high vitamin C concentrations, it is also dependent upon the physiologic concentration of iron and copper. This latter point about vitamin C further reinforces the fact that redox biology is complex. Because redox biology outcomes can be situation specific, it is important to note that blanket applications of exogenous antioxidant supplementation are generally unwarranted.

Vitamin E is an encompassing term for the lipid phase antioxidant system constituting eight chemical variations of tocopherols and tocotrienols. Found within lipid membranes, vitamin E, including the well-studied isomer a-tocopherol, is a chain-breaking antioxidant capable of converting several ROS to less reactive forms (Traber, 1994). While vitamin E–ROS reactions produce the vitamin E radical, vitamin E is recycled by interactions with the antioxidant network at the lipid–water interface (Traber, 1994). Scientific interest in vitamin E as a dietary supplement has

waned over the last decade due to reports of elevated cancer risk in some individuals. While in nonathletes, vitamin E supplementation is most prevalent in those above the age of 50 (Yang et al., 2011), recent survey evidence suggests that a majority of competitive athletes continue to ingest supplemental vitamin E as part of their training regimen. Subsequent discussion will address the potential consequences of vitamin E supplementation, beyond recommended dietary allowances, in those who exercise (Garelnabi et al., 2011; Strobel et al., 2011).

α-Lipoic acid is present in low quantities within biological systems, but remains an important cofactor for a-dehydrogenase complexes and performs antioxidant functions through thiol donor reactions. Because it is often complexed with enzymes, antioxidant functions of α-lipoic acid are somewhat limited by comparison to many compounds described previously. Free α-lipoic acid, however, does exhibit potent antioxidant properties against all major ROS. α-Lipoic acid is also an important "cog" in the antioxidant network, serving as a reducing agent for the vitamin C radical. Dietary consumption of α-lipoic acid is plausible in that it does not accumulate to toxic levels. While literature exists to support α-lipoic acid supplementation in the context of exercise and metabolic or vascular diseases (McNeilly et al., 2011), recent studies indicate that exercise adaptations are blunted by exogenous α-lipoic acid (Strobel et al., 2011). Additional details will be provided in a subsequent section.

Coenzyme Q (ubiquinone) is another lipid soluble compound with antioxidant properties. The antioxidant capacity of coenzyme Q is due to the reducing capacity of the phenol ring structure. In humans, coenzyme Q-10 is the predominant form derived from dietary sources including soybean oil, meats, fish, nuts, wheat germ, and some vegetables. The majority (40–50%) of tissue coenzyme Q is found in mitochondria. Plasma coenzyme Q levels circulate in the micromolar range, with roughly 80% being in the reduced rather than the oxidized form. Given the location and concentration of coenzyme Q, speculation persists that exogenous supplementation may attenuate oxidative stress and muscle damage responses to extreme exercise. Examination of an antioxidant supplement cocktail which contained coenzyme Q, however, failed to reveal fortification of antioxidant capacity in the blood plasma of rested individuals. The antioxidant supplement did not influence exercise performance, attenuate oxidative stress, or affect indices of muscle damage following a marathon run (Kaikkonen et al., 1998).

Carotenoids contribute to the coloration of many orange and yellow foods and are lipid soluble antioxidants capable of direct ROS scavenging. Due to their hydrophobicity, carotenoids affiliate with cellular and organelle membranes, thereby limiting lipid peroxidation during periods of ROS production. As with several antioxidants described previously, carotenoids, including β-carotene, exhibit pro- and antioxidant properties. However, during exercise, β-carotene most likely serves as a reducing agent to remove ROS. In most people, carotene intake is almost exclusively dietary in nature rather than from supplements. This observation is mostly true in 19- to 30-year-olds who are most likely to be in a competitive stage of athletic competition (Yang et al., 2011). At present, the role that dietary carotenoids play as an antioxidant in contracting skeletal muscle remains unclear. Flavonoid is a generic term for over 4000 plant-based compounds with antioxidant properties. The flavonoid family can be broadly subdivided into flavones, isoflavones, flavonones, anthocyanins, and catechins. Subdivisions of the flavonoid family represent subtle chemical variations on the common flavonoid structure, which are capable of producing vastly different physiologic responses including anti-inflammatory and cytoprotective responses in addition to antioxidant functions. Antioxidant properties are attributed to the polyphenolic molecular arrangement of flavonoids. Catechins, in particular, are potent polyphenol compounds found in green and black tea, red wine, and extracts from a variety of leafy plants. These compounds act as direct scavengers of several ROS and are also capable of supporting the antioxidant network. Within the realm of exercise and antioxidant research, recent interest has been directed toward a biologically potent flavonoid, quercetin. While an early study suggested that quercetin supplementation improved exercise performance, presumably through alterations in antioxidant capacity, we were unable to demonstrate any alterations in plasma

antioxidant capacity, oxidative stress, or exercise performance in ultramarathon athletes consuming quercetin (Quindry et al., 2008).

Identification of ROS and Biomarkers of Oxidative Damage

The topic of exercise and antioxidants is incomplete without understanding the analytical means of quantifying the radical-quenching properties of antioxidants and the identification of free radicals and oxidative damage, in addition to methods for assessing the influence of redox reactions on muscular performance. Parent ROS are very labile and initiate rapid redox chain reactions which make direct identification of ROS in tissues very difficult with current technology. This fact is further complicated by the whole animal nature of exercise-based research. Because of the compartmentalized nature of oxidative stress, different analytical techniques predominate to understand redox perturbations in blood plasma, skeletal muscle, and other tissues. Moreover, compartmentalization adheres to the free radical pecking order in that local antioxidants must be depleted prior to the appearance of oxidative damage markers (Buettner, 1993; Frei et al., 1988). This section summarizes current methodological approaches to ROS identification and biomarkers for oxidative stress and oxidative damage, in addition to strengths and weaknesses inherent to common laboratory approaches to investigate redox alterations on muscular contractions. Table 21.2

Table 21.2 Experimental techniques to identify oxidative stress

Biomeasures	Compartment: B, blood; O, organelle; T, tissue	Sensitivity: H, high; M, moderate; L, low	Comments
Direct measures			
Electron spin resonance (ESR)	B, T	H	Technically difficult, fresh samples preferred
Chemiluminescence detection	B, O	H	Fresh samples required
Fluorescence detection	B, O, T	H	Fresh samples required
Indirect measures—oxidative damage			
Lipid			
F_2-isoprostanes	B, T	H	Technically difficult, arachidonic acid metabolites
Lipid hydroperoxides	B, O, T	M	Limited storage sample time, membrane lipids
Malondialdehyde/TBARS	B, T	L	Most widely used, nonspecific
Protein			
Carbonyls – spectrophotometric	B, O, T	L	Simple and cost effective, but lacking in sensitivity
Carbonyls – antibody	B, O, T	H–M	Western blot, ELISA, histological
DNA			
8-hydroxydeoxyguanosine (8-OHdG)	B, O, T	M–L	Blood measures
Antioxidant capacity measures			
Trolox equivalent antioxidant capacity (TEAC)	B, O, T	M–L	Non-protein aqueous and lipid phase antioxidants, 60% UA
Ferric reducing antioxidant potential (FRAP)	B, O, T	M–L	Inhibitory method, protein and sulfhydryls, 30% albumin, 20% UA, 10% vitamin C
Oxidant reducing antioxidant capacity (ORAC)	B, O, T	M–L	Inhibitory method, 30% albumin, 10% UA, 60% nonspecific activity
Oxidized/reduced glutathione (GSH/GSSG)	B, O, T	H	Sample handling is critical for accuracy, fresh samples preferred

ELISA, enzyme-linked immunosorbent assay; UA, uric acid.

summarizes the key points regarding biomarkers and indices most common to oxidative stress research.

ROS Identification

Given current technology, the most direct means for ROS assessment within biological samples such as blood plasma and tissue homogenates, is the electron spin resonance spectroscopy (ESR) technique. Molecules are identified by the electron paramagnetic resonance signal which produces a chemical "finger print" based on the unpaired electrons for the specific ROS. Several studies have successfully applied the ESR technique in blood plasma samples taken from humans before and after exercise. ESR is sensitive enough to identify exercise-induced ROS production. Moreover, ESR exhibits similar sensitivity to established lipid biomarkers of oxidative damage (Ashton et al., 1999). Application of ESR to exercise-based research, however, has been limited, as fresh samples are preferred and the technique is difficult by comparison with other oxidative stress biomarkers.

Other techniques for direct ROS measurement involve the application of fluorescent and chemiluminescent probes which illuminate on reaction with ROS. The chemiluminescent assays rely on detection of a light producing reaction when lumigenic compounds such as lucigenin react directly with superoxide anions. Exercise applications of chemiluminescent assays often involve isolation of circulating leukocytes, though more physiologic whole blood variations of the assay are possible (Quindry et al., 2003). While these assays are generally quite sensitive, they require artificial stimulation with phorbol 12-myristate 13-acetate or related compounds. Because these experiments require artificial stimulation to elicit ROS production, the assay ultimately represents the potential for free radical production as opposed to the actual ROS load experienced. Alternate experimental approaches include the application of probes which produce a fluorescent signal in reaction with superoxide and hydrogen peroxide and can be detected with a high degree of sensitivity. Fluorescent approaches to ROS detection have advanced applied exercise science research applications. While uses of these fluorescent probes are generally limited to cell culture and isolated mitochondrial preparations, molecular biology is becoming routine in exercise physiology research (Kavazis et al., 2008, 2010). Nonetheless, these applications in whole human or animal studies are surgically invasive, time, and equipment intensive.

Biomarkers of Oxidative Damage Exercise and oxidative stress research is invariably marked by use of oxidative damage biomarkers. Use of these variables for oxidatively modified lipid, protein, and DNA molecules is necessary due to the rapidity of redox chain reactions. The popular biomarkers in use today are pragmatic in that they identify oxidized molecules which are stable relative to their ROS precursors. DNA oxidation products are commonly examined by variations on a common assay of 8-hydroxy-deoxyguanosine (8-OHdG). These sensitive biochemical techniques for DNA oxidative damage examine free radical modifications to guanine residues (Halliwell & Gutteridge, 1999). 8-OHdG can be measured in tissues as well as blood plasma and urine, but these latter applications are merely signal responses, making specific interpretation of findings difficult.

Protein oxidation assays are dominated by variations on the protein carbonyl assay, which identifies the presence of carbonyl group formation on specific amino acids predisposed to oxidation. Carbonyl groups are identified in assay by reaction with dinitrophenylhydrazine (DNPH), which is readily measured by spectrophotometric or antibody-directed assays. Advancements in assay kit technology, including enzyme-linked immunosorbent assays (ELISA), have furthered the use of the now preferred antibody-driven examination of protein carbonyls in the exercise sciences. When examined via western blot, additional information can be gained in regard to the molecular weights of proteins that are preferentially modified. Experiments in skeletal muscle exposed to disuse atrophy revealed protein oxidation at molecular weights common to actin and myosin (Zergeroglu et al., 2003). If combined with immunoprecipitation techniques, future work may better quantify the proteins

that are oxidatively modified following exercise. This point is relevant to tissue as well as plasma, where nonspecific elevations in protein carbonylation have been observed for more than a decade. In regard to examination of blood oxidative stress, antibody-directed protein carbonyls have proved to be an important and sensitive variable in recent years due to the fact that the postexercise circulating half-life of protein carbonyls lasts for several hours (Hudson et al., 2008; Quindry et al., 2011).

As compared to DNA and protein, lipid oxidative stress biomarkers have been examined with the most varied and most controversial experimental approaches. Polyunsaturated lipids interact with ROS to form malondialdehydes (MDA). The thiobarbituric acid reactive substances (TBARS) assay is the most common method for determining MDA in blood plasma and tissue samples. While TBARS remain among the most popular lipid peroxidation assay applications in exercise and oxidative stress research, the assay lacks specificity and is not recommended (Alessio et al., 2000; Fisher et al., 2011). Lipid hydroperoxides (LOOH) and F_2-isoprostanes are preferred lipid peroxidation biomarkers. LOOH represent lipid membrane oxidative products and can be measured by high-performance liquid chromatography and spectrophotometry. F_2-isoprostanes are relatively stable products of arachidonic acid metabolism, and include oxidatively modified signaling molecules. When examined by the highly sensitive gas chromatography–mass spectroscopy (GC–MS) technique, F_2-isoprostanes are considered a "gold standard" of lipid peroxidation. However, the gold standard title is somewhat misleading in that F_2-isoprostanes and LOOH are different compounds. Both F_2-isoprostanes and LOOH are responsive biomarkers to various exercise modalities, though observations suggest their half-life is shorter and sensitivity to exercise stimuli less potent than that observed for protein carbonyls. The reader is directed to a complete review of biomarkers for oxidative stress that are useful in exercising humans (Powers et al., 2010).

Antioxidant Capacity Experimental approaches to estimate antioxidant capacity in tissues have increased over the last decade. Two popular techniques, ferric reducing antioxidant potential (FRAP) and oxidant reducing antioxidant capacity (ORAC), examine the reducing potential of biological antioxidants exposed to an artificial ROS challenge. Both ORAC and FRAP are sensitive to protein and aqueous phase antioxidants, while the latter also captures sulfhydryl content. Nonprotein aqueous and lipid phase antioxidants can be measured with the trolox equivalent antioxidant capacity (TEAC) assay (Cao & Prior, 1998). When examined in the blood plasma following exercise, interpretations of these assays can be difficult in that high-intensity physical activity can stimulate renal release of ascorbic acid resulting in elevated values for these antioxidant metrics. Further increases in blood plasma FRAP and TEAC can be observed due to compartmental shifts in uric acid mentioned previously (Quindry et al., 2003, 2008). Within tissues, examination of GSH/GSSG has proved to be a sensitive metric for redox perturbations (Anderson et al., 2007; Jones, 2006). Widespread use of GSH/GSSG is limited by the fact that, in contrast to the previously described assays, sample-handling concerns are more pressing and fresh sample assay is preferred.

Redox Changes and Contractile Performance Physiologic responses to ROS and antioxidant countermeasures are the ultimate means for understanding exercise and oxidative stress. Broad arrays of experimental approaches have been employed for the study of redox changes on contractile performance. The spectrum of approaches includes subphysiologic examination of isolated mitochondria and isolated skeletal muscle fibers to more physiologic investigations of contractile force in muscle strips, isolated organs, and organism-based experiments. Early experiments demonstrated unequivocally that ROS and antioxidants influence skeletal muscle contractile strength. These "proof of principle" experiments were later verified in whole animal and human exercise studies by using antioxidant compounds such as the aforementioned NAC (Reid, 2008). Sophisticated experimental designs now include molecular biology techniques which demonstrate direct associations between work physiology, ROS, and gene-level adaptations to exercise. More so than experimental designs which rely only on

biomarkers of oxidative stress, these recent investigations demonstrate that many fundamental adaptations to exercise are dependent upon an oxidative stress response stimulus (Garelnabi et al., 2011; Matsumoto et al., 2011; Strobel et al., 2011).

Balancing Muscular Performance, Redox Regulation, and Antioxidants

Antioxidant Deficiencies and Exercise Performance

Antioxidant nutrient deficiency is the most obvious case for antioxidant supplementation against exercise-induced oxidative stress. Animal research demonstrates that severe vitamin E and vitamin C deficiencies predispose exercised muscle to pathologic oxidative damage (Davies et al., 1982). Caution is warranted, however, when extrapolating animal studies with lifelong dietary control to humans. While it is plausible that nutritional deficiencies would create a need for antioxidant supplementation in athletes undertaking heavy exercise loads, such instances are rare. Marginal deficiencies of vitamin C in humans do not appear to impair exercise tolerance. Similarly, exercise performance decrements and muscle weakness was not observed in conditions of chronic vitamin E deficiency.

ROS, Redox Balance, and Muscular Contractions

In contrast to conditions of antioxidant deficiency, many researchers have questioned whether ergogenic benefits are gained by antioxidant supplementation in well-fed subjects consuming a varied diet. To understand what is known to date about this topic, one must first consider fundamental facts about ROS as contributors to muscle fatigue and force production.

Oxidative Stress and Muscular Fatigue As stated earlier, repetitive muscular contractions result in an obligatory burst of ROS. Understanding the impact of these ROS on muscular contractile strength has been advanced significantly by basic science research in highly controlled laboratory settings. It is now well-understood that when contractile intensity and duration are of sufficient magnitude, muscular ROS production directly contributes to muscular fatigue (Reid et al., 1992) and that the cellular mechanisms responsible for muscular fatigue are both directly and indirectly linked to oxidative stress. Paradoxically, a modest increase in skeletal muscle ROS production also elicits a significant improvement in muscular contractions. Several years back, Reid proposed an eloquent working model to describe the optimal redox state for muscular performance on a continuum of reduced to oxidized conditions (Reid, 2001). In his conceptual model, ROS are a "double edged sword" in that an optimum redox state is required to maximize muscular performance. Figure 21.2 illustrates this understanding that ROS can both improve and diminish contractile strength. Within this conceptual model, activation of rested skeletal muscle initiates moderate ROS generation which improves force generation. The addition of supplemental antioxidants

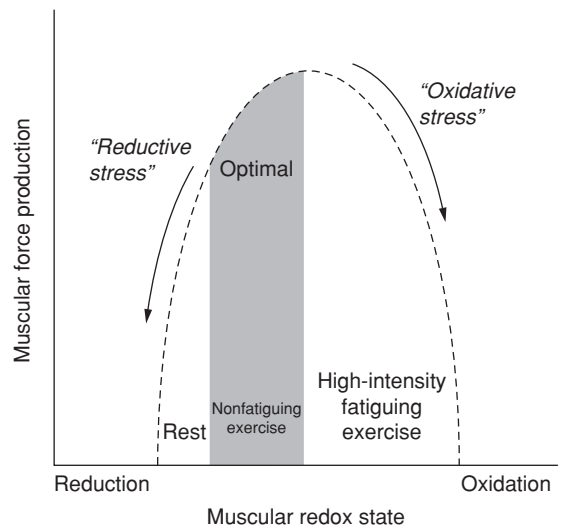

Figure 21.2 Conceptual model of redox balance and skeletal muscle force production. Studies performed in isolated muscle experiments designed to mimic repetitive exercise demonstrate that moderate levels of ROS production improve force production. Excessive ROS production, similar to what would be experienced during high intensity or prolonged duration exercise, results in oxidative stress and decrements in contractile strength. Reductive stress due to antioxidant supplementation also diminishes muscle force production. Modified from Reid (2001).

reduces the muscle system, blunting ROS-driven improvements in contractile force at the initiation of exercise. Larger magnitude ROS exposure during high-intensity or prolonged muscular contractions produces a fatigue response which can be countered with exogenous antioxidants (Reid, 2001).

Antioxidant Countermeasures Against ROS-Induced Muscular Fatigue Similar to ROS, exogenous antioxidant use has a variable impact on muscular strength and fatigue. The aforementioned increase in contractile strength in fresh muscle exposed to moderate ROS is blunted by administration of antioxidant compounds (Reid, 2001). Antioxidant administration during conditions of higher ROS, in contrast, directly counters the fatigue produced by oxidative stress experienced in isolated muscle experiments. Human-based research further demonstrated that antioxidant infusions prior to and during exercise effectively blunted ROS-induced muscular fatigue in controlled laboratory settings (Reid, 2001).

Longstanding excitement that antioxidant supplementation may prove to be ergogenic in athletic scenarios has been tempered by inconsistent findings. In fact, exercise performance during marathon competition was not improved even when indices of oxidative damage were blunted by dietary antioxidant use. Use of the antioxidant NAC is a notable exception to these previous studies in that well controlled laboratory investigations of NAC demonstrate consistent improvements in muscular contractility. To this end, NAC has been demonstrated as an effective antioxidant countermeasure in both cardiovascular and muscular strength exercise challenges (Reid, 2001; Ruffmann & Wendel, 1991). Nonetheless, important questions have been raised about whether findings which are largely limited to untrained subjects, clinical scenarios, and scientific paradigms will translate to athletic performance improvements (Reid, 2008). Further concerns regarding NAC supplementation as an ergogenic aid for training and competition remain due to a host of adverse NAC-induced side effects including nausea and gastrointestinal distress (Reid, 2008). In contrast, other antioxidant compounds have yielded equivocal results across exercise studies with varied scientific approaches. Future investigations must employ cutting-edge biochemical and molecular biology methodologies in translational exercise studies to better understand the role of other antioxidant compounds with potential ergogenic effects. One such compound which shows early promise in muscle applications is the mitochondrially-directed antioxidant termed SS31. This compound is able to diffuse into the mitochondria of skeletal muscle and quench locally produced ROS (Gilliam et al., 2011). While exercise-based investigations in humans have not been undertaken, novel pharmacologically derived antioxidants of this type may represent a promising horizon in exercise and oxidative stress research.

Antioxidants and Muscle as a Redox Sensing Organ Perturbations in redox balance within skeletal muscle indicate oxidative stress is an undeniable result of muscular contractions in addition to being a necessary stimulus for exercise adaptations. Inevitable as oxidative stress is during high intensity exercise, the response is transient and non-pathologic. The importance of brief oxidative stress during exercise is now recognized to be as fundamental to beneficial adaptations as the stimuli derived from moderate hyperthermia, calcium regulation, and bioenergetic control.

Evidence supporting the importance of exercise-induced ROS as a beneficial adaptive stimulus includes the fact that upregulation of endogenous antioxidant enzymes within heart and skeletal muscle is dependent upon ROS (Hamilton et al., 2003). Use of supplemental antioxidants to attenuate exercise-induced ROS effectively prevents oxidative stress, but also blunts upregulation of cellular antioxidant defenses (Gomez-Cabrera et al., 2008; Hamilton et al., 2003; Ristow et al., 2009). In addition to protective antioxidant enzymes, supplemental antioxidant use against exercise-induced oxidative stress also attenuates a host of important protective adaptations including heat shock proteins (Hamilton et al., 2003) and mitochondrial biogenesis (Strobel et al., 2011).

Because of these attributes, skeletal muscle has been described as a redox sensing organ (Reid, 2008). ROS generated during exercise

communicate cellular need, provoking a specific adaptive response by the genome. From an evolutionary perspective of "thrifty genetics," fortification of endogenous antioxidant enzymes and other cellular defenses is bioenergetically costly, and warranted only on an as-needed basis. The scientific rationale presented in this review supports the growing realization that ROS production during exercise is not detrimental to long-term health and underpins the adaptations to exercise training. For this review, we have developed a working model to summarize the understanding that exercise-induced oxidative stress is a necessary stimulus to beneficial adaptations (Figure 21.3). As illustrated, exercise elicits a transient oxidative stress which briefly leaves the zone of redox balance resulting in oxidative stress. This transient oxidative stress initiates an adaptive response that includes endogenous antioxidant enzymes, heat shock proteins, and other gene products described previously (Asha Devi et al., 2003; Coombes & Fassett, 2011; Gomez-Cabrera et al., 2008; Matsumoto et al., 2011; Ristow et al., 2009). Supplemental antioxidant use creates a subtle but significant reductive stress (represented by dashed lines) in that much of the adaptive stimulus provided by ROS is attenuated.

Concluding Statements

Muscular exercise elicits a transient oxidative stress that is also a cornerstone of the adaptive stimulus to exercise. Use of supplemental dietary antioxidants, though still a common practice among athletes and those who engage in regular exercise, attenuates exercise-induced oxidative stress and some of the associated benefits. Given the low incidence of nutritional deficiency in athletes and the

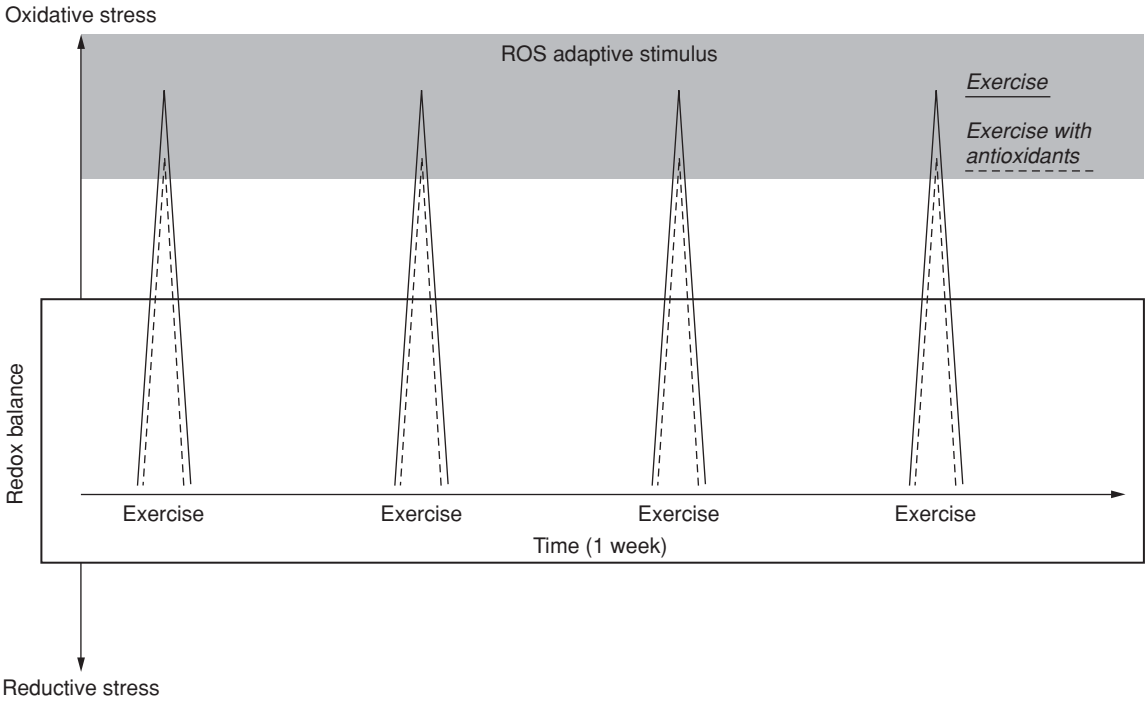

Figure 21.3 Working model of redox balance, antioxidants, and exercise adaptations. Over the course of a week, every-other-day exercise elicits a transient but potent ROS-dependent adaptive stimulus (solid lines). Exposure to the ROS-dependent adaptive stimulus during identical exercise in those consuming supplemental antioxidants (dashed lines) is attenuated.

importance of ROS for stimulating beneficial exercise adaptations, the unofficial consensus is that antioxidant supplementation is not warranted to prevent exercise-induced oxidative stress. Redox biology is a network of oxidant producing systems and antioxidant counterparts. Ongoing research efforts should employ the most rigorous experimental methodology borrowed from other disciplines, including biochemistry and molecular biology, to better understand oxidative stress in the applied work of exercise science. Exercise and nutritional scientists and practitioners should also work together to educate athletes and exercise consumers to the current scientific understanding regarding antioxidant supplements and exercise-induced oxidative stress. Education efforts should consider consumer demand for pill-based solutions to complex problems like exercise performance enhancement and antiaging desires. Further efforts should also reveal discrepancies between the scientific foundation and business-driven research which occasionally provides anecdotal support for antioxidants as a necessary countermeasure to exercise-induced oxidative stress.

References

Alessio, H., Hagerman, A., Fulkerson, B., et al. (2000). Generation of reactive oxygen species after exhaustive and isometric exercise. *Medicine & Science in Sports & Exercise* 32, 1576–1581.

Anderson, E.J., Yamazaki, H., & Neufer, P.D. (2007). Induction of endogenous uncoupling protein 3 suppresses mitochondrial oxidant emission during fatty acid-supported respiration. *Journal of Biological Chemistry* 282, 31257–31266.

Asha Devi, S., Prathima, S., & Subramanyam, M.V. (2003) Dietary vitamin E and physical exercise: II. Antioxidant status and lipofuscin-like substances in aging rat heart. *Experimental Gerontology* 38, 291–297.

Ashton, T., Young, I.S., Peters, J.R., et al. (1999) Electron spin resonance spectroscopy, exercise, and oxidative stress: an ascorbic acid intervention study. *Journal of Applied Physiology* 87, 2032–2036.

Boveris, A. & Chance, B. (1973) The mitochondrial generation of hydrogen peroxide. General properties and effect of hyperbaric oxygen. *Biochemical Journal* 134, 707–716.

Buettner, G.R. (1993) The pecking order of free radicals and antioxidants: lipid peroxidation, alpha-tocopherol, and ascorbate. *Archives of Biochemistry and Biophysics* 300, 535–543.

Cao, G. & Prior, R.L. (1998) Comparison of different analytical methods for assessing total antioxidant capacity of human serum. *Clinical Chemistry* 44, 1309–1315.

Coombes, J.S. & Fassett, R.G. (2011) Antioxidant therapy in hemodialysis patients: a systematic review. *Kidney International* 81, 233–246.

Davies, K.J., Quintanilha, A.T., Brooks, G.A., et al. (1982) Free radicals and tissue damage produced by exercise. *Biochemical and Biophysical Research Communications* 107, 1198–1205.

Fisher, G., Schwartz, D.D., Quindry, J., et al. (2011) Lymphocyte enzymatic antioxidant responses to oxidative stress following high-intensity interval exercise. *Journal of Applied Physiology* 110, 730–737.

Frei, B., Stocker, R., & Ames, B.N. (1988) Antioxidant defenses and lipid peroxidation in human blood plasma. *Proceedings of the National Academy of Sciences of USA* 85, 9748–9752.

Garelnabi, M., Veledar, E., White-Welkley, J., et al. (2011) Vitamin E differentially affects short term exercise induced changes in oxidative stress, lipids, and inflammatory markers. *Nutrition, Metabolism, and Cardiovascular Diseases* 22, 907–913.

Gilliam, L.A., Moylan, J.S., Patterson, E.W., et al. (2011) Doxorubicin acts via mitochondrial ROS to stimulate catabolism in C2C12 myotubes. *American Journal of Physiology. Cell Physiology* 102, C195–202.

Gomez-Cabrera, M.C., Domenech, E., & Vina, J. (2008) Moderate exercise is an antioxidant: upregulation of antioxidant genes by training. *Free Radical Biology & Medicine* 44, 126–131.

Halliwell, B. & Gutteridge, J. (1999) *Free Radicals in Biology and Medicine*, 3rd edn. Oxford University Press, New York.

Hamilton, K.L., Staib, J.L., Phillips, T., et al. (2003) Exercise, antioxidants, and HSP72: protection against myocardial ischemia/reperfusion. *Free Radical Biology & Medicine* 34, 800–809.

Hudson, M.B., Hosick, P.A., McCaulley, G.O., et al. (2008) The effect of resistance exercise on humoral markers of oxidative stress. *Medicine & Science in Sports & Exercise* 40, 542–548.

Jackson, M. (1998) Free radical mechanisms in exercise-related muscle damage. In *Oxidative Stress in Skeletal Muscle* (eds A.Z. Reznick, L. Packer, C.K. Sen, et al.), pp. 75–87. Birkhauser Verlag, Basel, Switzerland.

Ji, L.L. (1995) Exercise and oxidative stress: role of the cellular antioxidant systems. In *Exercise and Sport Science* (ed. J.O. Holloszy), pp. 135–166. Williams & Wilkins, Baltimore, MD.

Jones, D.P. (2006) Disruption of mitochondrial redox circuitry in oxidative stress. *Chemico-Biological Interactions* 163, 38–53.

Kaikkonen, J., Kosonen, L., Nyyssonen, K., et al. (1998) Effect of combined coenzyme Q10 and d-alpha-tocopheryl acetate supplementation on exercise-induced lipid peroxidation and muscular damage: a placebo-controlled double-blind study in marathon runners. *Free Radical Research* 29, 85–92.

Kavazis, A.N., McClung, J.M., Hood, D.A., et al. (2008) Exercise induces a cardiac mitochondrial phenotype that resists apoptotic stimuli. *American Journal of Physiology. Heart and Circulatory Physiology* 294, H928–H935.

Kavazis, A.N., Smuder, A.J., Min, K., et al. (2010) Short-term exercise training protects against doxorubicin-induced cardiac mitochondrial damage independent of HSP72. *American Journal of Physiology. Heart and Circulatory Physiology* 299, H1515–HI524.

Matsumoto, A., Mason, S.R., Flatscher-Bader, T., et al. (2011) Effects of exercise and antioxidant supplementation on endothelial gene expression. *International Journal of Cardiology* 158, 59–65.

McAnulty, S.R., Hosick, P.A., McAnulty, L.S., et al. (2007) Effect of pharmacological lowering of plasma urate on exercise-induced oxidative stress. *Applied Physiology, Nutrition, and Metabolism* 32, 1148–1155.

McNeilly, A.M., Davison, G.W., Murphy, M.H., et al. (2011) Effect of alpha-lipoic acid and exercise training on cardiovascular disease risk in obesity with impaired glucose tolerance. *Lipids Health in Disease* 10, 217.

Packer, L., Gohil, K., Delumen, B., et al. (1986) A comparative study on the effects of ascorbic acid deficiency and supplementation on endurance and mitochondrial oxidative capacities in various tissues of the guinea pig. *Comparative Biochemistry and Physiology B* 83, 235–240.

Powers, S.K., Smuder, A.J., Kavazis, A.N., et al. (2010) Experimental guidelines for studies designed to investigate the impact of antioxidant supplementation on exercise performance. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 2–14.

Quindry, J.C., Stone, W.L., King, J., et al. (2003) The effects of acute exercise on neutrophils and plasma oxidative stress. *Medicine & Science in Sports & Exercise* 35, 1139–1145.

Quindry, J.C., McAnulty, S.R., Hudson, M.B., et al. (2008) Oral quercetin supplementation and blood oxidative capacity in response to ultramarathon competition. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 601–616.

Quindry, J., Miller, L., McGinnis, G., et al. (2011) Muscle fiber type and blood oxidative stress following eccentric exercise. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 462–470.

Reid, M.B. (2001) Invited Review: redox modulation of skeletal muscle contraction: what we know and what we don't. *Journal of Applied Physiology* 90, 724–731.

Reid, M.B. (2008) Free radicals and muscle fatigue: of ROS, canaries, and the IOC. *Free Radical Biology & Medicine* 44, 169–179.

Reid, M.B., Shoji, T., Moody, M.R., et al.(1992) Reactive oxygen in skeletal muscle. II. Extra cellular release of free radicals. *Journal of Applied Physiology* 73, 1805–1809.

Ristow, M., Zarse, K., Oberbach, A., et al. (2009) Antioxidants prevent health-promoting effects of physical exercise in humans. *Proceedings of the National Academy of Sciences of USA* 106, 8665–8670.

Ruffmann, R. & Wendel, A. (1991) GSH rescue by N-acetylcysteine. *Klinische Wochenschrift* 69, 857–862.

Strobel, N.A., Peake, J.M., Matsumoto, A., et al. (2011) Antioxidant supplementation reduces skeletal muscle mitochondrial biogenesis. *Medicine & Science in Sports & Exercise* 43, 1017–1024.

Traber, M.G. (1994) Determinants of plasma vitamin E concentrations. *Free Radical Biology & Medicine* 16, 229–239.

Yang, M., Chung, S.J., Chung, C.E., et al. (2011) Estimation of total antioxidant capacity from diet and supplements in US adults. *British Journal of Nutrition* 106, 254–263.

Zergeroglu, M.A., McKenzie, M.J., Shanely, R.A., et al. (2003) Mechanical ventilation-induced oxidative stress in the diaphragm. *Journal of Applied Physiology* 95, 1116–1124.

Chapter 22

Dietary Phytochemicals

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Introduction

The use of phytochemicals as dietary supplements and food additives to maintain health, prevent disease, and improve performance is expanding rapidly. The purported benefits of phytochemicals have been attributed to their ability to function as antioxidants, anti-inflammatories, antimicrobials, antivirals, and anticancer agents as well as acting as mitochondrial "boosters," anti-aging agents, and central nervous system (CNS) stimulants (Manach et al., 2009; Spencer, 2010; Vang et al., 2011). They are easily accessible, less expensive, and generally have fewer side effects than manufactured drugs. Although traditional Greek, Indian, and Chinese medicine have used herbs and botanicals for thousands of years, only recently have these substances been subjected to systematic experimental validation. The potential ramifications of this effort go far beyond the traditional benefits provided by carbohydrates, proteins, fats, and even vitamins and minerals as components of a healthy diet and represent a new frontier in nutritional science.

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This chapter provides a brief overview of the potential effects of dietary phytochemicals on sports performance, including a definition and categorization of phytochemicals, hypothesized mechanisms of performance benefits, and a critical evaluation of the scientific evidence for selected phytochemicals that have received the most attention in this area. In addition, it will offer considerations for future research that must be addressed before appropriate conclusions can be made about their performance benefits. The chapter will not address the rapidly growing body of knowledge regarding the potential health benefits of dietary phytochemicals (Aggarwal et al., 2009; Crozier et al., 2009; Vang et al., 2011).

Defining Phytochemicals: Nomenclature, Structure, and General Function

A phytochemical is a general term used to describe a bioactive food component found in plants. They are typically produced as a natural defense against disease, competing plants, insects, and infection. Like vitamins and minerals, they offer no energy-based nutritional value (i.e., calories) for the consumer. However, unlike many vitamins and minerals that primarily function as antioxidants through

their ability to scavenge reactive oxygen species (ROS), most phytochemicals exert their effects primarily by interacting with specific proteins central to intracellular signaling cascades that modulate genes related to energy metabolism, inflammation, immune function, growth differentiation, and cell cycle control, among others (Williams et al., 2004). Phytochemicals are commonly described as antioxidants due to their chemical structure, but their H-donating activity *in vivo* is minimal at best, given their very low physiological concentrations, among other things (Spencer, 2010).

Phytochemicals include phenolic compounds, polyphenols, flavonoids, and many other chemical species. The two most common methods of categorization are by molecular structure and plant origin. Structurally, phytochemicals can be grouped according to their number of phenol rings and the structural elements that bind these rings to one another (Figure 22.1). The smallest are the phenolic acids such as gallic acid and caffeic acid, which typically contain seven carbon atoms and one phenolic ring. They are predominantly found in dried fruits following the decomposition of larger phytochemicals. They are also found as components of larger polyphenols. Phenolic acids have been researched less than other phytochemicals, presumably because of their meager concentration in edible plants. Stilbenes, found in the skins and seeds of grapes, are larger and contain two phenolic rings with 14 carbon atoms. Stilbenes are also found in very low quantities in the human diet, but as supplements have received much attention recently because of the exciting preclinical evidence involving the health and performance benefits of resveratrol (Lagouge et al., 2006; Sun et al., 2011; Vang et al., 2011).

Flavonoids are large polyphenolic compounds and make up the largest group of phytochemicals found in vegetables, berries, fruits, and cereals. They contain between two and four phenolic rings and are grouped into six general categories (Figure 22.1): (1) Flavonols, the largest subdivision, includes quercetin and kaempferol. Quercetin has been the focus of more sports performance-based research than any other phytochemical and is becoming a popular ingredient in ergogenic aids. Flavonols can be found in onions, broccoli, apples, berries, and others. (2) Flavones, such as apigenin and luteolin, are less common than flavonols and have been identified only in celery and parsley. (3) Anthocyanidins (including cyanidin and malvidin) are most commonly found in fruits. (4) Flavanols are a unique subdivision in that they can be found as monomers (catechins) or as polymers (proanthocyanidins). The richest sources of flavanols are green tea and chocolate. A flavanol that has gained notoriety for increasing fat utilization is epigallocatechin gallate (ECGC), a primary ingredient in tea extract. (5) Isoflavones like genistein and daidzein are compounds with structures strikingly similar to estrogens, earning them the term phytoestrogens. Isoflavones are found almost exclusively in soy products. (6) Flavanones including naringenin and hesperetin, found almost exclusively in citrus fruits, are thought to play an important role in immune function.

Various spices also contain polyphenolic phytochemicals. One such phytochemical, curcumin has generated recent interest given its powerful anti-inflammatory activity (Aggarwal et al., 2009). It is the most bioactive curcuminoid found in the herb turmeric (*Curcuma longa*), which is a primary component of curry used often in Asian cuisine. It has only two phenolic rings, but has more total carbons (21) than most of the flavonoids from fruits, vegetables, and berries (Figure 22.1).

Effects derived from phytochemicals could have major applications in sports performance, although most of the research to date has focused on general health and wellbeing. Originally, phytochemicals were sought by athletes because of their antioxidant activity (Figure 22.2); exercise-induced free radicals that can contribute to muscle fatigue were thought to be reduced by dietary antioxidant. However, this activity, while powerful *in vitro*, is severely reduced *in vivo* (Spencer, 2010). Further, recent scientific evidence has shown that the fatigue-reducing ability of antioxidants including vitamins and minerals is minimal (Powers & Jackson, 2008). Therefore, more recent work has focused on other properties of phytochemicals know to affect performance, in particular their powerful anti-inflammatory and metabolic effects that are mediated through their influence on intracellular signaling pathways. The bulk of the

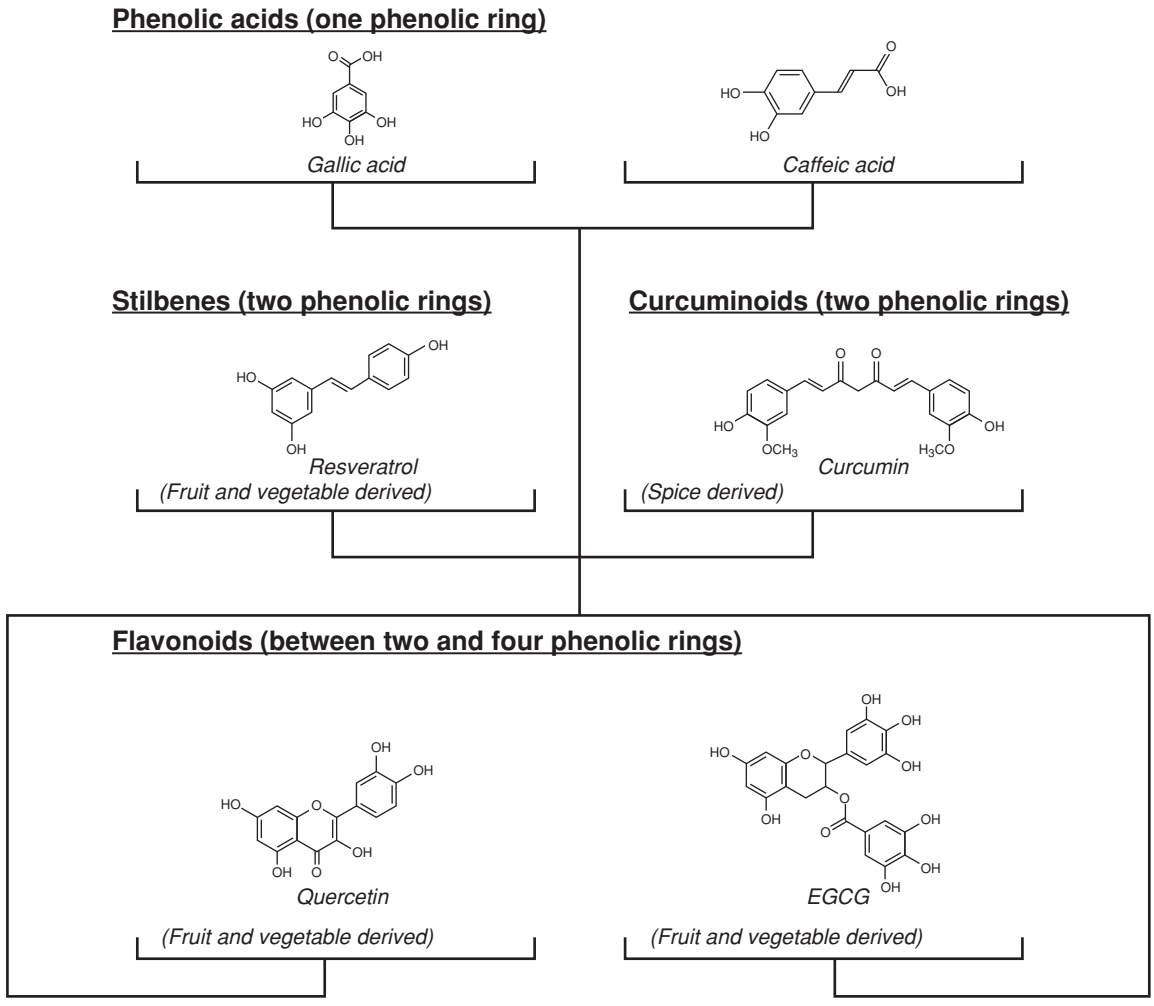

Figure 22.1 Major classes of phytochemicals discussed in this chapter including phenolic acids, stilbenes, curcuminoids, and flavonoids. The classes of phytochemical are based on chemical structure.

available research involves resveratrol, quercetin, ECGC, and curcumin, and this will be the focus of the rest of this chapter.

Phytochemicals and Exercise Performance

Investigation into the science of plant extracts and phytochemicals on mental and physical performance has increased dramatically in recent years. While novel phytochemicals are abundant within sports drinks and energy bars, few of these compounds have been subjected to the extensive investigation that is required to establish their ergogenic potential.

Quercetin

The flavonol quercetin has generated much interest for possible benefits to endurance performance. The first study to investigate possible benefits of quercetin on exercise performance was reported by MacRae and Medford (2006). Using a placebo-controlled study design they showed that a commercial

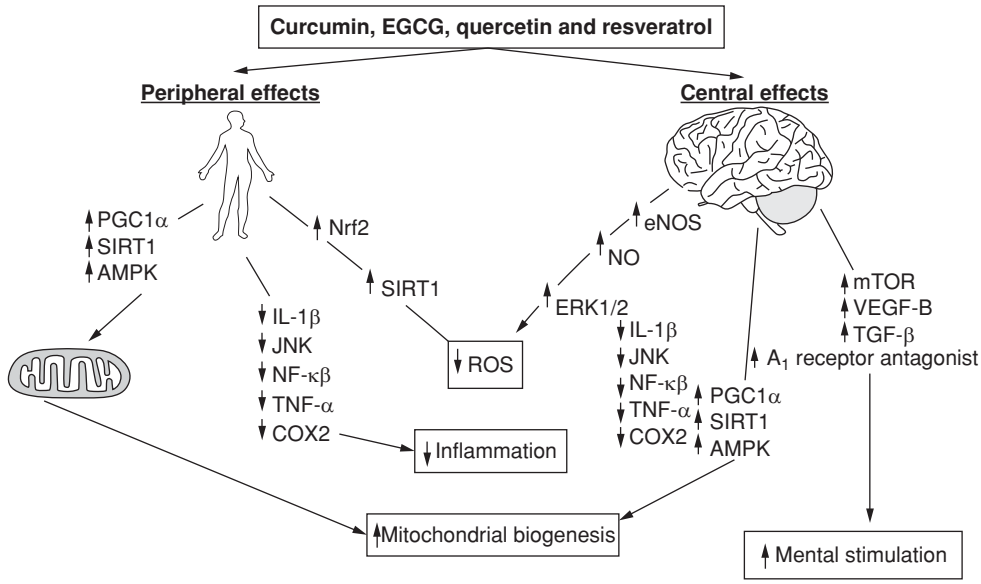

Figure 22.2 Proposed mechanisms by which curcumin, EGCG, quercetin, and resveratrol exert beneficial performance effects, both centrally and peripherally. The proposed mechanisms ultimately lead to decreases in reactive oxygen species (ROS) and inflammation and increases in mitochondrial biogenesis and mental stimulation.

beverage containing quercetin (FRS, The FRS® Company, Foster City, CA) administered at 600 mg/day (2 × 300 mg) for 6 weeks improved bike time trial performance in trained cyclists (MacRae & Mefferd, 2006). However, the small performance benefit could not be credited to quercetin alone since FRS contains small amounts of caffeine, tea catechins, and other ingredients that may have influenced this response. Nonetheless, this and a large amount of preclinical data spurred interest in quercetin as a potential ergogenic aid. Davis et al. (2009), following unpublished observations of a quercetin-induced upregulation of mitochondrial genes in cell culture, examined the effects of 7-day feedings of quercetin on mitochodrial biogenesis in both muscle and brain in mice and related this to changes in exercise performance. The short-term quercetin feedings increased various markers of mitochondrial biogenesis, including peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α), sirtuin 1 (SIRT1), cytochrome *c* gene expression, and mitochondrial DNA (mtDNA) in both skeletal muscle and brain. Further, these changes were associated with an increase in both forced treadmill running and voluntary wheel running. These findings were partially confirmed in two human studies (Davis et al., 2010; Nieman et al., 2010). Using a placebo-controlled crossover design, Davis et al. (2010) found an increase in both VO_2 max (3.9%) and bicycle ride time to fatigue (13.2%) following 7 days of quercetin (500 mg twice daily) in healthy untrained men and women. Similarly, Nieman et al. (2010), using a comparable crossover study design and dosage (1 g/day for 2 weeks), found a 2.9% increase in distance covered in a 12-minute treadmill time trial performance in untrained subjects. They also found consistent, albeit nonsignificant, increases in expression of genes associated with mitochondrial biogenesis in skeletal muscle.

However, not all studies have been positive as several have failed to find significant benefits of quercetin. Quindry et al. (2008) reported that quercetin supplementation (250 mg × 4, daily for 3 weeks) had no effects on race performance at the Western States 100-mile race. Similarly, Cureton et al. (2009) reported no benefits of quercetin on peak oxygen uptake, work performed on a 10-minute maximal efficiency cycling test, perception of effort, or

voluntary and electrically evoked strength loss in untrained men following 1 g of quercetin/day for 7–16 days. Similarly, 6 weeks of quercetin supplementation (1 g/day) was reported to have no effects on energy levels, fatigue, or sleep quality in soldiers during military training (Bigelman et al., 2011). In addition, Nieman et al. (2009) found no effect of quercetin or quercetin blended with EGCG on mitochondrial biogenesis and immunity in highly trained cyclists. In light of these inconsistent findings, a recent meta-analysis was conducted to evaluate the overall effect of quercetin as presented in the literature. It was concluded that there is a small (~2%), but significant, benefit of quercetin on endurance performance (Kressler et al., 2011).

Although the evidence for a beneficial effect of quercetin upon endurance performance is promising, strong conclusions are not yet possible given the dearth of sophisticated clinical trials. Further, the mechanisms responsible for these benefits have not been fully elucidated. Although increased mitochondrial biogenesis has been suggested as one possibility, there are currently no systematic mechanistic studies that demonstrate a specific cause and effect relationship between mitochondrial biogenesis and endurance performance in response to quercetin supplementation.

Resveratrol

Like quercetin, the animal studies on resveratrol show impressive benefits on endurance exercise. Perhaps the most frequently cited of these is a study by Lagouge et al. (2006) who found that 15 weeks of resveratrol feedings (400 mg/kg/day) in conjunction with a high-fat diet increased aerobic capacity that was associated with increased markers of mitochondrial biogenesis (SIRT1, PGC-1α, and citrate synthase), along with decreased fasting glucose and triglycerides and increased insulin sensitivity. In addition, the myofibers from resveratrol-treated mice experienced strikingly similar changes to those that are seen with aerobic exercise training, i.e., they were heavily saturated with mitochondria, exhibited enhanced oxidative capacity, and had a higher resistance to fatigue. Lastly, the authors also noted improvements in motor coordination, which is likely indicative of a central effect of resveratrol. Since this landmark study, Baur et al. (2006) found an increase in AMPK and PGC1-α gene expression as well as increased number of mitochondria per muscle cell. Another study performed by Murase et al. supplemented the diet of senescence-accelerated prone mice with resveratrol (0.2% w/w) in combination with exercise training for 12 weeks. The combination treatment improved endurance capacity, relative to exercise alone. In addition, the resveratrol-alone group was found to have more overall muscle mass following the 12 weeks of treatment compared to the control and exercise groups (Murase et al., 2009). Lastly, a study performed by Sun et al. (2011) found that exhaustive exercise combined with a blend of nutrients (including 5 mg/kg resveratrol) improved time to exhaustion on a treadmill in rats that was consistent with an increase in mitochondrial biogenesis. However, given that a blend of nutrients was used, these improvements cannot be attributed to resveratrol alone (Sun et al., 2011). However, these positive findings have not yet been confirmed in humans. In fact, there are no published clinical trials on the effects of resveratrol on human performance.

Review of the current resveratrol studies on endurance performance markers appears promising, yet there is a lack of substantial evidence, particularly from clinical trials. Like quercetin, the mechanisms for the benefits of resveratrol on endurance performance are not fully understood. Many of the animal studies suggest that these effects are mediated, at least in part, by alterations in AMPK and PGC-1α activity. This has led to investigations of synthetic pharmaceuticals that activate these molecules and essentially mimic some of the benefits of exercise (Narkar et al., 2008). However, the physiological adaptations resulting from exercise cannot be duplicated with a single pharmaceutical agent (Hawley & Holloszy, 2009). And there are no such studies in humans.

Green Tea Extract/EGCG

Green tea extract (GTE) and its components, in particular EGCG, have also been investigated for their potential benefits to performance. This is driven

largely by studies that have documented their ability to increase fat oxidation in human subjects (Gregersen et al., 2009), as well as performance enhancement in rodents (Murase et al., 2008). For example, consumption of GTE at a dose of 0.5% for 8–10 weeks increased endurance performance in mice by 30% which was accompanied by lower RER (respiratory exchange ratio), higher β-oxidation, lower malonyl-CoA, as well as lower lactate and higher FFA (free fatty acids) concentrations. However, the available human literature on performance is not as promising. A small increase in VO_2 max, independent of any changes in work rate, maximal RER, HR, or maximum cardiac output, was reported following ECGC (135 mg/day) treatment (Richards et al., 2010), while Eichenberger et al. (2010) reported no benefit of GTE (159 mg/day for 3 weeks) on time trial performance or energy metabolism in trained cyclists (Eichenberger et al., 2010). Likewise, Dean et al. (2009) reported no effects of EGCG on fat oxidation or cycling performance. Therefore, as is the case with quercetin and resveratrol, the animal evidence is rather convincing, but these effects have not been substantiated in human studies.

Inflammation and Performance

Nonsteroidal anti-inflammatory drugs (NSAIDs) like ibuprofen, indomethacin, Vioxx, and Celebrex are the most commonly used drugs by athletes of all levels (Ciocca, 2005) primarily in attempts to improve performance by alleviating pain, muscle soreness, stiffness, and overall inflammation that occurs as a result of intense exercise or competition. However, NSAID use has been associated with serious side effects, including gastrointestinal distress and cardiovascular complications (Mukherjee et al., 2001). Some phytochemicals, which are generally free of these undesirable side effects, possess powerful anti-inflammatory activity and are now being investigated as potential recovery aids following physical performance. Although certain phytochemicals like curcumin, quercetin, EGCG, and ellagitannin have well-documented anti-inflammatory activities, their role in recovery of exercise-induced inflammation has not yet been firmly established.

Curcumin

The spice derivative curcumin has been investigated by modern scientific methodology in a variety of anti-inflammatory models including exercise-induced muscle injury, but its efficacy as an anti-inflammatory agent has been known in Asia for centuries (Aggarwal et al., 2009). Its anti-inflammatory mechanisms are now well characterized and include alterations in various intracellular signaling pathways, especially the inhibition of the NF-$\kappa\beta$ pathway (Aggarwal et al., 2009). Inhibition of these pathways can reduce the expression of many inflammatory mediators that are associated with impaired performance, including COX-2, TNF-α, IL-1β, and IFN-γ (Carmichael et al., 2005; Malm et al., 2004). However, to date there is only one report of a benefit of curcumin on exercise-induced muscle damage. Davis et al. (2007) used a well-characterized mouse model of downhill running to examine the effects of curcumin on muscle inflammation and performance recovery. Curcumin (10 mg/day) was administered in a rodent pellet for 3 days prior to a 90-minute novel downhill running bout. Downhill running significantly increased the proinflammatory cytokine (IL-1β, IL-6, and TNF-α) response in the muscle, which was blocked by curcumin. Further, this apparent reduction in inflammation was associated with enhanced recovery of both voluntary wheel running and forced treadmill running. Once again, although the clinical utility seems promising, there are no available human studies. There are, however, several ongoing large-scale clinical trials of curcumin in inflammatory diseases, including colitis, cancer, arthritis, and others.

Quercetin

Quercetin, like curcumin, which has a similar chemical structure (Figure 22.1), also has relatively powerful anti-inflammatory activity given its ability to inhibit the activation of NF-$\kappa\beta$ (Harwood et al., 2007). Preliminary data from our lab indicate that quercetin, like curcumin, can improve performance recovery following downhill running in mice. There are a few available clinical investigations that have examined the anti-inflammatory effects of quercetin

following exercise. For example, Nieman et al. reported an attenuation of IL-8 and IL-10 mRNA in blood leukocytes in quercetin (1000 mg/day for 21 days) treated ultramarathoners completing the 160-km Western States Endurance Run (Nieman et al., 2007) However, quercetin failed to decrease any of the measured markers of muscle damage, inflammation, plasma cytokines, or muscle cytokine mRNA gene expression.

In a follow-up study by the same group, the influence of quercetin (Q) with or without EGCG, isoquercetin, eicosapentaenoic acid, and docosahexaenoic acid (Q+EGCG) on inflammation was examined following 3 days of high-volume cycling using a randomized double-blind placebo control trial (Nieman et al., 2009). IL-6 and C-reactive protein were significantly decreased immediately after cycling in the Q+EGCG group but not in the Q group, but it is not clear as to whether the reduced inflammation was due to the added EGCG or isoquercetin (the same as quercetin once absorbed).

Ellagitannin and Anthocyanin

Recently, investigations involving the ellagitannins and anthocyanins (similar large phytochemicals found primarily in pomegranates and berries) and physical performance recovery following stressful exercise have begun to emerge. As with curcumin and quercetin, ellagitannin research has focused on reducing inflammation (Gonzalez-Gallego et al., 2010), with relatively few studies that focus specifically on physical performance. Trombold et al. (2010) examined the effects of 9 days of pomegranate (POMx POM Wonderful, Los Angeles, CA; 95.5% ellagitannins) treatment on muscle damage and strength performance recovery following heavy resistance training. The results indicated that the POMx treatment group produced more isometric force in the exercised muscle at 48–72 hours after resistance training. However, there were no differences detected in markers of inflammation or muscle damage, including IL-6 and C-reactive protein. Similar results of improvement in recovery were seen with anthocyanin-rich tart cherry juice (Connolly et al., 2006; Howatson et al., 2010). Connolly et al. reported that subjects who supplemented with 350 ml of tart cherry juice for 8 days prior to a muscle-damaging exercise regimen had significantly less strength loss than the placebo group at all measured time points up to 96 hours (Connolly et al., 2006). Similarly, Howatson et al. (2010) found benefits of tart cherry juice in marathon runners. While promising, there is still much to sort out regarding the specific effects of individual phytochemicals versus food extracts containing multiple phytochemicals on inflammation and performance under these conditions.

Central Nervous System and Performance

Sports performance requires not only the capacity of muscle to maintain force production, but also adequate motivation/effort, mental alertness, and clarity of thought, decision-making, and mood. CNS factors, including higher-level cognitive processing, are important components of the fatigue state, whose symptoms at onset include decreased feelings of energy, motivation, arousal, and vigor, as well as increased tiredness, perception of effort, and force sensation. These feelings of fatigue almost always occur before the muscle actually loses the ability to maintain the required force or power output. Unfortunately, the biological basis of CNS fatigue is not very well understood, which makes it difficult to develop nutritional countermeasures. Among the hypothesized CNS fatigue mechanisms are (1) inadequate energy availability from glucose metabolism; (2) imbalance among neurotransmitters/neuromodulators including dopamine, serotonin, acetylcholine, and adenosine; and (3) neuroinflammation (Carmichael et al., 2005; Davis et al., 2000), all of which lend themselves to possible phytochemical intervention.

There has also been renewed interest in flavonoid-rich food extracts and isolated flavonoids that have been used as remedies for mental fatigue and other cognitive impairments in traditional Greek and Asian medicine. The evidence from cellular and animal studies is overwhelming with regard to the benefits of diet-derived flavonoids in modulating neural processes and cognitive function (Spencer, 2010). Various flavonoids have been shown to

improve cognitive function and mood, increase excitatory neurotransmitter activity, promote blood flow and glucose metabolism, and inhibit neural inflammation (Spencer, 2010), all of which are likely to be essential to the optimum performance of an athlete. These benefits go well beyond the assumption on an antioxidant-mediated mechanism. Instead, they interact directly with and modulate critical molecular signaling pathways, transcription factors, and gene and/or protein expression that control neural processing (refer to Figure 22.2) (Spencer, 2010). For instance, phytochemicals are known to affect proteins, protein kinases, and lipid kinases signaling cascades within the CNS, effectively increasing mitochondria and neural stimulation and decreasing neuroinflammation. More specifically, certain phytochemicals are known to activate SIRT1 with subsequent deacetylation of PGC-1α increasing mitochondrial biogenesis (Davis et al., 2009; Lagouge et al., 2006). Additionally, they can activate protein kinase C and MAPK signaling, and have been shown to act as adenosine antagonists (similar to the effect of caffeine), thereby enhancing neural stimulation (Alexander, 2006). Lastly, phytochemicals inhibit inflammatory transcription factors, such as NF-$\kappa\beta$, decreasing neuroinflammation (Spencer, 2010). However, appropriate human translation of these primarily cellular and animal studies is lacking, especially with respect to enhancement of sports performance. Few studies have specifically focused on potential performance-related CNS benefits and are limited to quercetin, curcumin, ginseng, *Ginkgo biloba*, and various flavonoid-rich foods/herb extracts.

It has been suggested that quercetin could delay CNS fatigue due to its caffeine-like ability to block adenosine receptors (Alexander, 2006; Davis et al., 2003). Although many flavonoids possess similar adenosine A1 receptor antagonism in the brain, quercetin has the highest affinity of those tested (Alexander, 2006). *In vivo* evidence for this has been demonstrated in a preliminary study in our laboratory showing increased brain adenosine A1 receptor gene expression following 7 days of quercetin feedings in mice. However, in a recent report, Olson et al. (2010) found only nonsignificant benefits of a single very large dose of quercetin (2000 mg) on a visual vigilance task and subjective ratings of fatigue and vigor.

Perhaps the most important and exciting aspect of quercetin supplementation with respect to CNS fatigue is its effects on mitochondrial biogenesis in the brain, an effect expected to increase energy production via glucose metabolism. Davis et al. (2009) examined the effects of 7 days of quercetin feedings in mice on markers of mitochondrial biogenesis in the brain and on endurance exercise tolerance. Quercetin increased mRNA expression of PGC-1α and SIRT1, mtDNA, and cytochrome *c* concentration in the brain. This was associated with an increase in endurance capacity on a motor-driven treadmill, which is generally thought to be influenced primarily by peripheral fatigue factors, as well as voluntary wheel-running activity, which is driven more by CNS factors related to the motivation/willingness to run. This, along with recent preliminary fMRI evidence in humans that shows quercetin produced region-specific increases in brain blood flow (BOLD) similar to and distinct from those produced by caffeine administration during prolonged cycling to fatigue, provides promising evidence for a benefit of quercetin on CNS fatigue during prolonged exercise. All of this is of particular importance to the brain given its extraordinarily high demand for glucose and oxygen supplied via the general circulation. In fact, at rest the brain accounts for approximately 20% of total body blood glucose utilization. Adult brain stores very small quantities of glycogen and relies almost solely on glucose as a source of fuel, so maintenance of adequate glucose delivery and oxidation in order to prevent deficits in CNS function and performance is of utmost importance.

Quercetin and other flavonoids also have strong anti-inflammatory activity due to their ability to modulate transcription factors and enzymes within pathways essential for inflammatory responses, both in the peripheral nervous system and CNS. For example, quercetin can reduce the expression of pro-inflammatory cytokines such as IL-1β and TNF-α and other inflammatory mediators such as COX-2, NF-κB, and p38MAPK from many types of brain cells, including astrocytes and microglia, following treatment with inflammatory agents (Spencer, 2010).

Curcumin has even more powerful anti-inflammatory activity than quercetin and is being rigorously examined in the prevention and treatment of neuroinflammation and Alzheimer's disease (Begum et al., 2008). This is relevant to CNS fatigue because exercise-induced muscle damage induces CNS as well as muscle inflammation. The CNS inflammatory response (specifically the increase in IL-1β) contributes significantly to the performance deficits associated with exercise-induced muscle damage (Carmichael et al., 2005) and is consistent with the variety of "sickness symptoms" including fatigue that result from CNS inflammation induced by traumatic injury and disease, such as infection and cancer (Dantzer & Kelley, 2007). While the investigation of phytochemicals, brain inflammation, and subsequent CNS fatigue is still in its infancy, there is some evidence to suggest a potential benefit. Davis et al. (2007) reported that curcumin administration improved recovery of both voluntary and forced treadmill performance following downhill running in mice. This was interpreted to indicate that curcumin's anti-inflammatory effect occurred both centrally (related to the willingness/motivation to run) as well as peripherally (related to muscle-specific function) (Carmichael et al., 2005; Dantzer & Kelley, 2007). In addition, blueberry extract (containing high concentrations of several flavonoids) has been shown to inhibit inflammatory mediators released from brain microglia (IL-1β, TNF-α) following LPS treatment (Lau et al., 2007). Further research should be undertaken to determine whether these effects may also benefit performance deficits associated with overuse injuries and overtraining in athletes where chronic inflammation is a primary etiology.

There are also various reports, albeit limited, with regard to possible benefits of flavonoid supplementation on cognitive function and performance in humans. Ginseng and *Ginkgo biloba* are the most studied in this regard. Reay et al. (2005, 2006) conducted two studies on *Panax ginseng* and showed improved cognitive performance and lowered feelings of mental fatigue. The results from studies examining the effects of *Ginkgo biloba*, however, have been mixed. For example, Elsabagh et al. (2005) showed that a single dose of *Ginkgo* (120 mg) improved sustained attention and pattern recognition memory but had no effects on mood. However, another study reported that chronic administration (6 weeks) had no effect on any of the cognitive tests measured or on mood (Burns et al., 2006). Whereas, higher doses of *Ginkgo* (360 mg) and mixtures of *Ginkgo* and ginseng have been shown to produce some positive cognitive effects (Kennedy et al., 2001).

Interpreting the Science and Future Directions

Although the large number of preclinical studies provides rather consistent support for benefits of various phytochemicals on sports performance, the limited number of human studies has produced mixed results making it difficult to draw firm conclusions. The apparent discrepancies between the mostly positive effects in cellular/animal studies and inconsistent effects in human studies could easily be misinterpreted. It is often difficult to compare studies given obvious differences in subjects studied and parameters measured, research design, individual differences among participants, dosing size, timing, and delivery vehicle, among others.

Proper doses eliciting the intended molecular changes are essential for clinical translation. Complicating interpretation of the current studies, animal study doses have been wide ranging, with doses for resveratrol, for example, ranging from 0.01 to 1500 mg/kg (Smoliga et al., 2011). Different doses are necessary for different phytochemicals, different effects, and different experimental subjects. Some phytochemicals (e.g., resveratrol and quercetin) are believed to have biphasic effects. An example in resveratrol shows that low doses (~5 mg/kg/day) caused weight gain in mice on a high-fat diet (Pearson et al., 2008), while in another study high doses of resveratrol (~400 mg/kg/day) resulted in weight loss (Lagouge et al., 2006). Once an appropriate dose is found, then the real difficulty arises in terms of extrapolation to a corresponding human dose.

Established doses considered safe and effective in humans are unknown for most phytochemicals. Typically, dosing has been based on animal studies and calculated by extrapolation of body weight. Some researchers argue that doses based on body weight are not as accurate as using body surface

area (BSA) (Reagan-Shaw et al., 2008). While BSA calculations seem to offer a more accurate estimation of dose, this cannot be discerned without performing sophisticated pharmacokinetic trials. In addition to size differences, the metabolism of phytochemicals is a confounding factor when translating doses between animals and humans. Metabolizing enzymes are found in different concentrations between species, and the few completed pharmacokinetic clinical studies have shown humans as having substantially higher rates of metabolism for most phytochemicals. This argues that currently used human doses may be safe, but ineffective in eliciting cellular changes.

High rates of liver metabolism following absorption result in low plasma concentrations of the parent phytochemical. However, determining total volume distribution requires knowing tissue as well as plasma distribution. Since most phytochemicals are lipophilic, they amalgamate well with membranes, lipoproteins, and other fat molecules. Lipophilic phytochemicals could have high total volume distribution and elicit cellular effects in tissue without having measurable plasma concentrations. Additionally, albeit parent compound plasma concentrations are low, their specific metabolites (e.g., sulfates, methylates, gluconates) may be high, which makes sense given that these metabolites are often much more water soluble. In some cases, these metabolites have been found to have more cellular effect than parent compounds (Manach et al., 2009), and some have been shown to revert to the parent compound upon entering body cells (Spencer, 2011). Finally, what appears to be very large individual differences among human subjects in the pharmacokinetics of most phytochemicals is perhaps more important. For example, we have found greater than sixfold differences in plasma concentrations of total quercetin among subjects following consumption of a 500 mg dose of quercetin (Figure 22.3), which also occurs with chronic doses (Jin et al., 2010). This would be especially troublesome in small sample size studies. Given this, subjects should probably be prescreened and grouped based on their pharmacokinetic response prior to examining the effects of these compounds.

Phytochemicals are also highly interactive with other compounds including enzymes, matrix components in a food extract, prescription drugs, and other phytochemicals, potentially altering the necessary dose (Aggarwal et al., 2009; Vang et al., 2011). Many researchers believe that blending phytochemicals or using them in a similar matrix as they occur in foods could be beneficial, emphasizing synergistic interactions (Aggarwal et al., 2009; Majumdar et al., 2009). However, little is known about these interactions.

Current published clinical trials have also used a wide range of study designs and methodologies that make it very difficult to compare studies. Perhaps more importantly, negative findings may be the result of a poor study design and methods rather than the lack of a biological effect. These differences may include subjects, environmental factors, test compounds and delivery methods, parameters measured, and limitations in trial design (Figure 22.4). In the clinical studies discussed in this review, only five utilized a double-blind, placebo-controlled crossover study design (Connolly et al., 2006; Davis et al., 2010; MacRae & Mefferd, 2006; Nieman et al., 2009; Trombold et al., 2010), the benchmark for trial design, and yielded significant positive results, while clinical trials using parallel/cross-sectional design were much less likely to find significance.

In addition to trial design, differences in subject demographics between investigations can also greatly modify results. Performance results can be highly influenced by the subject's fitness level. Additionally, inclusion criteria in the published clinical trials have varied greatly. Trials with more rigorous inclusion criteria generally have greater uniformity between subjects and therefore less likelihood of confounding subject differences. This subject variability is magnified in cross-sectional studies, because small groups are compared that may be different initially and may respond differently to the compounds tested.

Along with subject variability, test conditions can also change trial results. Trials conducted within laboratory settings help minimize the effect of individual variability on results. Environmental factors such as wind, temperature, humidity, and distractions can decrease the ability of finding a false-negative result. This is exaggerated in studies with small sample

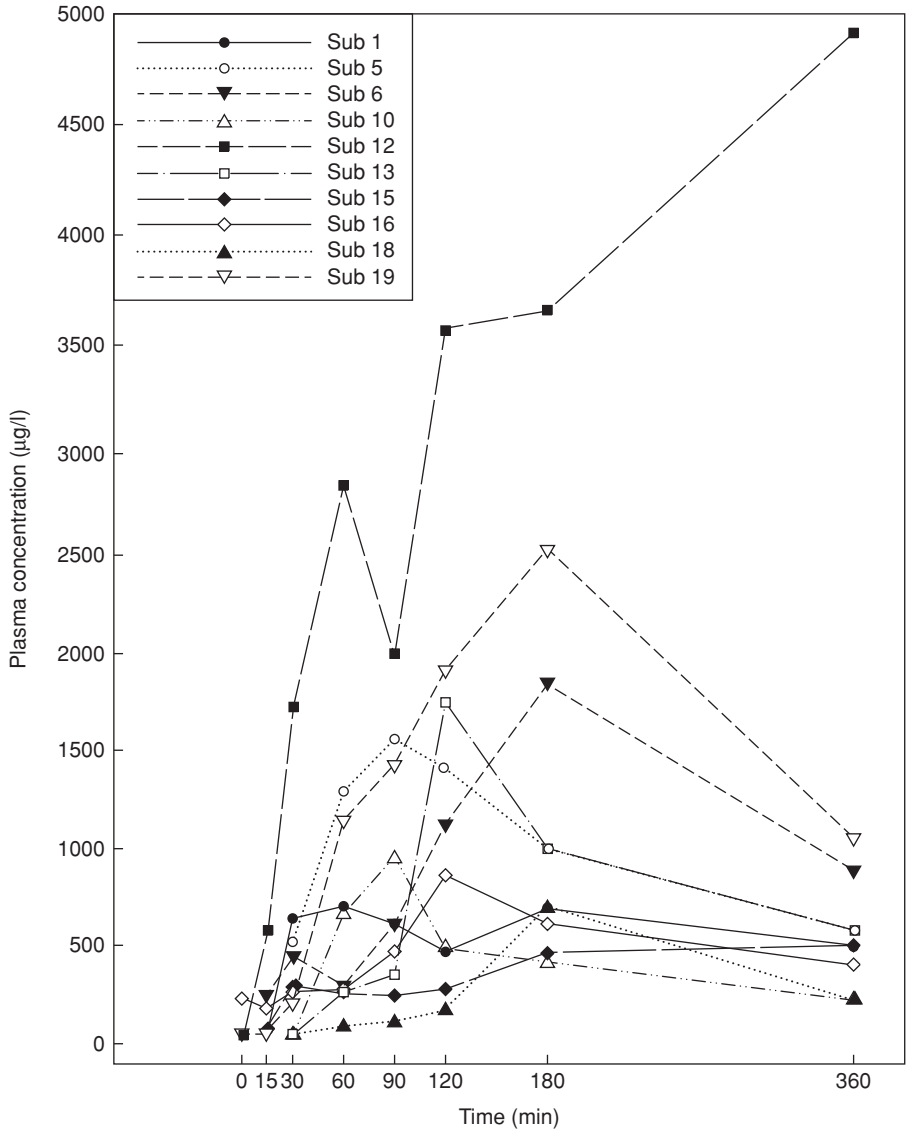

Figure 22.3 Intersubject variability in plasma concentration of total quercetin (quercetin and its methylated, sulfated, and glucanated conjugants) in fasted human subjects (n–10) following ingestion of 500 mg of quercetin.

sizes. In addition, measurements must be sensitive enough to detect a treatment effect. Improper measurements are found throughout the phytochemical clinical trials. An example can be found in a quercetin study performed on untrained cyclists, where an indirect measure of mitochondrial function was used that may not have been sensitive enough to reliably detect small increases in mitochondrial biogenesis, especially given the cross-sectional research design (Cureton et al., 2009).

Lastly, discrepancies in pretest meals and lack of control of the diet throughout the study (e.g., eating

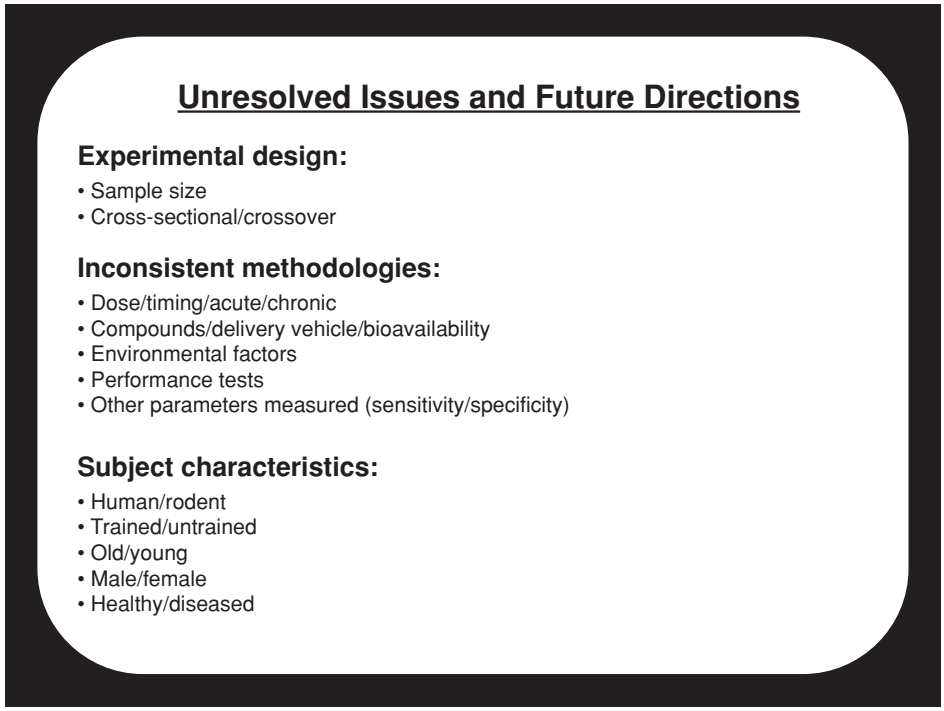

Figure 22.4 Unresolved issues regarding current phytochemical research that must be addressed before firm conclusions can be made with regard to the safety and efficacy of phytochemicals on exercise performance.

large amounts of foods containing the test compound) can create differences in results. Moreover, differences in the vehicle used to administer the treatment can have profound influences. For example, in the clinical studies presented in this chapter, the phytochemicals have been given in powder, liquid extracts sports drinks, pills, juice, and soft chews. Clearly, the bioavailability of most phytochemicals is greatly affected by the type and composition of the delivery vehicle (Goldberg et al., 2003).

Conclusions

It is difficult to accurately evaluate the safety and efficacy of phytochemical interventions at this early stage of investigation. Despite overwhelming evidence from cellular and animal research that supports the benefits of various phytochemicals on central and peripheral factors involved in athletic performance, the clinical translation remains unclear primarily due to the limited number of sophisticated and systematic studies. Significant differences in subjects studied and parameters measured, along with limitations in trial design, dose and delivery methods, number of participants, and individual differences among participants, greatly affect the outcome of the studies (Figure 22.4). At minimum, there are now substantial data to support testable hypotheses regarding phytochemical effects that go far beyond their typically studied antioxidant activity to the more powerful modulation of molecular signaling pathways that control the genetic regulation of cellular function. This forms the bases of a new field of study called nutrigenomics that in conjunction with the rapidly growing number of bioactive phytochemicals with potential benefits to mental and physical performance could turn out to be an exciting new frontier in sports nutrition research. Clearly, more sophisticated and systematic clinical trials are necessary to accurately assess the potential full range of benefits of dietary phytochemicals on athletic performance, as well as a clearer understanding of the mechanisms of action.

References

Aggarwal, B.B., Van Kuiken, M.E., Iyer, L.H., et al. (2009) Molecular targets of nutraceuticals derived from dietary spices: potential role in suppression of inflammation and tumorigenesis. *Experimental Biology and Medicine (Maywood, N.J.)* 234, 825–849.

Alexander, S.P. (2006) Flavonoids as antagonists at A1 adenosine receptors. *Phytotherapy Research* 20, 1009–1012.

Baur, J.A., Pearson, K.J., Price, N.L., et al. (2006) Resveratrol improves health and survival of mice on a high-calorie diet. *Nature* 444, 337–342.

Begum, A.N., Jones, M.R., Lim, G.P., et al. (2008) Curcumin structure-function, bioavailability, and efficacy in models of neuroinflammation and Alzheimer's disease. *Journal of Pharmacology and Experimental Therapeutics* 326, 196–208.

Bigelman, K.A., Chapman, D.P., Freese, E.C., et al. (2011) Effects of 6 weeks of quercetin supplementation on energy, fatigue, and sleep in ROTC cadets. *Military Medicine* 176, 565–572.

Burns, N.R., Bryan, J., & Nettelbeck, T. (2006) *Ginkgo biloba*: no robust effect on cognitive abilities or mood in healthy young or older adults. *Human Psychopharmacology* 21, 27–37.

Carmichael, M.D., Davis, J.M., Murphy, E.A., et al. (2005) Recovery of running performance following muscle-damaging exercise: relationship to brain IL-1beta. *Brain, Behavior, and Immunity* 19, 445–452.

Ciocca, M. (2005) Medication and supplement use by athletes. *Clinical Sports Medicine* 24, 719–738, x–xi.

Connolly, D.A., McHugh, M.P., Padilla-Zakour, O.I., et al. (2006) Efficacy of a tart cherry juice blend in preventing the symptoms of muscle damage. *British Journal of Sports Medicine* 40, 679–683; discussion 683.

Crozier, A., Jaganath, I.B., & Clifford, M.N. (2009) Dietary phenolics: chemistry, bioavailability and effects on health. *Natural Product Reports* 26, 1001–1043.

Cureton, K.J., Tomporowski, P.D., Singhal, A., et al. (2009) Dietary quercetin supplementation is not ergogenic in untrained men. *Journal of Applied Physiology* 107, 1095–1104.

Dantzer, R. & Kelley, K.W. (2007) Twenty years of research on cytokine-induced sickness behavior. *Brain, Behavior, and Immunity* 21, 153–160.

Davis, J.M. (2000) Nutrition, neurotransmitters and central nervous system fatigue. In *Encyclopaedia of Sports Medicine: Nutrition in Sport* (ed. R.J. Maughan), pp. 171–183. Blackwell Science, Oxford.

Davis, J.M., Zhao, Z., Stock, H.S., et al. (2003) Central nervous system effects of caffeine and adenosine on fatigue. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 284, R399–R404.

Davis, J.M., Murphy, E.A., Carmichael, M.D., et al. (2007) Curcumin effects on inflammation and performance recovery following eccentric exercise-induced muscle damage. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 292, R2168–R2173.

Davis, J.M., Murphy, E.A., Carmichael, M.D., et al. (2009) Quercetin increases brain and muscle mitochondrial biogenesis and exercise tolerance. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 296, R1071–R1077.

Davis, J.M., Carlstedt, C.J., Chen, S., et al. (2010) The dietary flavonoid quercetin increases VO(2max) and endurance capacity. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 56–62.

Dean, S., Braakhuis, A., & Paton, C. (2009) The effects of EGCG on fat oxidation and endurance performance in male cyclists. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 624–644.

Eichenberger, P., Mettler, S., Arnold, M., et al. (2010) No effects of three-week consumption of a green tea extract on time trial performance in endurance-trained men. *International Journal for Vitamin and Nutrition Research* 80, 54–64.

Elsabagh, S., Hartley, D.E., Ali, O., et al. (2005) Differential cognitive effects of *Ginkgo biloba* after acute and chronic treatment in healthy young volunteers. *Psychopharmacology (Berlin)* 179, 437–446.

Goldberg, D.M., Yan, J., & Soleas, G.J. (2003) Absorption of three wine-related polyphenols in three different matrices by healthy subjects. *Clinical Biochemistry* 36, 79–87.

Gonzalez-Gallego, J., Garcia-Mediavilla, M.V., Sanchez-Campos, S., et al. (2010) Fruit polyphenols, immunity and inflammation. *British Journal of Nutrition* 104(Suppl. 3), S15–S27.

Gregersen, N.T., Bitz, C., Krog-Mikkelsen, I., et al. (2009) Effect of moderate intakes of different tea catechins and caffeine on acute measures of energy metabolism under sedentary conditions. *British Journal of Nutrition* 102, 1187–1194.

Harwood, M., Danielewska-Nikiel, B., Borzelleca, J.F., et al. (2007) A critical review of the data related to the safety of quercetin and lack of evidence of in vivo toxicity, including lack of genotoxic/carcinogenic properties. *Food and Chemical Toxicology* 45, 2179–2205.

Hawley, J.A. & Holloszy, J.O. (2009) Exercise: it's the real thing! *Nutrition Reviews* 67, 172–178.

Howatson, G., McHugh, M.P., Hill, J.A., et al. (2010) Influence of tart cherry juice on indices of recovery following marathon running. *Scandinavian Journal of Medicine and Science in Sports* 20, 843–852.

Jin, F., Nieman, D.C., Shanely, R.A., et al. (2010) The variable plasma quercetin response to 12-week quercetin supplementation in humans. *European Journal of Clinical Nutrition* 64, 692–697.

Kennedy, D.O., Scholey, A.B., & Wesnes, K.A. (2001) Differential, dose dependent changes in cognitive performance following acute administration of a *Ginkgo biloba/Panax ginseng* combination to healthy young volunteers. *Nutritional Neuroscience* 4, 399–412.

Kressler, J., Millard-Stafford, M., & Warren, G.L. (2011) Quercetin and endurance exercise capacity: a systematic review and meta-analysis. *Medicine & Science in Sports & Exercise* 43, 2396–2404.

Lagouge, M., Argmann, C., Gerhart-Hines, Z., et al. (2006) Resveratrol improves mitochondrial function and protects against metabolic disease by activating SIRT1 and PGC-1alpha. *Cell* 127, 1109–1122.

Lau, F.C., Bielinski, D.F., & Joseph, J.A. (2007) Inhibitory effects of blueberry extract on the production of inflammatory mediators in lipopolysaccharide-activated BV2 microglia. *Journal of Neuroscience Research* 85, 1010–1017.

Macrae, H.S. & Mefferd, K.M. (2006) Dietary antioxidant supplementation combined with quercetin improves cycling time trial performance. *International Journal of Sport Nutrition and Exercise Metabolism* 16, 405–419.

Majumdar, A.P., Banerjee, S., Nautiyal, J., et al. (2009) Curcumin synergizes with resveratrol to inhibit colon cancer. *Nutrition and Cancer* 61, 544–553.

Malm, C., Sjodin, T.L., Sjoberg, B., et al. (2004) Leukocytes, cytokines, growth factors and hormones in human skeletal muscle and blood after uphill or downhill running. *Journal of Physiology* 556, 983–1000.

Manach, C., Hubert, J., Llorach, R., et al. (2009) The complex links between dietary phytochemicals and human health deciphered by metabolomics. *Molecular Nutrition and Food Research* 53, 1303–1315.

Mukherjee, D., Nissen, S.E., & Topol, E.J. (2001) Risk of cardiovascular events associated with selective COX-2 inhibitors. *Journal of the American Medical Association* 286, 954–959.

Murase, T., Haramizu, S., Ota, N., et al. (2008) Tea catechin ingestion combined with habitual exercise suppresses the aging-associated decline in physical performance in senescence-accelerated mice. *American Journal of Physiology. Regulatory, Integrative and Comparative Physiology* 295, R281–R289.

Murase, T., Haramizu, S., Ota, N., et al. (2009) Suppression of the aging-associated decline in physical performance by a combination of resveratrol intake and habitual exercise in senescence-accelerated mice. *Biogerontology* 10, 423–434.

Narkar, V.A., Downes, M., Yu, R.T., et al. (2008) AMPK and PPARdelta agonists are exercise mimetics. *Cell* 134, 405–415.

Nieman, D.C., Henson, D.A., Davis, J.M., et al. (2007) Quercetin ingestion does not alter cytokine changes in athletes competing in the Western States Endurance Run. *Journal of Interferon and Cytokine Research* 27, 1003–1011.

Nieman, D.C., Henson, D.A., Maxwell, K.R., et al. (2009) Effects of quercetin and EGCG on mitochondrial biogenesis and immunity. *Medicine & Science in Sports & Exercise* 41, 1467–1475.

Nieman, D.C., Williams, A.S., Shanely, R.A., et al. (2010) Quercetin's influence on exercise performance and muscle mitochondrial biogenesis. *Medicine & Science in Sports & Exercise* 42, 338–345.

Olson, C.A., Thornton, J.A., Adam, G.E., et al. (2010) Effects of 2 adenosine antagonists, quercetin and caffeine, on vigilance and mood. *Journal of Clinical Psychopharmacology* 30, 573–578.

Pearson, K.J., Baur, J.A., Lewis, K.N., et al. (2008) Resveratrol delays age-related deterioration and mimics transcriptional aspects of dietary restriction without extending life span. *Cell Metabolism* 8, 157–168.

Powers, S.K. & Jackson, M.J. (2008) Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. *Physiological Reviews* 88, 1243–1476.

Quindry, J.C., McAnulty, S.R., Hudson, M.B., et al. (2008) Oral quercetin supplementation and blood oxidative capacity in response to ultramarathon competition. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 601–616.

Reagan-Shaw, S., Nihal, M., & Ahmad, N. (2008) Dose translation from animal to human studies revisited. *FASEB Journal* 22, 659–661.

Reay, J.L., Kennedy, D.O., & Scholey, A.B. (2005) Single doses of *Panax ginseng* (G115) reduce blood glucose levels and improve cognitive performance during sustained mental activity. *Journal of Psychopharmacology* 19, 357–365.

Reay, J.L., Kennedy, D.O., & Scholey, A.B. (2006) Effects of *Panax ginseng*, consumed with and without glucose, on blood glucose levels and cognitive performance during sustained 'mentally demanding' tasks. *Journal of Psychopharmacology* 20, 771–781.

Richards, J.C., Lonac, M.C., Johnson, T.K., et al. (2010) Epigallocatechin-3-gallate increases maximal oxygen uptake in adult humans. *Medicine & Science in Sports & Exercise* 42, 739–744.

Smoliga, J.M., Baur, J.A., & Hausenblas, H.A. (2011) Resveratrol and health—a comprehensive review of human clinical trials. *Molecular Nutrition & Food Research* 55, 1129–1141.

Spencer, J.P. (2010) Beyond antioxidants: the cellular and molecular interactions of flavonoids and how these underpin their actions on the brain. *Proceedings of the Nutrition Society* 69, 244–260.

Sun, M., Qian, F., Shen, W., et al. (2011) Mitochondrial nutrients stimulate performance and mitochondrial biogenesis in exhaustively exercised rats. *Scandinavian Journal of Medicine and Science in Sports* 22, 764–765.

Trombold, J.R., Barnes, J.N., Critchley, L., et al. (2010) Ellagitannin consumption improves strength recovery 2–3 d after eccentric exercise. *Medicine & Science in Sports & Exercise* 42, 493–498.

Vang, O., Ahmad, N., Baile, C.A., et al. (2011) What is new for an old molecule? Systematic review and recommendations on the use of resveratrol. *PLoS One* 6, e19881.

Williams, R.J., Spencer, J.P., & Rice-Evans, C. (2004) Flavonoids: antioxidants or signalling molecules? *Free Radical Biology and Medicine* 36, 838–849.

Chapter 23

Risks and Rewards of Dietary Supplement Use by Athletes

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Many athletes, at all levels of competition, place great emphasis on the use of dietary supplements, but it is important to recognize that, of all the factors that determine athletic performance, supplements can play only a very small role. Compared with factors such as talent, training, tactics, and motivation, nutrition has a small effect on performance, and supplements can be no more than a small part of the athlete's nutrition strategy. Nevertheless, in most competitions, athletes are likely to be fairly evenly matched: one-sided competition gives little satisfaction to either participants or spectators. Competitors are therefore anxious to seek even the smallest advantage. At the highest levels of competition, all of the participants will be genetically gifted, all will have trained intensively, and all will be highly motivated. Where the margins between success and defeat are small, the small factors can become the ones that determine the outcome of sporting contests. This perhaps explains why athletes are constantly searching for any opportunity to gain an advantage over their competitors. Lack of awareness of basic nutrition principles also leaves athletes and nonathletes alike open to persuasion by the advertisements of those who profit from the sale of dietary supplements.

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Prevalence of Supplement Use

Surveys into the use of dietary supplements in the general population have consistently shown that supplements are used by a large part of the population (Hameen-Anttila et al., 2011; Timbo et al., 2006), and this is substantiated by the sales figures of what is now a multibillion dollar global industry. According to national surveys of the general population in the United States, more than 40% of the adult population used dietary supplements in the period from 1988 to 1994, and this had increased to over one-half in 2003–2006 (Centers for Disease Control and Prevention, 2011). Similar results are found in surveys of use by those who participate in sport (Lun et al., 2012). Hekkinen et al. (2011) also reported a high prevalence of supplement use in elite Finnish athletes (81% in 2002 and 73% in 2009). In contrast to this, however, Karimian and Esfahani (2011) examined supplement use in 250 male and 250 female bodybuilders in Iran: supplement use was reported by 87% of the men but by only 11% of the women.

An alternative way of assessing the prevalence of supplement use is by analysis of declaration forms completed by athletes selected for doping control. Among athletes competing at the 2004 Summer Olympic Games, almost half (45%) declared the use of a wide range of different food supplements (Tsitsimpikou et al., 2009), with vitamins (43%) and proteins/amino acids (14%) being the most widely used supplements. A large-scale analysis of elite track and field athletes (involving the review

of 3887 doping control forms undertaken during 12 International Association of Athletics Federations World Championships and 1 out-of-competition season) found 6523 declarations of the use of nutritional supplements, giving an average rate of use of 1.7 supplements per athlete (Tscholl et al., 2010). Similar high rates of supplement use were found in elite-level football players competing in the FIFA Football World Cup competitions of 2002 and 2006 (Tscholl et al., 2008).

The use of performance-enhancing substances seems to be endemic in the athletic population, and the use of these substances seems to begin at a young age. A recent large-scale survey of US youth suggested that use of all performance-enhancing substances ranged from 5% to 17% at different locations across the United States (Thorlton et al., 2012). Among elite young German athletes, the prevalence of dietary supplement use was estimated to be 80% (Braun et al., 2009), which is markedly higher than the data of the German National Nutrition Survey II, which showed that 16–19% of all German adolescents (aged 14–18 years) reported using dietary supplements (Federal Research Center for Nutrition and Food, 2008).

The prevalence of use seems to vary widely, and this may be explained in part by the lack of agreement as to what constitutes a dietary supplement. Some surveys have included sports drinks, energy bars, gels, and other sports foods, while others adopt a more restricted definition. In all surveys of supplement use among active or athletic populations, vitamins, minerals, and protein supplements are consistently among the most popular (Maughan et al., 2011).

Reasons for Supplement Use

Users cite many different reasons for consuming dietary supplements, though these reasons are more often based on unfounded beliefs than on any understanding of the issues at stake. In the general population, consumption of nutritional supplements is often driven by a belief that they confer health benefits above and beyond those that can be achieved by eating normal foods (Reinert et al., 2007). Among athletes and physically active individuals, several issues related specifically to the physical and mental aspects of exercise performance are added to the general health concerns of the population at large. Of course, athletes are also concerned to stay healthy as neither effective training nor successful competition is possible if health is compromised. Illness or injury that requires a break from training can disrupt preparations, and illness at the time of key competitions can ruin an athlete's competitive season. Supplements that promise to speed recovery, whether from illness, injury, or training, are popular with athletes.

An unpublished survey by Depiesse et al. of 310 competitors at the IAAF World Championships revealed that supplements were used by 86% of the athletes: this included 83% of males and 89% of females surveyed. Reasons for using supplements given by these athletes were as follows:

To aid recovery from training	71%
For health	52%
To improve performance	46%
To prevent or treat an illness	40%
To compensate for a poor diet	29%

Although dietary supplements may not be promoted for the prevention or treatment of illness, it is obvious that many consumers use them for this purpose. Sport-specific reasons for supplement use include a belief that the stress of intense training/competition cannot be met by food alone and that supplements can offer a specific advantage in either training or competition. There is also an awareness that successful competitors are using supplements, and the use of supplements is often endorsed or encouraged by influential individuals in the athlete's circle, including coaches and training partners. There is some evidence—largely anecdotal, but supported by evidence from surveys—that the amount of supplement used by athletes often exceeds the recommended amount. This "more is better" philosophy is encouraged by the belief that rivals are using even higher doses. Even elite athletes may not have access to professional advice that might counter some of these beliefs. Heikkinen et al. (2011) reported that only 27% of the 372 elite Finnish athletes they surveyed in 2009 had an opportunity

to consult a sports nutrition professional. Even when these opportunities are available, not all athletes choose to make use of them, preferring instead to get their nutrition advice from sources that they may see as being more congenial.

A recent review of the evidence suggested that the beliefs that supplements can confer health and performance benefits may be erroneous and concluded that "with the possible exceptions of Vitamin D and omega-3 fatty acids there is no data to support the widespread use of dietary supplements in Westernized populations; indeed, many of these supplements may be harmful" (Marik & Flemmer, 2012). Though the quality of the evidence on which these conclusions were based was often poor and the analysis of the evidence was perhaps not as rigorous as it might have been, this review does raise legitimate concerns regarding the widespread assumption that supplement use confers health benefits on the consumer and that it is free from any risk of adverse health outcomes.

Cost–Benefit Analysis of Supplement Use

There are several key questions that any athlete should ask before embarking on a program of supplement use. The first is an assessment of the evidence base relating to safety and the second is a similar analysis in relation to efficacy. Athletes should look for a good reason for using a supplement, and this means that there should be evidence of likely health or performance benefits from its use. The evidence relating to performance should be relevant to their own sport, preferably with data collected from athletes competing at a similar standard. It is often the case, however, that the evidence base is limited. For some of the products on sale, it is impossible to find any good evidence in the form of randomized, placebo-controlled intervention trials. Supplement sales often rely instead on endorsements by successful athletes, who may or may not have used the product and whose success is likely the result of various other factors. Where evidence does exist, measurements are seldom made on elite athletes and it is very likely that the factors that limit performance may be different in the elite athlete than in the untrained or recreationally active individuals who most often participate in laboratory studies. This may affect the response to supplementation. For example, several studies have shown that acute supplementation with nitrate can reduce the oxygen cost of exercise and enhance performance, but recent investigations have found no effect in well-trained subjects (Cermak et al., 2012; Wilkerson et al., 2012). Whether these discrepant findings result from differences in training status, exercise intensity and duration, supplementation protocol, or other factors remains as yet unclear, but this does highlight the need for care in the applications of laboratory findings to different sporting contexts.

The exercise models used in laboratory studies may or may not be relevant to the athlete's sport and may not even be real measurements of exercise performance. The "anaerobic threshold," for example, can be shown to correlate with endurance performance in a heterogeneous population, but it is not a good marker of performance in a more homogeneous population and it can change in the opposite direction from performance changes in response to nutritional interventions. In spite of this, however, changes in this measure in response to a supplement intervention are sometimes used as evidence of a performance effect (Graef et al., 2009). Similarly, the Physical Working Capacity at Fatigue Threshold (PWC(FT)) is an indirect measure, based on electromyographic measurements made during an incremental test that may or may not be relevant to exercise performance. Nevertheless, studies using the PWC(FT) have concluded that a period of supplementation with β-alanine can improve muscle endurance (Stout et al., 2008), even though no measure of performance was made.

No serious attempt has been made to evaluate the efficacy of many of the supplements used by athletes, nor indeed many of those that are popular with the general population. This is at least in part due to the difficulties involved in assessing efficacy. Many supplements are targeted at elite athletes, or at least at those training seriously and competing at a high level, but these individuals are seldom willing to participate in laboratory trials because of the inevitable disruption to training and competition

schedules. The relevance of tests carried out on recreational athletes to the elite performer is doubtful, as highlighted earlier in the case of nitrate supplements. It seems quite possible that the factors that limit performance differ in the truly elite athlete from those in the untrained or recreationally active individual.

There are also some real difficulties in assessing the efficacy of supplements. Even something as simple as exercise performance cannot be measured without some degree of uncertainty due to the inherent variability in performance. The validity, reliability, and sensitivity of the tests that are commonly employed as a measure of exercise performance have generated considerable controversy in recent years. Constant power tests to volitional exhaustion have been employed to examine the influence of various interventions on performance, but this method of testing has been criticized for a lack of ecological validity and poor test–retest reliability. This view is supported by the findings of Jeukendrup et al. (1996), who found a large day-to-day variability (coefficient of variation (CV) 27%) in time-to-exhaustion tests and a much smaller variability in a time trial protocol (<4%). Data from our research group report more consistent performance in a constant power to fatigue test (CV 6%; Maughan et al., 1989) when pre-exercise conditions are standardized and adequate familiarization with the test is given. Recent reports have highlighted similar errors of measurement when changes in performance are normalized across tests (Hinckson & Hopkins, 2005). A key factor to consider when selecting an appropriate exercise test is its sensitivity, and the smallest worthwhile effect that can be reliability detected. Amann et al. (2008) demonstrated that time-to-exhaustion and time trial protocols display a similar sensitivity to the effects of hypoxia and hyperoxia on performance and suggest that this finding will extend to other factors influencing performance. This is brought about by larger effects on performance in response to an intervention with constant power tests than are typically observed in time trial protocols: this compensates for the larger test–retest variability, resulting in a very similar signal-to-noise ratio to that seen with time trial protocols (Amman et al., 2008; Currell & Jeukendrup, 2008). In some research situations, the obvious limitation of time trial-type test is a difficulty in comparing the effect of an intervention on the physiological response to exercise, since at any given time one volunteer's relative workload may vary greatly from that of other participants.

It is essential that those who consider the use of supplements understand the limitations of the experimental evidence. The margin between first and second in an Olympic final is generally very small—as little as one thousandth of a second in a track cycling event lasting about 4 minutes—and even the difference between first and last may be much smaller than the sensitivity of laboratory tests of performance. Showing that a supplement does not improve the performance of a group of subjects in a laboratory test does not mean that it may not have worthwhile benefits for some athletes. There is also increasing evidence of "responders" and "non-responders" in populations of subjects who participate in any given study. To truly establish whether an individual falls into one or other of these categories, however, requires multiple tests to establish that the response is consistent. Where some athletes improve and some do not in a single test, the explanation may simply be random variation in response.

If something as simple as endurance performance cannot be measured reliably, the difficulties in assessing efficacy are magnified many times when there is a less well-defined endpoint. Even with a very large investment of time and money, it is unrealistic to expect clear evidence of efficacy in many of the areas that are important to athletes, such as wound healing, muscle soreness, immune function, and joint health. The Glucosamine/Chondroitin Arthritis Intervention Trial (GAIT) lasted 2 years and cost over US$12.5 million, but provided rather inconclusive results. Phase 1 of the study concluded that the dietary supplement combination of glucosamine plus chondroitin sulfate did not provide significant relief from osteoarthritis pain among all participants, although a small subgroup of study participants with moderate-to-severe pain showed significant relief with the combined supplements (Clegg et al., 2006). Phase 2 of the trial concluded that the supplements, either together or alone, appeared to fare no better than placebo in

slowing loss of cartilage in osteoarthritis of the knee (Sawitzke et al., 2008). Supplement companies are understandably reluctant to make such large financial investments in research where the outcome is uncertain.

It is also important to consider not only the potential benefits of supplement use but also the costs and risks (Maughan et al., 2011). Some supplements are expensive and do not represent a good financial investment for the average athlete. Others may pose a risk to health or performance, while some may cause the athlete to fail a drugs test. The issue of supplements and drugs tests will be discussed in more detail later, but it is clearly difficult for the athlete to make an informed decision on supplement use as many of the issues at stake are unknown.

Supplements That May Be Effective

Of the many hundreds, or even thousands, of supplements on sale, there is good evidence to support the use of only a very few, including creatine, caffeine, buffering agents, and perhaps also nitrate. There is some evidence for performance-related or health-related effects from a few others, including arginine, glutamine, β-alanine, *Echinacea*, glucosamine, antioxidants, carnitine, and zinc, but prospective, randomized clinical trials of these products in athletes are limited or almost completely absent. The athlete should also recognize that some of these supplements may have benefits in some exercise contexts, but not in others. It is important to know whether the supplement is likely to offer a benefit in the athlete's own sport and how and when it should be used, i.e., whether in training or acutely in competition. Other chapters in this volume will focus on specific supplements and on the potential benefits in particular situations, and these will not be discussed here.

Adverse Effects of Supplement Use

Consumers are entitled to expect that supplements purchased are fit for purpose: it seems reasonable to expect that a supplement purchased in a retail outlet or via the internet will contain the ingredients listed on the label in the stated amounts and that it does not contain anything else. Although the majority of supplement companies probably do offer reliable products, there is some evidence that this may not always be the case (Maughan, 2005). There are some well-publicized examples of lack of adherence to good manufacturing practice and also some clear examples of fraudulent practices. It is difficult, though, to assess the extent of these problems, as there is no comprehensive testing program for dietary supplements.

Two recent assessments of protein powders, which are generally considered to be low-risk products, illustrate some of the concerns. A 2010 review of 24 commercially available protein supplements revealed lead contamination in two protein supplements: at the levels of contamination found, these products would deliver a daily dose of 6–18 μg of lead (ConsumerLab®, 2010). The cumulative effects of lead intake are potentially harmful: even though the typical dose provided by these supplements may not be excessive when the recommended intake is consumed, it is recognized that many consumers far exceed the recommended amounts. In a similar analysis, ConsumerReports.org® (2012) reported results of analysis of 15 protein powders and drinks, which were purchased mainly in the New York metropolitan area or online, and were tested for the presence of arsenic, cadmium, lead, and mercury. Three products were found to contain levels of heavy metals in excess of the safe levels proposed by the US Pharmacopoeia.

These problems are consistent with poor quality control in manufacture and storage processes and there is no other obvious explanation for the presence of these low-level contaminants. A more significant concern is raised by various reports of supplement contamination from the US Food and Drug Administration (FDA). The FDA web site contains frequent reports of problems with supplements, with many of these reports relating to the presence in a wide range of supplement products of undeclared allergens, microbiological contamination, or foreign objects. Only rarely is action taken, and that action seldom amounts to more than a product recall. The extent of these recalls is apparent, however, from the FDA web site, suggesting

a rather widespread problem. Where relatively expensive ingredients are involved, some products are reported to contain little or no active ingredient but only inexpensive substitutes (Green et al., 2001). The substitute is sometimes difficult to identify, but some protein supplements have been shown to contain melamine, which had apparently been added as a low-cost substitute for the more expensive protein ingredients (Champagne & Emmel, 2011).

Sports Doping Issues

A series of claims by athletes that positive doping outcomes—in particular for the anabolic-androgenic steroid nandrolone—were blamed by athletes on their use of dietary supplements led to an investigation commissioned by the Medical Commission of the International Olympic Committee. In an initial investigation, ingestion of supplements containing nandrolone precursors that were not declared on the label was found to result in the presence of the diagnostic metabolites for nandrolone in the urine of volunteers (Geyer et al., 2000). Even though the alarm was raised, athletes, and those responsible for their care, were slow to act on this information and positive doping tests continued to result from the use of dietary supplements that contained prohibited substances. Geyer et al. (2004) purchased a total of 634 nonhormonal nutritional supplements from 215 different suppliers in 13 different countries in 2000–2001. On analysis of these supplements, 11 different anabolic-androgenic steroids, mainly prohormones of testosterone and nandrolone, were detected. Ninety-four samples (14.8%) contained prohormones. No reliable data were obtained for 66 samples (10.4%) because of matrix effects. Twenty-three samples contained prohormones of nandrolone and testosterone, 64 contained only prohormones of testosterone, and 7 contained only prohormones of nandrolone.

In a similar, more recent survey, an analysis of 58 supplements purchased through standard retail outlets in the United States found that 25% of the purchased products contained low levels of steroid contaminants and 11% were contaminated with stimulants (HFL, 2007). There are now many reports of the contamination of supplements with a wide range of agents that are prohibited by the antidoping regulations, including many that are potentially harmful to health (Ayotte et al., 2001; Catlin et al., 2000; Kamber et al., 2000). In some cases, the amounts are small and variable, and it seems likely that accidental cross-contamination during manufacture, processing, or packaging may be responsible. An analysis of the reasons for recalls of dietary supplements by the FDA reveals a clear pattern: anabolic-androgenic steroids are most commonly found in muscle building products, stimulants (such as ephedrine and amphetamine analogues) in tonic products, anorectic agents (such as sibutramine and fenfluramine) in weight loss products, and sildenafil and related compounds in sexual enhancement products. These undeclared pharmaceuticals can make what would otherwise be ineffective products into products that are effective in achieving their stated aims: this cannot simply be coincidence. Many of these potent pharmaceuticals, however, have significant side effects: they may not only confer a high degree of efficacy on these products but also have adverse health effects.

Small amounts of testosterone are unlikely to result in a positive doping result, but ingestion of even small amounts of some prohibited substances is likely to result in a positive test for nandrolone. In a study by Watson et al. (2009), 20 volunteers ingested 500 ml of water containing 5 g of creatine monohydrate (which was analyzed and shown to be free of steroids) to which 1.0, 2.5, or 5.0 μg of 19-norandrostenedione had been added. Subsequent urine samples were collected and analyzed for 19-norandrosterone (19-NA), the diagnostic metabolite for nandrolone, which also results from the metabolism of 19-norandrostenedione. The threshold urinary concentration for detection of a doping infringement is 2.0 ng/ml. Ingestion of the supplement resulted in mean peak urinary 19-NA concentrations of 0.7, 1.6, and 3.9 ng/ml in the 1.0, 2.5, and 5.0 μg trials, respectively. Under current World Anti-Doping Agency regulations, ingestion of the 1.0 μg dose produced no positive doping tests, 5 subjects (20%) tested positive in the 2.5 μg trial, and 15 subjects (75%) had urinary 19-NA concentrations exceeding 2 ng/ml after ingesting 5.0 μg of the steroid.

Adverse Health Outcomes from Supplement Use

Some supplements may actually cause harm to health, but adverse events can be difficult to identify and products are usually withdrawn from the market only after a significant number of adverse events have occurred. In 2009, a range of products containing hydroxycitric acid was withdrawn from sale in the United States: according to the FDA, this action was based on 23 reports of serious health problems ranging from jaundice and elevated liver enzymes, to liver damage requiring liver transplant and one death linked to liver damage (Food and Drugs Administration, 2009). In these cases, the adverse outcomes can be related to the known presence of harmful agents in the supplements. Where the presence of potentially toxic agents is not declared on the label, the likelihood of a link being established between supplement use and adverse outcomes is much more remote. However, in addition to numerous published case reports linking supplement use to adverse health outcomes, a recent report has described the development of hepatitis in a group of 20 Iranian male body-builders taking a cocktail of dietary supplements (Timcheh-Hariri et al., 2012).

Even when adverse health outcomes of anabolic steroid use are well recognized by those who consume them, use of these agents continues in more than half of users (Melia et al., 1996). It is likely that similar responses will be observed with supplements, so education alone is unlikely to prevent the use of harmful supplements where a benefit is perceived by users. Many users seem to see potential benefits without recognizing the real risks.

Quality Assurance Programs

Various quality assurance programs for sports nutrition supplements are available. Unlike the testing carried out by the FDA, which is primarily concerned with consumer protection issues such as the presence of the active ingredients in the stated amounts and the absence of substances that may be harmful to health, the focus of these programs is on the testing of samples provided by manufacturers or distributors for the presence of WADA-prohibited substances. These sports-related programs are not complete quality assurance programs in that the presence of active ingredients is not usually verified.

Although athletes and those who are responsible for their care often see these programs as a guarantee of the integrity of products that have been tested, it is important to recognize that a limited panel of substances is tested for and that the tests have limited sensitivity. In supplements tested through Informed-Sport program in the United Kingdom, for example, the level of detection is set at 10 ng/g for steroids and 100 ng/g for stimulants (Informed-Sport, 2012). Some other schemes operate at different levels, and it is important to recognize this. For supplements that are consumed in large amounts, such as protein powders or drinks, a much more sensitive test is required than for supplements taken as small pills or capsules. If a protein powder contained 90 ng/g of a steroid such as nandrolone or one of its precursors, this would appear as a negative test if the limit of detection is set at 100 ng/g. However, a 25 g portion of this product would deliver a dose of 2.3 μg of the steroid and there is a good chance that this might result in a positive test for nandrolone if a sample was collected within a few hours of ingestion of the supplement (Watson et al., 2009, 2010). Even when a product batch is tested prior to release for sale, there may therefore still be a risk, albeit a very low risk.

Consumers must recognize that although supplement quality assurance schemes do offer considerable protection, these schemes are not an absolute guarantee of quality.

Supplements or Food?

Unlike the situation with many dietary supplements, food can easily and cheaply provide protein and all of the essential amino acids that proteins contain. This calls into question the use of dietary protein supplements, especially given the observation that these are consistently among the best-selling supplement products. The use of food sources

of protein is discussed elsewhere and will not be discussed in detail here. However, it is appropriate to consider some of the issues as they affect the cost–benefit analysis relating to supplemental protein use.

It is generally agreed that an intake of about 20–25 g of high-quality protein soon after a resistance training session will maximize net protein synthesis (see Chapter 11). Athletes often use protein supplements, rather than normal foods, to achieve this intake, citing reasons of convenience and assurance as to the dose of protein delivered. This also applies to other nutrients: where athletes have little knowledge of nutrition, they are unsure whether foods will allow them to meet their nutritional goals. The financial costs of protein products are highly variable, as are the costs of protein-containing foods, and a financial comparison that has meaning on a global scale is impossible. Protein powders, though, are not expensive and a daily dose taken after training will cost only a few US dollars per week. Most foods will contain a range of other nutrients that will contribute to the individual's nutrition goals, and this consideration must also be included in any financial comparison.

It must also be recognized that some foods also suffer from poor quality control in manufacture and distribution and problems similar to those cited above are not uncommon in the food industry. It has also recently been recognized that the use of anabolic pharmacological agents as growth promoters in the farming industry may lead to residues in meat products that can cause an athlete to fail a drug test in the same way as can happen with contaminated supplements (Guddat et al., 2012).

Conclusions

The use of dietary supplements is no substitute for a good diet, and athletes are cautioned against indiscriminate use of supplements. The use of dietary supplements is widespread in the general population and among athletes and recreational exercisers. Where energy intake or food choice is restricted, a low-dose, broad-spectrum vitamin–mineral supplement may help. A few supplements may have role where a specific need is demonstrated, but a careful analysis of the potential benefits of supplement use must be balanced against the potential for harmful effects. A small number of supplements may also confer performance benefits in some specific situations. There are significant risks associated with the use of unregulated dietary supplements. Risks include the absence of active ingredients, the presence of harmful substances (including microbiological agents and foreign objects), the presence of toxic agents, and the presence of potentially dangerous prescription-only pharmaceuticals. There is ample evidence of athletes who have failed doping tests because of the use of dietary supplements. There is also growing evidence of risks to health and of serious adverse events, including a small number of fatalities, as a result of supplement use. The risk associated with the use of protein powders produced by major manufacturers is probably low, and the risk can be further reduced by using only products that have been tested under one of the recognized supplement quality assurance programs that operate in various countries. Nevertheless, a small risk remains, and athletes and other consumers should conduct a cost–benefit analysis before using any dietary supplements.

References

Amann, M., Hopkins, W.G., & Marcora, S.M. (2008) Similar sensitivity of time to exhaustion and time-trial time to changes in endurance. *Medicine & Science in Sports & Exercise* 40, 74–78.

Ayotte, C., Levesque, J.F., Cleroux, M., et al. (2001) Sport nutritional supplements: quality and doping controls. *Canadian Journal of Applied Physiology* 26, S120–S129.

Braun, H., Koehler, K., Geyer, H., et al. (2009) Dietary supplement use among elite young German athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 97–109.

Catlin, D.H., Leder, B.Z., Ahrens, B., et al. (2000) Trace contamination of over-the-counter androstenedione and positive urine test results for a nandrolone metabolite. *Journal of the American Medical Association* 284, 2618–2621.

Centers for Disease Control and Prevention (2011) *Dietary Supplement Use among U.S. Adults Has Increased Since NHANES III (1988–1994).* Available: http://www.cdc.gov/nchs/data/databriefs/db61.htm (accessed December 4, 2012).

Cermak, N.M., Res, P., Stinkens, R., et al. (2012) No improvement in endurance performance following a single dose of beetroot juice. *International Journal of Sport Nutrition and Exercise Metabolism* 22, 470–478.

Champagne, A.B. & Emmel, K.V. (2011) Rapid screening test for adulteration in raw materials of dietary supplements. *Vibrational Spectroscopy* 55, 216–223.

Clegg, D., Reda, D.J., Harris, C.L., et al. (2006) Glucosamine, chondroitin sulfate, and the two in combination for painful knee osteoarthritis. *New England Journal of Medicine* 354, 795–808.

ConsumerLab.com® (2010) *Product Review: Protein Powders & Drinks (Including Sports, Nutrition, and Diet Products)*. Available: https://www.consumerlab.com/reviews/Nutrition_Powders_Shakes_and_Drinks_Including_Protein_Diet_Meal-Replacement_and_Sports_Endurance_Recovery_Products/NutritionDrinks/ (accessed December 5, 2012).

ConsumerReports.org® (2012) *Protein Drinks*. Available: http://www.consumerreports.org/cro/2012/04/protein-drinks/index.htm (accessed December 5, 2012).

Currell, K. & Jeukendrup, A.E. (2008) Validity, reliability and sensitivity of measures of sporting performance. *Sports Medicine* 38, 297–316.

Federal Research Center for Nutrition and Food (2008) *Nationale Verzehrs Studie II. Ergebnisbericht, Teil 2*. Available: http://www.was-esse-ich.de/uploads/media/NVSII_Abschlussbericht_Teil_2.pdf (accessed December 6, 2012).

Food and Drugs Administration (2009) *Warning on Hydroxycut Products*. Available: http://www.fda.gov/ForConsumers/ConsumerUpdates/ucm152152.htm (accessed November 27, 2012).

Geyer, H., Mareck-Engelke, U., Reinhart, U., et al. (2000) Positive doping cases with norandrosterone after application of contaminated nutritional supplements. *Deutsche Zeitschrift fur Sportmedizin* 51, 378–382.

Geyer, H., Parr, M.K., Mareck, U., et al. (2004) Analysis of non-hormonal nutritional supplements for anabolic-androgenic steroids—results of an international study. *International Journal of Sports Medicine* 25, 124–129.

Graef, J.L., Smith, A.E., Kendall, K.L., et al. (2009) The effects of four weeks of creatine supplementation and high-intensity interval training on cardiorespiratory fitness: a randomized controlled trial. *Journal of the International Society of Sports Nutrition* 6, 18.

Green, G.A., Catlin, D.H., & Starcevic, B. (2001) Analysis of over-the-counter dietary supplements. *Clinical Journal of Sports Medicine* 11, 254–259.

Guddat, S., Fussholler, G., Geyer, H., et al. (2012) Clenbuterol—regional food contamination a possible source for inadvertent doping in sports. *Drug Testing and Analysis* 4, 534–538.

Hameen-Anttila, K.P., Niskala, U.R., Siponen, S.M., et al. (2011) The use of complementary and alternative medicine products in preceding two days among Finnish parents—a population survey. *BMC Complementary and Alternative Medicine* 11, 107. doi:10.1186/1472-6882-11-107

Heikkinen, A., Alaranta, A., Helenius, I., et al. (2011) Dietary supplementation habits and perceptions of supplement use among elite Finnish athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 271–279.

HFL (2007) *Supplements and Banned Substance Contamination: Offering an Informed Choice*. Available: http://www.informed-sport.com/sites/default/files/pdf/Supplements-Banned-Substance-Contamination.pdf (accessed December 6, 2012).

Hinckson, E.A. & Hopkins, W.G. (2005) Reliability of time to exhaustion analyzed with critical-power and log–log modeling. *Medicine & Science in Sports & Exercise* 37, 696–701.

Informed-Sport (2012) *What Detection Levels Should Supplements Be Tested At?* Available: http://www.informed-sport.com/faq# (accessed November 29, 2012).

Jeukendrup, A., Saris, W.H., Brouns, F., et al. (1996) A new validated endurance performance test. *Medicine & Science in Sports & Exercise* 28, 266–270.

Kamber, M., Baume, N., Saugy, M., et al. (2000) Nutritional supplements as a source for positive doping cases? *International Journal of Sport Nutrition and Exercise Metabolism* 11, 258–262

Karimian, J. & Esfahani, P.S. (2011) Supplement consumption in body builder athletes. *Journal of Research in Medical Sciences* 16, 1347–1353.

Lun, V., Erdman, K.A., Fung, T.S., et al. (2012) Dietary supplementation practices in Canadian high-performance athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 22, 31–37.

Marik, P.E. & Flemmer, M. (2012) Do dietary supplements have beneficial health effects in industrialized nations: what is the evidence? *Journal of Parenteral and Enteral Nutrition* 36, 159–168.

Maughan, R.J. (2005) Contamination of dietary supplements and positive drugs tests in sport. *Journal of Sports Sciences* 23, 883–889.

Maughan, R.J., Fenn, C.E., & Leiper, J. (1989) Effects of fluid, electrolyte and substrate ingestion on endurance capacity. *European Journal of Applied Physiology and Occupational Physiology* 58, 481–486.

Maughan, R.J., Greenhaff, P.L., & Hespel, P. (2011) Risks and rewards of dietary supplement use by athletes. *Journal of Sports Sciences* 29, S57–S66.

Melia, P., Pipe, A., & Greenberg, L. (1996) The use of anabolic-androgenic steroids by Canadian students. *Clinical Journal of Sport Medicine* 6, 9–14.

Reinert, A., Rohrmann, S., Becker, N., et al. (2007) Lifestyle and diet in people using dietary supplements: a German cohort study. *European Journal of Nutrition* 46, 165–173.

Sawitzke, A.D., Shi, H., Finco, M.F., et al. (2008) The effect of glucosamine and/or chondroitin sulfate on the progression of knee osteoarthritis: a report from the glucosamine/chondroitin arthritis intervention trial. *Arthritis & Rheumatism* 58, 3183–3191.

Stout, J.R., Graves, B.S., Smith, A., et al. (2008) The effect of beta-alanine supplementation on neuromuscular fatigue in elderly (55–92 years): a double-blind randomized study. *Journal of the International Society of Sports Nutrition* 5, 21.

Thorlton, J.R., McElmurry, B., Park, C., et al. (2012) Adolescent performance enhancing substance use: regional differences across the US. *Journal of Addictions Nursing* 23, 97–111.

Timbo, B.B., Ross, M.P., McCarthy, P.V., et al. (2006) Dietary supplements in a national survey: prevalence of use and reports of adverse events. *Journal of the American Dietetic Association* 106, 1966–1974.

Timcheh-Hariri, A., Balali-Mood, M., Aryan, E., et al. Toxic hepatitis in a group of 20 male body-builders taking dietary supplements. *Food and Chemical Toxicology* 50, 3826–3832. doi:10.1016/j.fct.2012.07.006

Tscholl, P., Junge, A., & Dvorak, J. (2008) The use of medication and nutritional supplements during FIFA World Cups 2002 and 2006. *British Journal of Sports Medicine* 42, 725–730.

Tscholl, P., Alonso, J.M., Dolle, G., et al. (2010) The use of drugs and nutritional supplements in top-level track and field athletes. *American Journal of Sports Medicine* 38, 133–140.

Tsitsimpikou, C., Tsiokanos, A., Tsarouhas, K., et al. (2009) Medication use by athletes at the Athens 2004 Summer Olympic Games. *Clinical Journal of Sport Medicine* 19, 33–38.

Watson, P., Judkins, C., Houghton, E., et al. (2009) Supplement contamination: detection of nandrolone metabolites in urine after administration of small doses of a nandrolone precursor. *Medicine & Science in Sports & Exercise* 41, 766–772.

Watson, P., Houghton, E., Grace, P.B., et al. (2010) The effect of delivery mode on excretion patterns of nandrolone metabolites after ingestion of a nandrolone pro-hormone. *Medicine & Science in Sports & Exercise* 42, 754–761.

Wilkerson, D.P., Hayward, G.M., Bailey, S.J., et al. (2012) Influence of acute dietary nitrate supplementation on 50 mile time trial performance in well-trained cyclists. *European Journal of Applied Physiology* 112(12), 4127–4134.

Chapter 24

Creatine

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Introduction

Creatine (methylguanidine acetic acid), named from the Greek word for "flesh," is a naturally occurring nutrient first identified in meat in 1835. In 1927, it was noted that the phosphorylated form of creatine, phosphorylcreatine (PCr), is hydrolyzed during muscular contraction and resynthesized during recovery, but it was not until the reintroduction of the percutaneous needle biopsy technique in the 1960s by Bergström and Hultman that the physiological role of PCr in maintaining ATP supply to the working skeletal muscle in humans was elucidated. Further work from Hultman's laboratory in the early 1990s (e.g., Greenhaff et al., 1993; Harris et al., 1992) demonstrated that feeding creatine at quantities several times greater than that normally consumed in the average non-vegetarian diet increased muscle creatine and PCr content in humans which had favorable effects on muscle energetics. Today, creatine monohydrate is one of the most popular dietary supplements among individuals taking part in recreational and competitive sport, with some outstanding achievements by elite athletes being perceived as related to creatine supplementation. This is reflected in annual global creatine sales in the late 1990s and early 2000s in excess of US$400 million. However, a report from the American College of Sports Medicine (Terjung et al., 2000) suggested that there appeared to be little appreciation of the pertinent research evaluating creatine supplementation and the nature of its potential impact on exercise performance, leading to unnecessary supplementation among many individuals. The purpose of this chapter is to firstly focus on the role of creatine and PCr in skeletal muscle energetics, and identify the conditions where PCr availability can influence muscle contractile function. This will set the scene for situations where there may be a benefit from creatine supplementation. Secondly, we will consider the efficacy of creatine supplementation at increasing the muscle total creatine content, and with the metabolic roles of creatine and PCr in mind, how such an increase can affect performance in sports. Side effects and safety issues of creatine supplementation will also be considered.

Is There a Requirement for Creatine Supplementation?

Creatine Homeostasis

Creatine homeostasis in humans is maintained by endogenous creatine synthesis and degradation, dietary creatine intake, and efficient conservation of creatine by the kidney, which collectively maintain a total body content of around 120 g for an average male (Walker, 1979). Creatine biosynthesis has been shown to proceed via two main reactions. The first reaction, catalyzed by glycine transamidinase, takes place in the kidneys, small intestine, pancreas, and liver, and involves an amidine group being

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reversibly transferred from arginine to glycine to form guanidinoacetic acid. Guanidinoacetate is subsequently transported to the liver for the second reaction, where it is methylated to creatine in process catalyzed by guanidinoacetate methyltransferase. The creatine formed rapidly diffuses into the circulation to be actively taken up by tissues that cannot synthesize creatine, such as skeletal muscle, or efficiently reabsorbed by the kidney. In order to maintain turnover, creatine is irreversibly degraded non-enzymatically within tissues to creatinine, which is then freely filtered by the kidney. The degradation of creatine to creatinine occurs at a steady rate of around 2 g per day which is lost in the urine in the average 70 kg human. To date, no studies have shown any function for creatinine in the human body, but since skeletal muscle contains most of the body's creatine and PCr, the urinary excretion of creatinine will vary as a function of muscle mass, being on the average less in females than males.

Omnivorous humans obtain around 1 g of creatine from the diet by the consumption of meat products (replacing around half of that lost to creatinine). Vegetarians have a negligible dietary intake of creatine, which is not matched by an increased endogenous synthesis as tissue creatine stores and daily urinary creatinine excretion are marginally lower than in omnivores. Most of the creatine in uncooked meat is found in the free form, with negligible amounts of PCr. The creatine concentration in uncooked chicken, beef, and lamb is ~3, 4.5, and 5 g/kg (wet weight) respectively, but the process of cooking degrades creatine to creatinine at a rate of around 10% per 20 minutes cooking. Almost all (>95%) of the ingested creatine is absorbed by the intestine. This remarkable ability of the intestine to absorb creatine appears to be due to the high specificity of an active creatine transporter that has been identified in the jejunum. Following absorption from the intestine, creatine is transported by the systemic circulation to tissues unable to synthesize creatine. In healthy individuals, fasting plasma creatine concentration is in the range of 20–50 μmol/l (2–5 mg/l) and increases five- to sixfold following the ingestion of a meal containing around 2 g of creatine.

Skeletal muscle has the greatest content of total creatine in the human body (~125 mmol/kg dry weight), which is twice as high as the total creatine content of cardiac muscle. Creatine is also found in small quantities in the brain, liver (2 mmol/kg dry weight), kidney, and testes. Thus, due to its large mass, more than 95% of all creatine within the body is stored within the skeletal muscle, of which 60–70% is in the form of PCr. Human skeletal muscle contains at least two major fiber types, which can be distinguished from differences in myofibrillar ATPase activity and oxidative capacity. Contractile speed is closely related to ATPase activity and, therefore, fiber types with low- or high-ATPase activity are designated slow (type I) and fast (type II) twitch, respectively (although type II fibers can be further sub-classified). These characteristics of muscle fibers result in different patterns of use (recruitment) depending on the exercise intensity. Type II fibers have up to a 50% greater total creatine content than type I fibers (e.g., 150 vs. 100 mmol/kg dry weight, respectively) and a correspondingly greater content of PCr, which coincides with the metabolic and physiological characteristics of the two fiber types.

Metabolic Roles of Creatine within Skeletal Muscle

ATP is the sole substrate that can be used directly by skeletal muscle to fuel contraction. However, the store of ATP in human skeletal muscle is relatively small (~24 mmol/kg dry muscle) and the energy required to maintain contraction (as well as ion pumps, etc.) during intense exercise would hypothetically deplete muscle ATP stores within 3 seconds. Thus, ATP must be continually resynthesized from its breakdown products ADP, AMP, and inorganic phosphate (P_i). Resynthesis of ATP during exercise is provided by anaerobic (nonoxygen using) and aerobic (oxygen using) processes, with the contribution from each depending principally on the relative exercise intensity and duration. During a number of physiological conditions, e.g., the transition from rest to exercise, the change from one power output to a higher power output, at power outputs above 90–100% maximal oxygen

consumption (VO_2 max), and in situations where the availability of oxygen is reduced (e.g., altitude exposure), the relatively slow activation and relatively low rate of energy delivery of oxidative ATP resynthesis cannot meet the ATP requirements of contraction. Under these conditions, the major contributors to the necessarily rapid resynthesis of ATP are ADP (in which 2 ADP combine to form ATP and AMP) and PCr hydrolysis, and glycolysis.

PCr is broken down by the enzyme creatine kinase (CK) to produce creatine and P_i, which is then transferred to ADP in order to resynthesize ATP ($PCr + ADP + H^+ \rightarrow ATP + Cr$). The PCr concentration in mixed fibered skeletal muscle is three- to fourfold greater than the ATP reservoir, amounting to around 80 mmol/kg dm at rest, although this value is higher in type II muscle fibers than in type I (90 vs. 70 mmol/kg dm). At the immediate onset of contraction there is a momentary rise in muscle ADP concentration, which triggers PCr hydrolysis via the CK reaction in order to rapidly rephosphorylate ADP and, thus resynthesize ATP. For each mole of PCr degraded, one mole of ATP is resynthesized, with the rate of PCr hydrolysis being greater in type II muscle fibers. The importance of this immediate onset of PCr hydrolysis lies in the extremely rapid rates at which it can resynthesize ATP, especially during maximal short duration exercise. However, the muscle PCr store is finite, and can only maintain ATP resynthesis during maximal exercise for a few seconds. For example, Figure 24.1 shows the results of an experiment in human volunteers by Hultman et al. (1991) that demonstrated the rate of ATP resynthesis from PCr hydrolysis during 30 seconds of fatiguing maximal isometric contraction in mixed fibered muscle was at its greatest within 1.3 seconds of the onset of contraction, at around 9 mmol/kg dm/s. However, after only 2.6 seconds of contraction, the rate of ATP resynthesis from PCr had declined by about 15% and in the final 10 seconds of a 30-second bout of muscle contraction, was relatively small, amounting to just 2% of the initial yield. If we then allow for a further level of complexity by considering PCr degradation in different muscle fiber types during this type of exercise, it can be seen that after the first 20 seconds of maximal muscle contraction, the rate

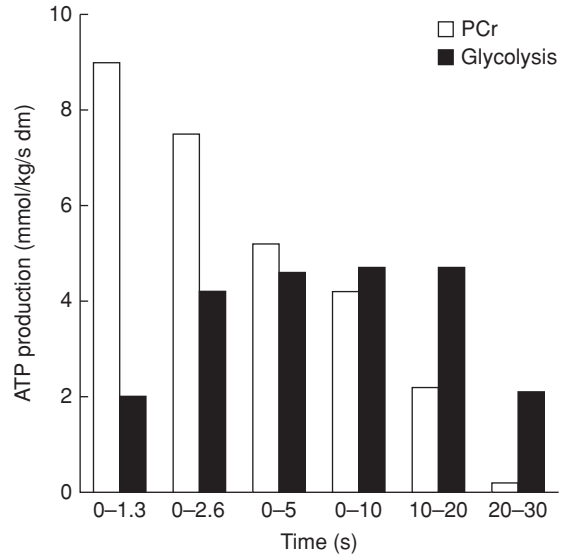

Figure 24.1 Rates of anaerobic ATP resynthesis from PCr and glycolysis during 30 seconds of near maximal intensity isometric contraction in humans. Adapted from Hultman et al. (1991).

of PCr utilization in type II fibers declines by about 60%, such that by the end of contraction, PCr stores are nearly depleted. The corresponding rate of PCr degradation in type I fibers, however, remains relatively unchanged, perhaps due to the lower rate of ATP demand of type I fibers. Thus, for maximal exercise to continue beyond only a few seconds, there is a marked increase in the contribution from glycogenolysis and glycolysis to ATP resynthesis.

It was thought for many years that PCr was the sole fuel utilized at the initiation of contraction, with glycogen utilization occurring at the onset of PCr depletion. However, this now known not to be the case and, as Figure 24.1 shows, ATP resynthesis from glycolysis during maximal contraction begins to occur almost immediately at the onset of exercise. Indeed, the activation of muscle contraction by calcium and the accumulation of the products of ATP and PCr hydrolysis (ADP, AMP, IMP, and P_i) act as stimulators of glycogenolysis. Furthermore, unlike PCr hydrolysis, ATP resynthesis from glycolysis does not reach its maximal rate until after 5 seconds of maximal exercise (suggesting that PCr may act as

a buffer of ATP resynthesis until glycolysis is fully activated) and is maintained at this high rate for several seconds, such that over 30 seconds of maximal exercise, the contribution from anaerobic glycolysis to ATP resynthesis is nearly double that from PCr hydrolysis. This is reflected by the very high muscle lactate concentrations (more than 100 mmol/kg dm) which are achieved during maximal exercise lasting 30 seconds or more. Thus, the relative contribution of PCr hydrolysis to ATP resynthesis during a bout of maximal exercise falls off dramatically as the exercise duration is increased beyond a few seconds.

The depletion of PCr and a fall in the rate of glycogenolysis during maximal intensity exercise, particularly in type II muscle fibers, is mirrored by a decline in muscle ATP production, suggesting that PCr availability may, at least in part, be limiting to performance of this type of exercise. Indeed, fatigue (defined as the inability to maintain a given or expected power or work output) is an inevitable feature of high intensity to maximal intensity exercise, and it has been demonstrated that force production and power output during intermittent, electrically evoked maximal isometric contractions and maximal treadmill sprinting in human volunteers can decline by as much as 40% after only 30 seconds (e.g., Figure 24.2). In support of this suggestion, studies involving repeated bouts of maximal exercise have shown that a significant relationship exists between the extent of PCr resynthesis in type II fibers during recovery between exercise bouts and subsequent exercise performance.

The PCr system also plays an important role in buffering the hydrogen ions (H^+) produced in glycolysis during high-intensity exercise, and thus delaying the development of intracellular acidosis by the absorption of a proton during the CK reaction (H^+ also promote activation of the key enzymes in glycogenolysis). The normal resting pH of the muscle cell is ~7.1, but actually increases at the onset of maximal exercise due to this function of the CK reaction. However, as exercise continues, muscle pH falls (to about 6.5), and studies using animals have concluded that this causes the contractile mechanism to fail, resulting in a reduction in force production. Furthermore, PCr availability appears to affect caffeine-induced sarcoplasmic reticulum (SR),

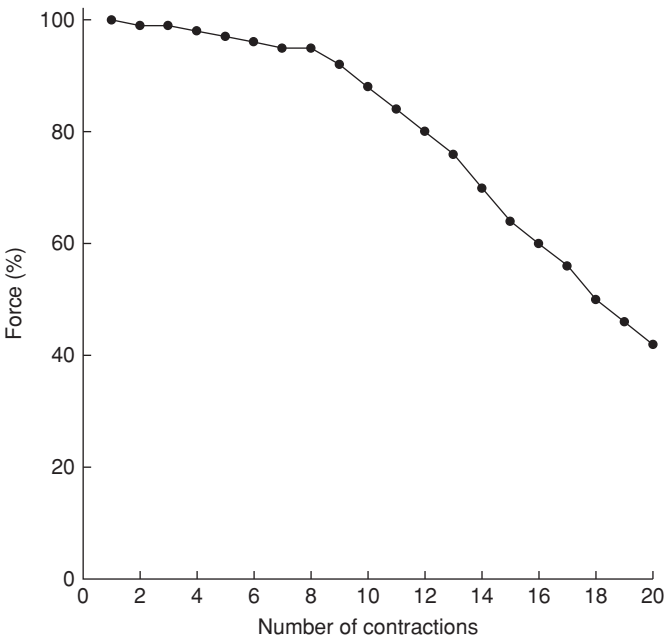

Figure 24.2 Reduction in force development during 20 intermittent (1.6 seconds contraction at 50 Hz, 1.6 seconds rest) maximal electrically evoked isometric quadriceps femoris contractions in human volunteers. Adapted from Söderlund et al. (1992).

calcium release and reuptake. A study by Duke and Steele (2001) in skinned rat skeletal muscle fibers demonstrated that acute PCr depletion results in a loss of calcium from the SR and a reduced rate of SR calcium reuptake. The existence of differing CK isoenzymes located on the inner mitochondrial membrane and bound to the M-line of myofibrils, has also led to the proposal that creatine connects sites of energy production (e.g., mitochondria) with sites of energy utilization (e.g., myofibrils). It is thought that ATP is functionally compartmentalized into mitochondrial and myofibrillar pools, and that PCr plays a role as the carrier of high-energy phosphate and the regulatory signal linking to two sites, i.e., a "PCr shuttle." In support of this theory, type I muscle fibers, which are specialized for aerobic energy production and are needed to link oxidative phosphorylation with sites of energy utilization, contain a relatively high percentage (30%) of mitochondrial CK compared to type II fibers. In this context, it is plausible that the predominant role played by the "creatine system" may differ between fiber types, e.g., with an energy reservoir function dominating in type II fibers, and an energy shuttle role predominating in type I fibers.

Taken together, it is clear that PCr depletion is limiting to ATP production and performance during maximal muscle contraction and exercise conditions where the aerobic energy supply is inadequate. Furthermore, if PCr has not been fully resynthesized during a short recovery period between bouts of maximal exercise, then it will have an even greater impact on the subsequent bout of high-intensity exercise. However, it is unlikely that creatine or PCr availability is limiting to submaximal exercise metabolism or performance. These findings have raised the question of whether an increase in the availability of muscle creatine and PCr via creatine supplementation can, therefore, improve maximal intensity exercise performance by delaying PCr depletion and, thus, the rate of ATP degradation during maximal exercise and/or increasing PCr resynthesis during recovery by maximizing mitochondrial CK flux. Thus, if creatine supplementation is to impart a potential benefit in energy provision during short-term very high-intensity exercise, especially when performed in repeated succession, it is vital that the creatine supplementation increases the muscle total creatine store.

Efficacy of Creatine Supplementation

Creatine Supplementation Increases Muscle Total Creatine Content

As mentioned above, intestinal absorption of creatine is close to 100%, and under normal conditions creatine is lost slowly from body tissues (in the main skeletal muscle) in the form of creatinine and excreted in the urine. Early studies demonstrated that creatine administration resulted in a small increase in urinary creatinine excretion, which rose slowly and, upon cessation of creatine feeding, began to fall after 5 weeks, suggesting that creatine must have been retained in the body tissues for some time. These early studies demonstrated that there was no increase in creatinine excretion until a significant amount of the administered creatine had been retained, which also suggested that creatine retention in the body pool was much greater during the initial stages of administration. There is now a substantial body of evidence to show that supplementation of the diet with creatine can increase skeletal muscle creatine content by up to 50%, which can be achieved within a few days of supplementation.

Muscle cells do not possess the ability to synthesize creatine, so they are dependent on the availability of creatine from the systemic circulation. There is around a 1000-fold concentration gradient between plasma (20–50 μmol/l) and muscle (~35 mmol/l intracellular water), and so creatine is actively transported from the extracellular space into the intracellular space by a saturable high-capacity, high-velocity, Na+-dependent process. In order to saturate the creatine transporter, a plasma creatine concentration of greater than 500 μmol/l is required and Harris et al. (1992) demonstrated that ingestion of a 5 g creatine bolus was capable of rapidly increasing plasma creatine concentration 20- to 30-fold (within 15 minutes) to 600–900 μmol/l. Thus, the 5 g creatine dose has become the standard single dose prescribed to humans during muscle

creatine loading. In terms of dietary consumption, 5 g of creatine could be found in 1.1 kg of fresh uncooked steak and represents a dose of around 70 mg/kg body weight for an average adult. This contrasts with the typical daily intake from the diet for nonvegetarians (about 15 mg/kg body weight/day), although some athletes who are focused on a high intake of animal protein may achieve high intakes from the diet. The plasma half-life of ingested creatine (1.5 hours) dictates that after consumption of a 5 g dose of creatine, the plasma concentration has returned to close to basal within approximately 5 hours, and so repeating this procedure at four evenly spaced occasions over the day is likely to maximize muscle total creatine uptake. Thus, Harris et al. (1992) demonstrated that supplementation of 4–6 doses of 5 g of creatine per day for 5–7 days increased muscle total creatine content by an average of 17% (range from 0 to 40%), from 126.8 ± 11.7 to 148.6 ± 5.0 mmol/kg dm. An increase occurred in all but two volunteers, suggesting that there were "non-responders" to supplementation (Figure 24.3). These two non-responders had a physiologically high initial muscle total creatine content (>145 mmol/kg dm), suggesting this may be the upper limit for muscle total creatine content. Furthermore, looking at responses across all volunteers it appeared that the magnitude of increase in muscle total creatine content with supplementation was at least partly an inverse function of the initial muscle total creatine content. These observations have since been replicated on many occasions, with the increase in muscle total creatine comprising increases in both free Cr and PCr (although the magnitude of increase in free Cr tends to be greatest). Consequently, 4 × 5 g of creatine per day for 5 days supplementation strategy has been adopted by the scientific and commercial literature and has been dubbed as "creatine loading."

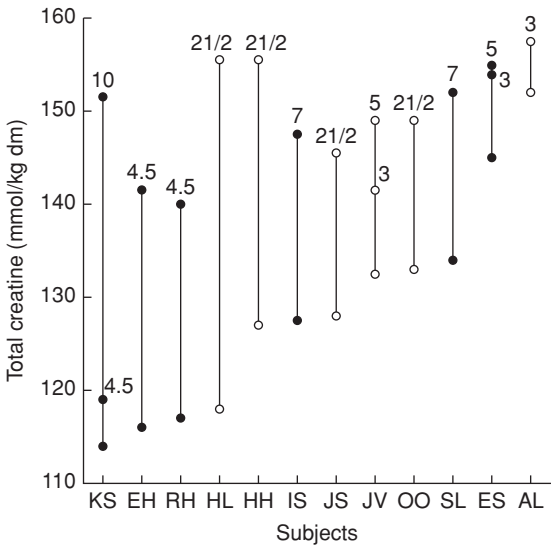

Figure 24.3 Muscle total creatine content before and after different durations (3–21 days indicated next to each point) of creatine ingestion at rates of 20 g/day (filled circles) and 30 g/day (open circles). 21/2 indicates creatine was ingested every other day for 21 days. Source: Reproduced with permission, from Harris et al. (1992). © The Biochemical Society.

In agreement with the early creatine supplementation studies based on urinary creatinine excretion, the majority of muscle creatine uptake occurs during the initial days of creatine loading, with close to 30% of the administered dose being retained during the initial 2 days of supplementation compared with 15% from days 2 to 4. Thus, the rate of muscle creatine uptake appears to be influenced by the intracellular creatine content, and extending the ingestion of 20 g of creatine per day beyond a few days is unwarranted and simply results in "expensive" creatine-enriched urine. However, if creatine supplementation is subsequently discontinued then, again in agreement with the early creatinine excretion studies, muscle total creatine content returns toward baseline over 4–6 weeks. For this reason, it has been suggested that a dose of creatine that will maintain muscle creatine delivery at slightly above the rate of muscle creatine degradation to creatinine (i.e., about 2 g/day) should be continued. Indeed, Hultman et al. (1996) demonstrated that following rapid "creatine loading" achieved by ingesting 20 g of creatine for 6 days, muscle stores can be maintained for at least a month by ingestion of 2 g per day of creatine thereafter. Furthermore, the same authors showed that ingesting creatine at a dose of 3 g per day for 4 weeks increases muscle total creatine content to the same values observed with 5–6 days of 20 g per day. The longer duration of supplementation required likely reflects that a single 3 g dose would not increase and

maintain plasma creatine concentration to a degree that would saturate muscle creatine transport for a prolonged period of the day.

As discussed earlier, muscle creatine uptake is Na^+ dependent, and via a muscle specific transport protein. A body of evidence now exists to show that stimulation of muscle Na^+/K^+ activity by exercise or insulin promotes muscle creatine accumulation above than seen with creatine supplementation alone, presumably by enhancing muscle creatine transport down an electrochemical gradient. In keeping with this, blocking muscle Na^+/K^+ activity using ouabain inhibits muscle creatine transport. In the seminal study by Harris et al. (1992), a group of six volunteers performed submaximal unilateral cycling before ingesting creatine during the loading phase. This strategy resulted on average in a 10% greater muscle total creatine content in the exercised limb than in the non-exercised limb, and this effect was observed in all six volunteers. Furthermore, it is now known that high circulating insulin levels (>100 mU/l) can stimulate muscle creatine uptake in humans under insulin clamp conditions (Steenge et al., 1998). Insulin infusion is clearly impractical, but combining 5 g of creatine with 95 g of simple carbohydrates has been shown to be an effective strategy of manipulating circulating insulin and, remarkably, increases the magnitude of both muscle PCr and Cr accumulation and reduces the variation in responses among individuals compared to creatine alone (Green et al., 1996a). For example, consumption of four doses of 5 g of creatine with 95 g of carbohydrate for 5 days augmented muscle total creatine accumulation (>20 mmol/kg dm in all volunteers) compared to a control group that underwent the same creatine supplementation strategy but without ingesting simple sugars (<20 mmol/kg dm in 50% of the volunteers; Green et al., 1996b). However, ingesting nearly 400 g of carbohydrates with 20 g of creatine per day during the loading phase was close to the limit of palatability of the volunteers and, in practical terms, would perhaps be difficult for athletes to achieve. Subsequent studies from the same group were unable to illustrate the same effect when each creatine dose was ingested with 50 g of simple sugars, but they were able to demonstrate that ingestion of a mixture of 5 g of creatine is combined with a mixture of 45 g dextrose and 50 g protein (Steenge et al., 2000) or 14 g protein, 7 g leucine, 7 g phenylalanine, and 57 g dextrose (Pittas et al., 2010), which increased serum insulin concentration similar to ingesting 5 g of creatine and 95 g of simple sugars, increased whole body creatine retention compared to creatine ingestion alone, and importantly, to a similar extent as ingesting 5 g of creatine with 95 g of simple sugars. Taken together, this would suggest that non-responders to creatine ingestion can become responders via exercise and nutritional strategies, although the effects of prior exercise on muscle creatine uptake appear to be less than the effect of insulin and are negated if individuals subsequently ingest creatine and carbohydrate.

Increasing Muscle Creatine Content Enhances Muscle Energy Metabolism and Increases Performance during Exercise

The first report of beneficial effects of creatine supplementation was by Sipilä et al. (1981), who described anecdotal evidence for a sensation of strength gain in a group of patients receiving 1 g of creatine per day for the treatment of gyrate atrophy over the course of a year. Indeed, one athlete in the group of patients improved his personal best time for the 100 m by 2 seconds. Greenhaff et al. (1993) subsequently conducted the first controlled investigation into the effect of oral Cr supplementation on exercise performance in humans in the early 1990s. Twelve subjects undertook 5 bouts of 30 maximal voluntary isokinetic contractions, interspersed with 1-minute recovery periods, before and after 5 days of placebo (4×6 g of glucose/day) or creatine (4×5 g of creatine plus 1 g of glucose/day) supplementation. Whereas no difference was seen when comparing peak torque production during exercise before and after placebo ingestion, peak torque production was greater by around 6% during the final ten contractions of exercise bout 1 ($p < 0.05$), throughout the whole of exercise bouts 2, 3, and 4, and during contractions 11–20 of the final exercise bout, when compared with the corresponding measurements made before creatine ingestion. Since then the effect of creatine supplementation on performance during many different exercise modalities and sporting

activities has been documented. Based on the published results from placebo-controlled laboratory experiments in young healthy subjects of mixed athletic ability and training status, it is now clear that the ingestion of 4 × 5 g of creatine per day for 5 days can significantly increase the amount of work that can be performed during short maximal sprinting, as well as repeated bouts of maximal knee extensor, sprinting, isokinetic, and cycling exercise performed with short recovery periods. The effects are also observed in resistance exercise performance, multiple-sprint endurance exercise events such as football and other team sports, and controlled field experiments undertaken by athletes over 4 × 300 m and 4 × 1000 m.

The exact mechanism by which short-term creatine supplementation improves maximal exercise performance is not yet clear, but it is likely to be due to a reduction in the extent of ATP degradation during exercise. For example, in a study by Greenhaff et al. (1994a), eight healthy male subjects performed two bouts of maximal isokinetic cycling exercise at 80 rpm before and after creatine or placebo ingestion (4 × 5 g/day for 5 days). Each exercise bout lasted for 30 seconds and bouts were interspersed by a 4-minute rest. Importantly, creatine ingestion resulted in a 25 mmol/kg dm increase in muscle total creatine content, of which 8.5 and 16.5 mmol/kg dm was in the form of PCr and creatine at rest, respectively, and an increase in work output in all eight subjects by over 4% during both bouts of exercise. When comparing total muscle ATP loss over the two bouts of exercise, the degradation was 25% less after creatine ingestion compared with before. Casey et al. (1996) also observed that creatine feeding (4 × 5 g/day for 5 days) resulted in a 30% reduction in ATP depletion compared to control during two bouts of 30 seconds maximal isokinetic cycling exercise. Interestingly, this effect was dependent on the magnitude of increase in muscle total creatine content, suggesting that the extent of creatine uptake during feeding is critical to subsequent exercise performance.

The greatest improvements in performance seem to be found during a series of repetitive high-power output exercise bouts (e.g., maximal cycling that can be maintained for only a short period, usually seconds) separated by fairly brief periods of rest (20–120 seconds), suggesting that an increased ability to resynthesize muscle PCr during recovery in the creatine loaded state is also a major mechanism by which creatine supplementation can affect exercise performance. Indeed, individuals who experience a marked (25%) increase in muscle total creatine content following creatine supplementation show an accelerated rate of PCr resynthesis during recovery from intense muscle contraction. In general, PCr resynthesis follows an exponential curve after intense muscle contraction and the half time for resynthesis in human skeletal muscle is approximately 30–40 seconds. Factors that will undoubtedly influence the rate of resynthesis include free ATP, ADP, H^+, and creatine concentrations, due to their role in the CK equilibrium reaction. The *in vitro* Michaelis constant (K_m) of CK for ATP and ADP are relatively low (~0.6 and 1 mmol/l, respectively). Conversely, the K_m of CK for creatine is relatively high, close to 18 mmol/l. Free creatine concentration ranges from around 13 mmol/l at rest to 40 mmol/l following maximal exercise. Thus, during the initial stages of recovery from maximal exercise, when the rate of mitochondrial ADP rephosphorylation to ATP will be at its highest, it is unlikely that the rate of PCr formation by CK will be dependent upon the availability of free creatine, because its concentration will be far in excess of the K_m value. However, as PCr resynthesis continues and muscle free creatine concentration declines toward 18 mmol/l, it is suggested that free creatine concentration may then limit PCr resynthesis. Indeed, following 2 minute of recovery from maximal exercise, muscle free creatine concentration is around 20 mmol/l (60 mmol/kg dm), whereas following creatine ingestion (4 × 5 g/day for 5 days) is around 25 mmol/l (75 mmol/kg dm) and PCr resynthesis is around 30% greater (Figure 24.4). Thus, the short-term rest periods between bouts are apparently sufficient to permit an enhanced recovery of the muscle PCr concentration in those individuals with a greater muscle total creatine concentration.

Taken together, it is clear that creatine supplementation improves exercise performance in events that require explosive, high-intensity activities especially of a repeated nature, which is

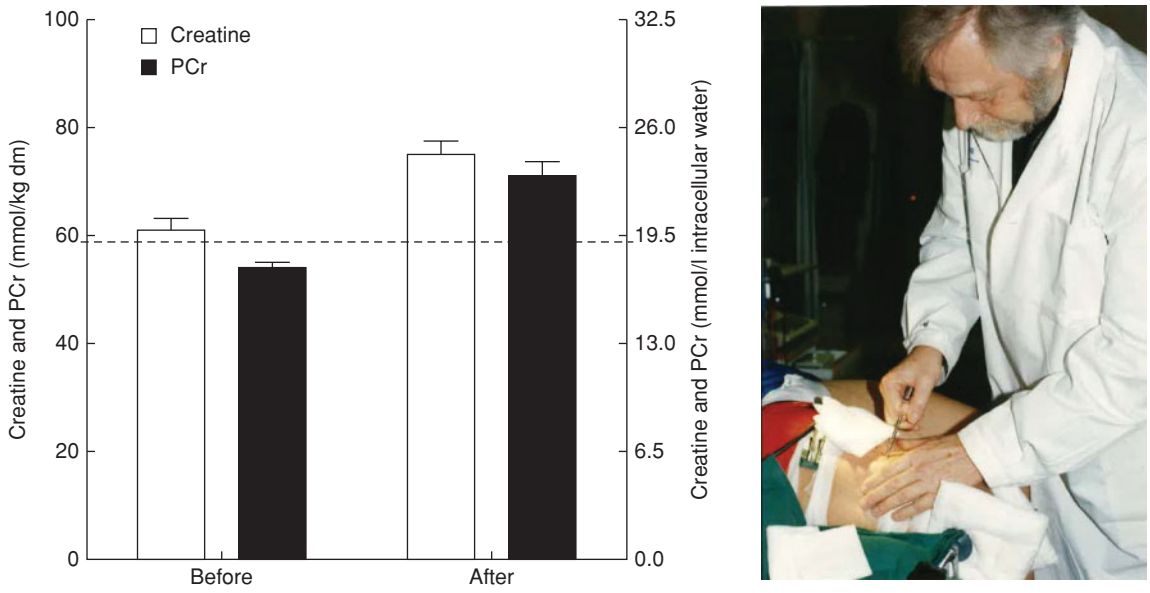

Figure 24.4 (a) Muscle PCr (filled) and creatine (open) content after 2 minutes of recovery from maximal contraction before and after 4 doses of 5 g of creatine per day for 5 days, which increased total creatine content by 25%. The dashed line represents the K_m of CK for creatine. Adapted from Greenhaff et al. (1994b). (b) Experimental set up of muscle PCr resynthesis study with Professor Eric Hultman.

an example where an enhancement in exercise performance matches the expectations identified from our knowledge of fundamental energetics of PCr in muscle. On the other hand, creatine supplementation does not appear to enhance endurance exercise type activities, which is in keeping with the near trivial contribution of PCr to the net energy expenditure of aerobic exercise. Indeed, van Loon et al. (2003) demonstrated that creatine loading for 6 days (4 × 5 g/day) in healthy young volunteers did not influence 20 minute time trial endurance performance on a cycle, nor did it increase in muscle citrate synthase activity or maximal aerobic workload capacity (W_{max}) compared to placebo. Despite a 30% increase in muscle creatine content compared to placebo, there was also no increase in the capacity to oxidize fat, and no reduction in blood lactate and ammonia concentrations during cycling exercise at 40%, 50%, and 60% of W_{max}, all of which would be expected with an increase in muscle and/or whole body aerobic capacity.

Does Increasing Muscle Creatine Content Have Any Other Effects?

Although increasing muscle creatine content does not increase maximal isometric muscle strength, or alter the rate of maximal force production, a number of studies have indicated that creatine ingestion can enhance the normal physiological adaptations of untrained individuals to resistance exercise training programs of greater than 4 weeks in duration. Typical training adaptations include increases in body fat-free mass, maximal strength and power, lifting volume, and muscle fiber hypertrophy, which are all significantly enhanced with concurrent creatine supplementation. Interestingly, an increase in maximal isometric strength has been observed after only 5 days of resistance training combined with creatine supplementation (2 × 5 g/day) in trained individuals (Maganaris & Maughan, 1998). Louis et al. (2003a, 2003b) investigated the effects of creatine loading (21 g/day for 5 days) on muscle protein

turnover in the fasted and fed state and following a single bout of resistance exercise (20 × 10 repetitions of leg extension–flexion at 75% one-repetition maximum in one leg). However, despite a 20% increase in muscle creatine content, no effect was observed on myofibrillar or sarcoplasmic protein fractional synthetic rate or muscle protein breakdown under fed or fasted conditions, or compared to the non-exercised control leg. Taken together, this would suggest that the increased muscle mass associated with training in the muscle creatine loaded state is not due to an effect of creatine *per se* on muscle protein synthesis, but more likely to the increased training load that can be achieved during high intensity, repetitive forms of resistance exercise training. Nevertheless, there is some evidence that creatine supplementation can facilitate recovery of muscle volume and functional capacity following muscle atrophy induced by leg immobilization (Hespel et al., 2001). Given the high incidence of musculoskeletal injury and consequent muscle disuse atrophy in athletes, creatine supplementation may be a worthwhile option to enhance post-injury rehabilitation and thereby speed return to training and competition.

Creatine supplementation has also been shown to have effects on muscle glycogen storage. Bergström and Hultman demonstrated a clear relationship between pre-exercise muscle glycogen content and exercise performance in the 1960s. The muscle glycogen store in healthy humans is ~350 mmol/kg dm, and is fairly resistant to change in non-exercised muscles. However, Bergström and Hultman (1967) demonstrated that the point of volitional exhaustion in ten healthy human subjects bicycling at an exercise intensity of 80% VO_2 max (approximately 70 minutes) coincided with near complete depletion of the mixed muscle glycogen store. It was also noted in an experiment by Bergstrom and Hultman (1966) that if subjects consumed a high-carbohydrate diet following glycogen depleting one-legged bicycling exercise, then muscle glycogen stores in the exercised muscles were increased to supranormal levels (~900 mmol/kg dry muscle), whereas it was relatively unchanged in the non-exercise leg. In another of Bergström and Hultman's pioneering experiments, it was clearly demonstrated that the greater the pre-exercise muscle glycogen content then the longer it took for the subjects to fatigue (Bergström et al., 1967). These findings clearly demonstrate that muscle glycogen availability is a limiting factor to prolonged exercise performance, and based on these experiments, the practice of "carbohydrate loading" in order to improve prolonged exercise performance is now common amongst athletes worldwide. Supplementation with creatine in combination with a high-carbohydrate diet can augment post-exercise muscle glycogen storage during a conventional "carbohydrate loading" regimen in humans, and is of a magnitude that could be expected to produce a significant improvement in endurance exercise performance (~150 mmol/kg dm; Robinson et al., 1999). The mechanisms that underpin this phenomenon remain to be elucidated. It does not appear to be due to an osmotic effect, increased muscle glucose transporter (GLUT4) content, or sensitivity to insulin, but may be related to a creatine-mediated reduction in free AMP concentration blunting AMP kinase activation and thereby augmenting glycogen synthase activity.

Another additional effect of creatine supplementation regularly reported in the scientific literature is an increase in body mass over the first few days of supplementation, ranging from 0.5 to 2 kg, which is caused by an increase in whole body water retention, as opposed to an increase in muscle protein mass. Indeed, there is a substantial reduction in urine production on the first 3 days of the loading period, which is thought to be related to an osmotic load caused by creatine retention. Although the effect may seem small, it could be problematic for those in weight category or weight-sensitive sports. According to a report from the French Agency for Food Safety (AFSSA, 2001), around 65% of published studies show significant increases of 0.8–2.9% in body weight over the first few days of creatine supplementation, with no subsequent alteration.

Safety of Creatine Supplementation

Creatine monohydrate is the most common form of commercially available creatine, and has been used in majority of the studies discussed above. There

are numerous anecdotal reports of creatine monohydrate supplementation causing gastrointestinal, cardiovascular and muscular problems, and the safety of creatine supplementation has frequently been questioned. However, the evidence is far from definitive and nearly 20 years of research involving feeding studies do not confirm any cause for concern. The European Food Safety Authority (EFSA) (2004) published an opinion on the use of creatine monohydrate as a food supplement and identified it as being safe within the dosage limits described here (more recently they have suggested that in order to "increase physical performance during short-term, high-intensity, repeated exercise bouts" healthy adults should consume 3 g of creatine per day; EFSA, 2011). One exception is that it is widely accepted that creatine supplementation should not be prescribed to individuals with established renal disease or at risk for renal dysfunction (e.g., diabetes, hypertension, impaired glomerular filtration rate), although this is only based on a single case study and it is also worth considering that the amount of creatine and creatinine handled by the kidney following creatine supplementation is small compared with the solute load that results from the typical protein intakes of athletes. Nevertheless, a lack of information cannot be taken as assurance that creatine supplementation is free from health risks. It should also be acknowledged that the safety and the regulatory status of the many different creatine salts and derivatives that are currently on sale to athletes (including creatine malate, creatine pyruvate, creatine citrate, creatine–magnesium chelate, creatine ethyl ester, and many more) remain unknown at the present time. Creatine salts are more soluble in water than creatine monohydrate and, therefore, easier to dissolve into a beverage. However, this does not appear to improve the bioavailability of ingested creatine and there is no evidence to support marketing claims that these newer forms are more effective in increasing muscle creatine content (Jäger et al., 2011). A final consideration to bear in mind is that, despite the potential "performance gains" from creatine supplementation described within this chapter, the changes in muscle total creatine and PCr caused by creatine supplementation do not mimic any adaptive changes which occur in response to exercise training programs. Conversely, neither aerobic, high-intensity resistance, nor sprint training is accompanied by significant changes in muscle PCr or total creatine content.

Acknowledgment

This chapter is dedicated to the memory of Professor Eric Hultman MD, PhD (1925–2011), clinician, innovator, mentor, scientist and, above all, a wonderful friend to those who knew him well.

References

AFSSA (2001) *Opinion of the French Agency for Food Safety and report on the assessment of the risks of creatine on the consumer and of the veracity of the claims relating to sports performance and the increase of muscle mass*. Available at: http://www.anses.fr/sites/default/files/documents/NUT-Ra-CreatineEN.pdf (accessed May 3, 2013).

Bergström, J. & Hultman, E. (1966) Muscle glycogen synthesis after exercise: an enhancing factor localized to the muscle cells in man. *Nature* 210, 309–310.

Bergstrom, J. & Hultman, E. (1967) A study of the glycogen metabolism during exercise in man. *Scandinavian Journal of Clinical and Laboratory Investigation* 19, 218–228.

Bergström, J., Hermansen, L., Hultman, E., et al. (1967) Diet, muscle glycogen and physical exercise. *Acta Physiologica Scandinavica* 71, 140–150.

Casey, A., Constantin-Teodosiu, D., Howell, S., et al. (1996) Creatine ingestion favorably affects performance and muscle metabolism during maximal exercise in humans. *American Journal of Physiology* 271, E31–E37.

Duke, A.M. & Steele, D.S. (2001) Interdependent effects of inorganic phosphate and creatine phosphate on sarcoplasmic reticulum Ca^{2+} regulation in mechanically skinned rat skeletal muscle. *Journal of Physiology* 531, 729–742.

EFSA (2004) Opinion of the Scientific Panel on Food Additives, Flavourings, Processing Aids and Materials in Contact with Food on a request from the commission related to creatine monohydrate for use in foods for particular nutritional uses. *EFSA Journal* 36, 1–6.

EFSA (2011) Scientific Opinion on the substantiation of health claims related to creatine and increase in physical performance during short-term, high intensity, repeated exercise bouts (ID 739, 1520, 1521, 1522, 1523, 1525, 1526, 1531, 1532, 1533, 1534, 1922, 1923, 1924), increase in endurance capacity (ID 1527, 1535), and increase in endurance performance (ID 1521, 1963) pursuant to Article 13(1) of Regulation (EC) No 1924/2006. *EFSA Journal* 9, 2303.

Green, A.L., Hultman, E., MacDonald, I.A., et al. (1996a) Carbohydrate ingestion augments skeletal muscle

creatine accumulation during creatine supplementation in humans. *American Journal of Physiology* 271, E821–E826.
Green, A.L., Simpson, E.J., Littlewood, J.J., et al. (1996b) Carbohydrate ingestion augments creatine retention during creatine feeding in humans. *Acta Physiologica Scandinavica* 158, 195–202.
Greenhaff, P.L., Casey, A., Short, A.H., et al. (1993) Influence of oral creatine supplementation on muscle torque during repeated bouts of maximal voluntary exercise in man. *Clinical Science* 84, 565–571.
Greenhaff, P.L., Bodin, K., Söderlund, K., et al. (1994a) Effect of oral creatine supplementation on skeletal muscle phosphocreatine resynthesis. *American Journal of Physiology* 266, E275–E300.
Greenhaff, P.L., Bodin, K., Casey, A., et al. (1994b) Dietary creatine supplementation and fatigue during high intensity exercise in humans. In *Biochemistry of Exercise IX* (eds. R.J. Maughan & S.M. Shirreffs), pp. 219–242. Human Kinetics, Champaign, IL.
Harris, R.C., Söderlund, K., & Hultman, E. (1992) Elevation of creatine in resting and exercised muscle of normal subjects by creatine supplementation. *Clinical Science* 83, 367–374.
Hespel, P., Op't Eijnde B., Van Leemputte, M., et al. (2001) Oral creatine supplementation facilitates the rehabilitation of disuse atrophy and alters the expression of muscle myogenic factors in humans. *Journal of Physiology* 536, 625–633.
Hultman, E., Greenhaff, P.L., Ren, J.M., et al. (1991) Energy metabolism and fatigue during intense contraction. *Biochemical Society Transactions* 19, 347–353.
Hultman, E., Söderlund, K., Timmons, J.A., et al. (1996) Muscle creatine loading in men. *Journal of Applied Physiology* 81, 232–237.
Jäger, R., Purpura, M., Shao, A., et al. (2011) Analysis of the efficacy, safety, and regulatory status of novel forms of creatine. *Amino Acids* 40, 1369–1383.
Louis, M., Poortmans, J.R., Francaux, M., et al. (2003a) No effect of creatine supplementation on human myofibrillar and sarcoplasmic protein synthesis after resistance exercise. *American Journal of Physiology* 285, E1089–E1094.
Louis, M., Poortmans, J.R., Francaux, M., et al. (2003b) Creatine supplementation has no effect on human muscle protein turnover at rest in the postabsorptive or fed states. *American Journal of Physiology* 284, E764–E770.
Maganaris, C.N. & Maughan, R.J. (1998) Creatine supplementation enhances maximum voluntary isometric force and endurance capacity in resistance trained men. *Acta Physiologica Scandinavica* 163, 279–287.
Pittas, G., Hazell, M.D., Simpson, E.J., et al. (2010) Optimization of insulin-mediated creatine retention during creatine feeding in humans. *Journal of Sports Sciences* 28, 67–74.
Robinson, T.M., Sewell, D.A., Hultman, E., et al. (1999) Role of submaximal exercise in promoting creatine and glycogen accumulation in human skeletal muscle. *Journal of Applied Physiology* 87, 598–604.
Söderlund, K., Greenhaff, P.L., & Hultman, E. (1992) Energy metabolism in type I and type II human muscle fibres during short term electrical stimulation at different frequencies. *Acta Physiologica Scandinavica* 144, 15–22.
Sipilä, I., Rapola, J., Simell, O., et al. (1981) Supplementary creatine as a treatment for gyrate atrophy of the choroid and retina. *New England Journal of Medicine* 304, 867–870.
Steenge, G.R., Lambourne, J., Casey, A., et al. (1998) Stimulatory effect of insulin on creatine accumulation in human skeletal muscle. *American Journal of Physiology* 275, E974–979.
Steenge, G.R., Simpson, E.J., & Greenhaff, P.L. (2000) Protein- and carbohydrate-induced augmentation of whole body creatine retention in humans. *Journal of Applied Physiology* 89, 1165–1171.
Terjung, R.L., Clarkson, P., Eichner, E.R., et al. (2000) American College of Sports Medicine roundtable. The physiological and health effects of oral creatine supplementation. *Medicine & Science in Sports & Exercise* 32, 706–717.
van Loon, L.J., Oosterlaar, A.M., Hartgens, F., et al. (2003) Effects of creatine loading and prolonged creatine supplementation on body composition, fuel selection, sprint and endurance performance in humans. *Clinical Science* 104, 153–162.
Walker, J.B. (1979) Creatine: biosynthesis, regulation, and function. *Advances in Enzymology and Related Areas of Molecular Biology* 50, 177–242.

Chapter 25

Caffeine and Exercise Performance

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Introduction

Caffeine occupies a unique position in the sports world as it is a drug that is an accepted component of the diet and also the training and competition arsenal of many athletes throughout the world. Caffeine is a trimethylxanthine drug that has no nutritional value, but it has been shown to have a potent "work-enhancing" or "ergogenic" effect in many sporting situations. The use of caffeine as an ergogenic aid has entered a new era for a number of reasons: (1) the removal of caffeine from the World Anti-Doping Agency (WADA) prohibited or controlled list of substances on the world stage in 2004; (2) strong evidence that low doses of caffeine, or doses that may be taken in the normal course of daily living, are ergogenic; (3) the realization that the ergogenic benefits of low doses of caffeine appear to be due to effects on the central nervous system (CNS); (4) extension of the strong laboratory-based evidence for the ergogenic effects of caffeine in running and cycling studies with recreational and sub-elite level subjects to near-elite and elite athletes in field-based individual and team sports in training, competition, and sport settings; and (5) the recognition that the effects of caffeine are variable and while some generalizations can be made, attention to individual responses and trialing is needed with all athletes.

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This chapter provides a brief overview of the research related to the usefulness of caffeine to enhance exercise performance in humans. The caffeine literature is large and has been reviewed on numerous occasions (Graham et al., 1994; Spriet, 1995, 2003; Spriet & Howlett, 2000). This chapter does not revisit these studies, but cites key and recent articles which provide the reader with access to many earlier reports. The chapter emphasizes the caffeine research from the past 10–15 years that deals with contemporary issues related to the metabolic and ergogenic effects of caffeine. Lastly, there is an attempt to identify areas where information is lacking.

Evolution of Caffeine Research

While research into the effects of caffeine on human performance can be traced back as far as 100 years, the field began in earnest with the work of Costill's group in the late 1970s (Costill et al., 1978). However, as late as the early 1990s, review articles were still concluding that the effects of caffeine ingestion on exercise performance and metabolism were inconsistent (e.g., Conlee, 1991; Nehlig & Debry, 1994). The authors summarized the literature and concluded that many experiments had not controlled for possible confounding factors: (1) the nutritional status and previous caffeine use of the subjects, (2) the exercise modality employed, (3) the power output used in the exercise testing and performance tests, (4) the caffeine dose used in the experimental design, (5) the untrained or recreationally trained status of the subjects, leading to poor repeatability

of exercise performance trials, and (6) the individual variation between subjects.

Research in the 1990s and the first years of the twenty-first century generally controlled for these factors and demonstrated a consistent ergogenic effect of caffeine during exercise lasting ~4 minutes and longer. Several review articles have summarized this work (Burke, 2008; Ganio et al., 2009; Keisler & Armsey, 2006; Spriet, 2003; Spriet & Howlett, 2000). In addition, several meta-analyses have also reported positive effects of caffeine on endurance exercise and on shorter term and graded exercise, and that caffeine decreased the ratings of perceived exertion during exercise (Doherty & Smith, 2004, 2005). Recently, new meta-analyses have reported that caffeine has a positive effect on muscular strength and endurance (Warren et al., 2010), is ergogenic in studies that employed only time trial performance tests lasting longer than 5 minutes (Ganio et al., 2009), and is ergogenic when ingested during exercise with carbohydrate (Conger et al., 2011). It is also clear that ingesting caffeine in preparation for training or a competition while consuming a high carbohydrate diet does not interfere with the ergogenic effects of caffeine. There has also been an increase in the number of studies examining the potential ergogenic effects of caffeine in team sports (e.g., Astorino & Robertson, 2010; Gant et al., 2010; Roberts et al., 2010) and for short-term high-intensity exercise that requires large amounts of the so-called "anaerobic" energy (Astorino & Robertson, 2010; Davis & Green, 2009).

Caffeine Is a Legal Supplement

The WADA removed caffeine from the restricted or controlled list in 2004 (Burke, 2008) though its use in competition is still monitored. Prior to this time, athletes were allowed to consume caffeine, but only to urine levels of 12 μg/ml following competitions. This level coincided with the consumption of ~10 mg caffeine/kg body mass (bm), which is a high dose (~700 mg of caffeine for a 70 kg individual). Presently, any level of caffeine consumption or urinary level is considered acceptable. This was seen as a positive step for the athletic world for many reasons, including that low amounts of caffeine that are readily available in foods and sports products are ergogenic, assessments of urinary caffeine levels have never been good predictors of consumption (Duthel et al., 1991), low levels of caffeine consumption are not harmful to health, and it would be impossible to control caffeine use during training.

Low Doses of Caffeine Are Ergogenic

Previous dose–response caffeine studies demonstrated that low doses of caffeine (~3 mg/kg bm) ingested 1 hour before exercise were just as effective at improving endurance performance as moderate and high doses (~6 to 13 mg/kg bm) (Graham & Spriet, 1995; Pasman et al., 1995). Many of the physiological responses reported with moderate and higher doses of caffeine (increased norepinephrine and epinephrine and increased free fatty acid (FFA) and glycerol levels) were not present with low caffeine doses (Graham & Spriet, 1995). This suggested that the traditional theory that caffeine mediated its ergogenic effect through a cascade of events including increased catecholamine release, increased fat release from storage sites in adipose tissue and muscle, increased FFA delivery/provision to the contracting muscles, increased muscle fat oxidation and sparing of muscle glycogen, and ultimately increased performance in exercise to exhaustion tasks (Costill et al., 1978) was not tenable when ingesting low doses of caffeine. These studies helped shift the focus for the explanation of the ergogenic effect of caffeine toward the CNS.

Caffeine is rapidly absorbed by the body and appears in the blood within 10–20 minutes after ingestion and peaks between 45 and 90 minutes (Figure 25.1). Plasma caffeine levels rise to ~60 to 70 μM with a caffeine dose of 9 mg/kg bm and ~40 and ~15 to 20 μM with lower doses of 6 and 3 mg/kg bm (Figure 25.1). Caffeine also has a long half-life (~3 to 5 hours) which makes it well suited to interact with many tissues in the body. However, since caffeine interacts with many tissues, it is difficult to independently study its effects on the CNS and the peripheral tissues (skeletal muscle, liver, and adipose tissue) in resting and exercising humans.

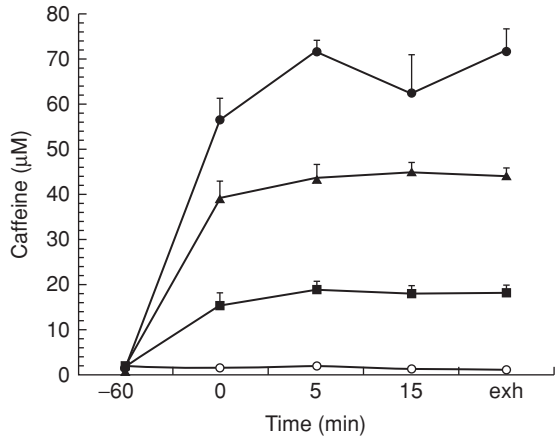

Figure 25.1 Plasma caffeine concentrations for 1 hour at rest and exercise to exhaustion at 80–85% VO_2 max in recreationally active subjects after the consumption of a placebo (open circles) or 3 (filled squares), 6 (filled triangles), or 9 (filled circles) mg/kg body mass of caffeine. Data are means ± SE, $n = 8$. Reprinted from Graham and Spriet (1995).

Pharmacological levels of methylxanthines affect several steps in skeletal muscle excitation–contraction coupling *in vitro*: (1) increasing the release of Ca^{2+} from the sarcoplasmic reticulum, (2) enhancing troponin/myosin Ca^{2+} sensitivity, and (3) decreasing the reuptake of Ca^{2+} by the sarcoplasmic reticulum (Tarnopolsky, 1994). Methylxanthines also stimulate Na^+–K^+ ATPase activity in inactive skeletal muscle leading to increased rates of K^+ uptake and Na^+ efflux. This attenuates the rise in plasma [K^+] with exercise, which may help maintain the membrane potential in contracting muscle and contribute to caffeine's ergogenic effect during exercise (Lindinger et al., 1993). Any of these changes could produce increases in skeletal muscle force production, but the caffeine levels needed to affect these changes are substantially higher than the levels required to affect adenosine receptors, and it seems unlikely that they could exert major ergogenic effects with caffeine doses of ~3 mg/kg bm or less where plasma levels are 15–20 μM.

Caffeine and the Central Nervous System

Caffeine freely passes through the blood–brain barrier and its concentration in the brain and CNS increases rapidly following ingestion in animals (Nehlig et al., 1992). Caffeine increases brain neurotransmitter levels, causing increases in spontaneous locomotor activity and neuronal firing (Nehlig et al., 1992). It is generally accepted that the mechanism for increases in neurotransmitter concentrations is adenosine receptor antagonism, and high adenosine receptor levels in the brain support this hypothesis (Daly, 1993; Fredholm, 1995). While adenosine and adenosine analogues cause lowered motor activity, decreased wakefulness and vigilance, and decreases in other neurotransmitter levels, caffeine and adenosine receptor antagonists block adenosine receptors and have the opposite effect. Caffeine increases the concentration, synthesis, and turnover of all major neurotransmitters, including serotonin, dopamine, acetylcholine, norepinephrine, and glutamate, and these neurotransmitters are all inhibited by adenosine (Fredholm, 1995). Complicating the effects of caffeine on adenosine antagonism is the existence of several isoforms of adenosine receptors, each having differing affinities for endogenous adenosine and xanthines and differentially affecting the release of several neurotransmitters (Daly, 1993; Graham et al., 1994).

Central Drive and Prolonged Exercise

The ability to generate maximal voluntary force declines with prolonged exercise and the central drive to maintain a given intensity of exercise increases (Kalmar & Cafarelli, 2004). Caffeine can improve central drive at many sites: increased spinal excitability and self-sustained firing, increased voluntary activation and maximal force, and decreased force and pain sensation during exercise (Kalmar & Cafarelli, 2004). For example, there is a significant body of literature that has examined the pain-reducing capacity of caffeine during various forms of exercise (Gliottoni et al., 2009). Most of these studies have reported reductions in leg pain at a given power output following moderate doses (5–6 mg/kg bm) of caffeine, given 1 hour before exercise. However, two reports used lower doses (2–3 mg/kg bm) of caffeine ingested before exercise and reported no pain-reducing effects in neutral and cool

environments (Astorino et al., 2011; Ganio et al., 2011). There do not appear to be any studies that have examined the effects of low caffeine doses given during exercise.

Delivery of Caffeine to the Brain Improves Exercise Performance

A recent study with rodents has provided strong evidence that the ergogenic effects of caffeine are manifested through the adenosine receptors in the CNS (Davis et al., 2003). Male rats randomly received one of four treatments (intracerebroventricular injections via an indwelling catheter) 30 minutes before they ran to fatigue on a treadmill: (1) caffeine, (2) an adenosine (A1/A2) agonist (5′-*N*-ethylcarboxamidoadenosine, NECA), (3) caffeine and NECA, and (4) a control trial of injection fluid (vehicle) only. Rats were trained to treadmill run before the experiment and received all four treatments, with 4–7 days between treatments. Mean run time in the control or vehicle trial was 76 ± 9 minutes while caffeine increased run time by ~60% to 119 ± 12 minutes and NECA decreased it by ~68% to 24 ± 7 minutes. When caffeine and NECA were given together, they essentially cancelled each other out as the run time was not different from the control trial (64 ± 15 minutes). Importantly, measurements of muscle glycogen use and blood FFA, glucose, and corticosterone levels after 30 minutes of the run to exhaustion were not different between the four trials. This suggested that the effects of the intraventricular injections did not affect the peripheral responses to exercise and were limited to central effects. In addition, when the same experiment was repeated, only this time with peripheral injections of the same four conditions (intraperitoneal), there was no difference in the run times to exhaustion. This work demonstrated that caffeine can work in the CNS to delay fatigue, at least in part by blocking adenosine receptors (Davis et al., 2003). Since caffeine can cross the blood–brain barrier when ingested orally, this effect may extend to the exercising human, but it is difficult to translate the caffeine effects of this work to the levels of caffeine that may be present in the brain following oral ingestion.

Low Caffeine Dose Studies in Humans

Kovacs et al. (1998) examined the effects of three low caffeine doses given with a carbohydrate (CHO) electrolyte solution on the ability to complete a set amount of work that took ~1 hour. Well-trained cyclists and triathletes randomly received placebo or a caffeine dose of 2.1, 3.2, or 4.5 mg/kg bm, with ~60% of the dose administered ~20 minutes before exercise, 20% after 20 minutes of cycling, and the remaining 20% of the dose at 40 minutes into the time trial. The time trial performance was 62.5 ± 1.3 minutes in the placebo trial and sequentially decreased with increasing caffeine doses to 61.5 ± 1.1, 60.4 ± 1.0, and 58.9 ± 1.2 minutes.

Well-trained athletes may be very sensitive to small doses of caffeine late in prolonged exercise without taking any caffeine before exercise (Cox et al., 2002). Scientists at the Australian Institute of Sport noticed that endurance cyclists preferred to switch from a sports drink during prolonged cycling to flat cola late in a 2- to 3-hour training session. They tested whether this practice was ergogenic and if so, whether the active ingredient in the cola was the extra carbohydrate (~11% CHO vs. ~6% in the sports drink) or the caffeine. Eight well-trained cyclists completed four double-blinded and random trials where they cycled for 2 hours at ~70% VO_2 max followed by a time trial where they completed 7 kJ/kg bm as fast as possible (~27 minutes). The subjects were mild caffeine users (>150 mg/day), but abstained from caffeine use for 48 hours before every trial. In all trials they consumed 5 ml of sports drink at 20, 40, and 60 minutes. At 80 and 100 minutes (and 120 minutes, if desired) they received one of four conditions: CONTROL (decaffeinated cola, 6% CHO), CAFFEINE (90 mg caffeine +, 6% CHO), Extra CHO (decaffeinated cola, 11% CHO), and COKE (90 mg caffeine +, 11% cola). Performance times for CONTROL and CAFFEINE were 27:05 ± 0:42 and 26:36 ± 0:42 (minutes:seconds) and for the Extra CHO and COKE trials were 26:55 ± 0:43 and 26:15 ± 0:43 (minutes:seconds). Coke enhanced the time trial performance by 3.3%, with all trials showing a 2.2% time trial enhancement with caffeine (Cox et al., 2002). The authors concluded that

~67% of the improvement in time trial performance was the effect of caffeine with the remaining 33% due to the additional carbohydrate. The average total caffeine intake at 80, 100 (and for some 120) minutes of cycling was only 133 mg or ~1.9 mg/kg bm, resulting in plasma levels of less than 10 μM. These low levels of caffeine intake and plasma accumulation did not affect any physiological responses to exercise, suggesting that the beneficial effects of caffeine were centrally manifested late in exhausting exercise.

A second study examined the effects of two low doses of caffeine on time trial performance following a prolonged cycle ride (Talanian & Spriet, 2007). Fifteen well-trained cyclists and triathletes, who were not caffeine users, completed four trials in a double-blinded and random fashion where they cycled for 2 hours at ~60% VO_2 max with five hill-climbs at ~85% VO_2 max followed by a time trial where they were asked to complete 6 kJ/kg bm as fast as possible (~25 to 30 minutes). In all trials, subjects consumed 5 ml of sports drink (6% CHO, 20 mM sodium) throughout the 2 hours. At 80 minutes, subjects received one of four conditions in their sports drink: Placebo (regular sports drink), CAF1 (100 mg caffeine, ~1.5 mg/kg bm), CAF2 (200 mg caffeine, ~3 mg/kg bm), or a random "repeat" of one of the other three conditions. The "repeat" trial helped complicate the subjects' perception of what they had received and post-trial questionnaires confirmed that the study was in fact double-blinded. Subjects completed the time trial in 28:41 ± 0:38 (minutes:seconds) in the Placebo condition and were significantly faster in the CAF1 (27:36 ± 0:32 minutes: seconds) trial and faster again in the CAF2 (26:36 ± 0:22 minutes:seconds) trial (Figure 25.2). Time trial performances in the "repeat" trials (five subjects completed two Placebo trials, five did two CAF1 trials, and five did two CAF2 trials) were 27:19 ± 0:30 for the first and 27:30 ± 0:35 minutes:seconds for the second trial. Plasma caffeine levels were not measurable in the Placebo condition and reached 14.9 and 13.8 μM before (120 minutes) and after the time trial in CAF1 and 24.9 and 25.6 μM in CAF2. These results demonstrated that two low caffeine doses corresponding to ~1.5 and ~3 mg/kg bm, ingested late in exhaustive exercise, were ergogenic in well-trained cyclists during a time trial (Talanian & Spriet, 2007). The 200 mg dose was more potent than 100 mg of caffeine. There were no differences between the conditions in the physiological responses to 120 minutes of submaximal exercise (heart rate, respiratory exchange ratio, and epinephrine, glucose, lactate, glycerol, and FFA levels) prior to the time trials, supporting a central mechanism for the improvement in performance. Other studies have recently reported significant improvements in cycling time trial performance following the ingestion of either 2 mg (Jenkins et al., 2008) or 3 mg caffeine/kg bm (Desbrow et al., 2012; Jenkins et al., 2008) taken before exercise, although others have not reported a significant effect with 1, 1.5, or 3 mg caffeine/kg bm, taken before exercise (Desbrow et al., 2009; Jenkins et al., 2008).

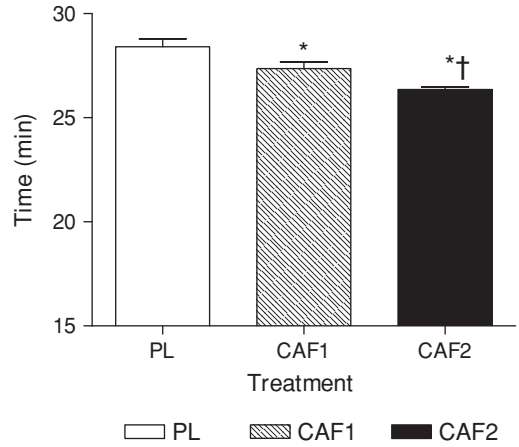

Figure 25.2 The effects of intracerebroventricular injections of an adenosine agonist (5′-*N*-ethylcarboxamidoadenosine, NECA), vehicle, Caffeine, or Caffeine and NECA on treadmill run time to fatigue in male rats. Data are means + SE, $n = 10$. *Run time significantly different from vehicle.

In all of the cited studies, the subjects were well-trained male athletes who were not caffeine users or mild caffeine users who abstained from caffeine in the 24–48 hours before trials. Exercise performance was consistently improved when caffeine was given during exercise at low to very low doses. In

the studies where low doses of caffeine were given 1 hour before exercise, the performance effects were not always present. It appears that the adenosine receptors in the CNS may be sensitized to caffeine's effects when fatigue is already present late in exercise, as very low doses of caffeine are ergogenic.

Metabolic Effects of Caffeine

There is some evidence that peripheral metabolic mechanisms are part of the explanation for the improvement in endurance exercise performance when the dose of caffeine is moderate to high (6–13 mg/kg bm; Spriet, 2003). The traditional theory is that caffeine mediates its ergogenic effect through a cascade of events including increased catecholamine release, increased FFA release from storage sites in adipose tissue and muscle, increased FFA delivery/provision to the contracting muscles, increased muscle fat oxidation and sparing of muscle glycogen, and ultimately increased performance (Costill et al., 1978). A greater role for fat metabolism in the first few minutes of exercise following caffeine doses of ≥5 mg/kg bm was supported by the findings of increased plasma (FFA) at the onset of exercise in numerous caffeine studies, glycogen sparing in the initial 15–30 minutes of exercise (Spriet et al., 1992), and increased intramuscular triacylglycerol use during the first 30 minutes of exercise (Essig et al., 1980). However, recent work has been less supportive of a metabolic explanation and has reported large between-subject variability and this theory cannot explain the ergogenic effects of low doses of caffeine (2–4 mg/kg bm).

Chesley et al. (1996) reported a variable effect of 9 mg caffeine/kg bm on muscle glycogen sparing during the initial minutes of exercise at 80–85% VO_2 max in recreationally trained males. Only 6 of 12 subjects used less glycogen (≥10% reduction) following caffeine ingestion. In subjects that did spare muscle glycogen, caffeine improved the energy status of muscle in the early stages of intense exercise, resulting in less phosphocreatine use and lower accumulations of free adenosine monophosphate (AMP) and diphosphate (ADP). Since AMP and ADP are allosteric activators of the active form of glycogen phosphorylase, lower accumulations would decrease the flux through phosphorylase and explain the reduction in muscle glycogen use. It is not presently clear how caffeine defends the energy state of the cell at the onset of intense exercise. However, it was hypothesized that it may be due to increased fat availability and a greater provision of the reduced form of nicotinamide adenine dinucleotide (NADH) in the mitochondria at the onset of exercise following caffeine ingestion 1 hour prior to exercise (Chesley et al., 1996). This would enable greater aerobic energy production from fat-derived NADH and reduce the need for glycogenolysis. In the subjects that did not reduce glycogen use, there were no reductions in phosphocreatine use and free AMP and ADP accumulations. However, it remains unclear why some subjects respond to high doses of caffeine by glycogen sparing and others do not as there were no other apparent differences between the groups.

Two more recent studies reported that caffeine had no effect on substrate metabolism during exercise when examined on a group basis. Graham et al. (2000) reported that 6 mg caffeine/kg bm did not alter fat or CHO metabolism during 1 hour of cycling at 70% VO_2 max in healthy male subjects. Direct measurements of leg glucose and FFA uptake, and lactate and glycerol release were made and were unaffected by caffeine. Caffeine ingestion did increase pre-exercise (FFA) and appeared to increase fat use early in exercise, but no measurements were made until 10 minutes into the exercise. However, if fat use was increased in the caffeine trial, it did not result in less glycogen use by the group of subjects in the initial 10 minutes of exercise (Graham et al., 2000). Laurent et al. (2000) also reported no effect of 6 mg caffeine/kg bm on muscle glycogen use during 2 hours of cycling at 65% VO_2 max in glycogen "supercompensated" and well-trained military personnel. These two studies reported only mean glycogen data and did not examine the effects of caffeine on individual muscle fiber glycogenolysis. However, it is not clear why the three studies discussed here reported either a variable glycogen sparing effect or no effect of caffeine on muscle glycogenolysis. It may be related to the exercise power output, the training and dietary status of the subjects, the duration of the exercise period examined, or simply biological variation

between subjects. However, it is clear that the recent data argue against an important metabolic contribution to the performance enhancement effects of high doses of caffeine during aerobic exercise.

Effects of Caffeine Ingestion on Intestinal Uptake and Oxidation of Glucose

Vergauwen et al. (1994) reported that adenosine receptors are involved in the stimulation of glucose uptake and transport by insulin and contractions in rat skeletal muscle and demonstrated that the administration of caffeine, an adenosine receptor antagonist, decreased glucose uptake during contractions. However, the plasma caffeine level in this study was 77 μM, which is at the high end of the physiological range and commensurate with a high caffeine dose (Figure 25.1). A more recent human study reported that the ingestion of 6 mg/kg bm caffeine 1 hour before exercise had no effect on directly measured leg glucose uptake during cycling at 70% VO_2 max, suggesting that other regulatory factors overwhelm any adenosine-related effects on glucose uptake (Graham et al., 2000). In this study the subjects were overnight fasted and no CHO was ingested during the exercise period. However, there is some evidence that caffeine ingestion can increase intestinal absorption and glucose oxidation when CHO is ingested during exercise. Van Nieuwenhoven et al. (2000) had well-trained subjects cycle for 90 minutes at ~70% VO_2 max after an overnight fast on three occasions. Just prior to exercise and at 20 and 40 minutes they ingested either water, a sports drink (~7% CHO), or the sports drink and ~100 mg caffeine: caffeine increased intestinal glucose absorption by 23% over the other two conditions. Jentjens et al. (2004) concluded from their own work that intestinal absorption was one of the main factors limiting exogenous glucose oxidation during prolonged exercise and subsequently tested whether the caffeine-induced increase in intestinal absorption would increase glucose oxidation. They exercised overnight-fasted and well-trained subjects for 2 hours at ~55% VO_2 max on three occasions: water ingestion only (~1.65 l), ingestion of the same volume of a CHO solution (~48 g/h), or the CHO solution with caffeine (10 mg/kg bm over 2 hours). Exogenous glucose oxidation over the final 30 minutes of exercise was 26% higher in the caffeine trial (Yeo et al., 2005), a result the authors attributed to increased intestinal absorption. While it remains possible that the increased glucose oxidation could be due to other factors like increased gastric emptying, decreased liver glucose disposal, or increased glucose uptake, there is little evidence to support these effects. Because this study used high levels of caffeine and did not measure performance, the study was repeated under conditions that simulated a field setting to a greater extent. Well-trained subjects cycled for 105 minutes at ~62% VO_2 max and then completed a time trial that required the completion of 7 kJ/kg bm as quickly as possible (~45 minutes) on three occasions: drinking a placebo solution, ingesting a CHO solution (43 g/h), or the CHO solution with 5.3 mg caffeine/kg bm (Hulston & Jeukendrup, 2008). With the lower caffeine dose there was no effect on exogenous CHO oxidation during the steady-state portion of the exercise, but performance with CHO was improved over placebo and the caffeine and CHO condition increased performance over the CHO-only condition. Similar results were reported when fed and trained cyclists cycled for 120 minutes at ~70% VO_2 max followed by a time trial requiring the completion of 7 kJ/kg bm, after ingesting a placebo, or 1.5 or 3 mg caffeine/kg bm 1 hour before exercise. A CHO solution (~1.2 l fluid/h and 80 g CHO/h) was provided in all trials at 15-minute intervals. The maximum rates of exogenous glucose oxidation were not altered by the caffeine and the time trial performance was also not affected. Plasma caffeine levels reached ~10 and 15–20 μM in the final 30 minutes of exercise in the two caffeine trials.

Collectively, these results argue that a high dose of caffeine is required to increase CHO oxidation during prolonged exercise. These findings are not present when ingesting moderate to low doses of caffeine and lend further support to a CNS explanation at the lower doses.

Caffeine Ingestion and Muscle Glycogen Resynthesis Following Exercise

Caffeine ingested before an oral glucose tolerance test has been shown to reduce insulin-driven glucose

disposal (Graham et al., 2001), suggesting that caffeine may impair glycogen resynthesis following exercise. However, there was no impairment of glycogen resynthesis following prolonged exercise when a total of 6 mg caffeine/kg bm was ingested before and during exercise and CHO was provided during 5 hours of recovery (Battram et al., 2004). This may be due to the fact that much of the glucose uptake and glycogen resynthesis in recovery that occurred was due to exercise-related factors like insulin-independent glucose uptake and/or the stimulatory effect of low postexercise muscle glycogen levels.

Surprisingly, Pedersen et al. (2008) reported that muscle glycogen resynthesis following exhaustive exercise and a low CHO overnight diet was enhanced when 4 mg caffeine/kg bm doses were consumed right after exercise and again after 2 hours of recovery while subjects consumed 1 g CHO/kg/h over a 4-hour recovery period. Muscle glycogen levels were ~75 mmol/kg dry muscle (dm) after exercise and overnight fasting in both trials and increased to 313 + 38 and 234 + 48 mmol/kg dm in the caffeine and CHO versus the CHO-only trials. The majority of the difference in glycogen resynthesis occurred in the final 3 hours of recovery, a finding that coincided with higher blood glucose levels in the caffeine and CHO trial. Plasma caffeine levels reached 31 and 77 μM after 1 and 4 hours of recovery. The rate of glycogen resynthesis reported in the caffeine trial appears to be the highest ever reported for human subjects in normal postexercise conditions (~60 mmol/kg bm/h). A recent study used the same experimental design as Pedersen et al. (2008) and demonstrated an improved performance during subsequent high-intensity interval running trials (48 vs. 32 minutes) in recreationally active men when caffeine was ingested along with CHO versus CHO alone in the 4-hour recovery period (Taylor et al., 2011). The authors speculated that the improved performance may have been due to increased glycogen resynthesis in the caffeine trial, but made no muscle measurements.

A recent paper by Beelen et al. (2012) reexamined this issue but was not able to support the findings of Pedersen et al. (2008). They exercised trained cyclists to deplete glycogen in the morning on two occasions, followed by a 6-hour recovery period where subjects ingested 1.2 g CHO/kg bm/h with or without 1.7 mg caffeine/kg/h. The CHO and/or caffeine were given every 30 minutes during recovery, and plasma caffeine levels reached 24 and 48 μM following 3 and 6 hours of recovery in the caffeine trial. The glycogen resynthesis rates were identical (31 ± 4 mmol/kg dm/h in each trial) in the two conditions (CHO: 172 + 40 to 360 + 31 mmol/kg dm vs. CHO and caffeine: 138 + 22 to 326 + 24 mmol/kg dm). Histochemical analyses of type I and II fiber glycogen contents also showed no change between trials post recovery. The plasma glucose concentrations and glucose appearance (intestinal glucose absorption) rates during recovery were also not different. It is not clear why the results of these two studies are divergent as both studies used trained subjects and high levels of CHO ingestion during recovery. The postexercise glycogen contents were not as low in the latter study, but the resynthesis rates were still quite high over a 6-hour recovery period. Both studies had subjects refrain from caffeine ingestion for 48 hours before trials, although subjects were essentially non-caffeine users in the Pedersen et al. (2008) study, whereas the subjects in the Beelen et al. (2012) study consumed about 3.5 units of caffeine or ~240 mg/day. However, these differences do not point to a mechanistic explanation for the divergent findings. It is also important to note that ingestion of such a large caffeine dose after exercise may interfere with other aspects of recovery like the ability to relax and sleep. There is a need to determine if lower doses of caffeine during recovery have any effect on glycogen resynthesis and subsequent exercise performance before this practice is likely to gain popularity. Another important consideration is the length of time an athlete has to recover, as it may be that any caffeine effect on glycogen resynthesis is compensated for when longer recovery periods are available (12–24 hours). Lastly, using high caffeine doses in recovery may also be contraindicated if relaxation and sleep patterns are disturbed, especially if the recovery time is short. These questions have not been tested to date.

Habitual Caffeine Consumption

An athlete's normal caffeine intake habits do not appear to affect whether acute caffeine ingestion

improves performance. Most studies have employed subjects who either did not consume any caffeine or were mild to moderate consumers (~50 to 300 mg/day) of caffeine. Most investigators have also asked users to refrain from caffeine use for 24–48 hours before experiments. Caffeine metabolism is not increased by use, but the effects of caffeine could be altered by habitual use via alterations in adenosine receptor populations. However, this possibility does not appear to dampen the ergogenic effects of caffeine, though other actions may be affected. For example, endurance performance increased in all subjects when both caffeine users and nonusers were examined and users abstained from caffeine for 48–72 hours prior to experiments (Graham & Spriet, 1991, 1995; Spriet et al., 1992). In addition, Van Soeren et al. (1993) reported no effect of up to 4 days of CAF withdrawal on caffeine-induced increases in performance and exercise hormonal and metabolic responses to exercise at 80–85% VO_2 max in recreational cyclists. These experiments were undertaken with moderate to high doses of caffeine, so it seems unlikely that user–nonuser and withdrawal issues would be of any concern at low caffeine doses.

Caffeine, Fluid Balance, Temperature Regulation, and Heat Tolerance

Consuming ~250 to 300 mg of caffeine has a mild diuretic effect at rest, at least in subjects who have not consumed caffeine for days or weeks, and it has been suggested that caffeine ingestion may lead to poor hydration status prior to and during exercise (as reviewed by Armstrong et al., 2007; Maughan & Griffin, 2003). If this were to occur, the ability to regulate exercise body temperature could be compromised and exercise tolerance, especially in the heat, may be reduced. However, a tolerance to the mild diuretic effect of caffeine at rest rapidly occurs and the diuretic effect is not present in subjects who regularly consume some caffeine (Maughan & Griffin, 2003). With the lower caffeine doses that are normally present in regularly consumed beverages such as tea, coffee, and cola drinks (<3 mg/kg bm or ~200 mg), there is no diuretic effect at rest and so there is no need to avoid these beverages prior to exercise. In spite of this, enough experimental evidence has appeared in this area to prompt the authors of a review article to conclude that there is no evidence to suggest that caffeine intake up to ~500 mg (~7 mg/kg bm) leads to dehydration at rest or, importantly, negatively affects exercise hydration status, performance, or the ability to thermoregulate in a cool or hot environment (Armstrong et al., 2007).

Summary

Moderate to high caffeine doses (5–13 mg/kg bm) ingested ~1 hour before activity increase exercise performance in laboratory and field-simulations of sporting situations. These effects extend to recreationally and well-trained subjects and are independent of habitual caffeine use, withdrawal of caffeine, high carbohydrate diets prior to exercise, and consumption of carbohydrate during exercise. Recent work suggests that caffeine is ergogenic in some short-term high-intensity exercise and sport situations and also in team sport simulations. Lower caffeine doses (up to ~3 mg/kg bm) taken before exercise also increase athletic performance and recent evidence also demonstrated a potent ergogenic effect of low to very low doses of caffeine taken during prolonged exercise. Lower caffeine doses do not cause changes in the peripheral whole body responses to exercise, are associated with few if any side effects, and have no effect on hydration status and the ability to thermoregulate during exercise. Therefore, the ergogenic effect of low caffeine doses appears to lie in the CNS. While all human caffeine performance studies have been unable to separate the central effects of caffeine from the peripheral effects, there is direct evidence in rodents that caffeine positively affects the CNS via adenosine receptor antagonism. Recent evidence suggesting that moderate to high doses of caffeine increase the intestinal absorption of carbohydrate during exercise and speed up the resynthesis of muscle glycogen in recovery from exercise is equivocal and unlikely to be of importance at lower caffeine doses (up to ~3 mg/kg bm or ~200 mg). As caffeine is now a permitted nutritional supplement, athletes should determine whether the ingestion of ~200 mg of caffeine before and/or during training and competitions is ergogenic on an individual basis.

References

Armstrong, L.E., Casa, D.J., Maresh, C.M., et al. (2007) Caffeine, fluid-electrolyte balance, temperature regulation, and exercise-heat tolerance. *Exercise and Sports Science Reviews* 35, 135–140.

Astorino, T.A. & Robertson, D.W. (2010) Efficacy of acute caffeine ingestion for short-term high intensity exercise performance: a systematic review. *Journal of Strength and Conditioning Research* 24, 257–265.

Astorino, T.A., Terzi, M.N., Roberson, D.W., et al. (2011) Effect of caffeine intake on pain perception during high intensity exercise. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 27–32.

Battram, D.S., Shearer, J., Robinson, D., et al. (2004) Caffeine ingestion does not impede the resynthesis of proglycogen and macroglycogen after prolonged exercise and carbohydrate supplementation in humans. *Journal of Applied Physiology* 96, 943–950.

Beelen, M., van Kranenburg, J., Senden, J.M., et al. (2012) Impact of caffeine and protein on postexercise muscle glycogen resynthesis. *Medicine & Science in Sports & Exercise* 44, 692–700.

Burke, L.M. (2008) Caffeine and sports performance. *Applied Physiology, Nutrition, and Metabolism* 33, 1319–1334.

Chesley, A., Howlett, R.A., Heigenhauser, G.J.F., et al. (1996) Regulation of muscle glycogen phosphorylase activity during intense aerobic exercise following caffeine ingestion. *American Journal of Physiology* 275, R596–R603.

Conger, S.A., Warren, G.L., Hardy, M.A., et al. (2011) Does caffeine added to carbohydrate provide additional ergogenic benefit for endurance? *International Journal of Sport Nutrition and Exercise Metabolism* 21, 71–84.

Conlee, R.K. (1991) Amphetamine, caffeine and cocaine. In *Ergogenics: Enhancement of Performance in Exercise and Sport* (eds. D.R. Lamb & M.H. Williams), pp. 285–330. Brown and Benchmark, Indianapolis, IN.

Costill, D.L., Dalsky, G.P., & Fink, W.J. (1978) Effects of caffeine on metabolism and exercise performance. *Medicine & Science in Sports* 10, 155–158.

Cox, G.R., Desbrow, B., Montgomery, P.G., et al. (2002) Effect of different protocols of caffeine intake on metabolism and performance. *Journal of Applied Physiology* 93, 990–999.

Daly, J.W. (1993) Mechanism of action of caffeine. In *Caffeine, Coffee, and Health* (ed. S. Garatttini), pp 97-150. Raven Press, New York.

Davis, J.K. & Green, J.M. (2009) Caffeine and anaerobic performance: ergogenic value and mechanisms of action. *Sports Medicine* 39, 813–832.

Davis, J.M., Zhao, Z., Stock, H.S., et al. (2003) Central nervous system effects of caffeine and adenosine on fatigue. *American Journal of Physiology* 284, R399–R404.

Desbrow, B., Barrett, C.M., Minahan, C.L., et al. (2009) Caffeine, cycling performance, and exogenous CHO oxidation: a dose-response study. *Medicine & Science in Sports & Exercise* 41, 1744–1751.

Desbrow, B., Biddulph, C., Devlin, B., et al. (2012) The effects of different doses of caffeine on endurance cycling time trial performance. *Journal of Sports Sciences* 30, 115–120.

Doherty, M. & Smith, P.M. (2004) Effects of caffeine on exercise testing: a meta-analysis. *International Journal of Sports Nutrition and Exercise Metabolism* 14, 626–646.

Doherty, M. & Smith, P.M. (2005) Effects of caffeine on rating of perceived exertion: a meta-analysis. *Scandinavian Journal of Medicine, Science and Sports* 15, 69–78.

Duthel, J.M., Vallon, J.J., Martin, G., et al. (1991) Caffeine and sport: role of physical exercise upon elimination. *Medicine & Science in Sports & Exercise* 23, 980–985.

Essig, D., Costill, D.L., & VanHandel, P.J. (1980) Effects of caffeine ingestion on utilization of muscle glycogen and lipid during leg ergometer cycling. *International Journal of Sports Medicine* 1, 86–90.

Fredholm, B.B. (1995) Adenosine, adenosine receptors and the actions of caffeine. *Pharmacology and Toxicology* 76, 93–101.

Ganio, M.S., Klau, J.F., Casa, D.J., et al. (2009) Effect of caffeine on sport-specific endurance performance: a systematic review. *Journal of Strength and Conditioning Research* 23, 315–324.

Ganio, M.S., Johnson, E.C., Lopez, R.M., et al. (2011) Caffeine lowers muscle pain during exercise in hot but not cool environments. *Physiological Behaviour* 102, 429–435.

Gant, N., Ali, A., & Foskett, A. (2010) The influence of caffeine and carbohydrate coingestion on simulated soccer performance. *International Journal of Sports Nutrition and Exercise Metabolism* 20, 191–197.

Gliottoni, R.C., Meyers, J.R., Arngrimsson, S.A., et al. (2009) Effect of caffeine on quadriceps muscle pain during acute cycling exercise in low versus high caffeine consumers. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 150–161.

Graham, T.E. & Spriet, L.L. (1991) Performance and metabolic responses to a high caffeine dose during prolonged exercise. *Journal of Applied Physiology* 71, 2292–2298.

Graham, T.E. & Spriet, L.L. (1995) Metabolic, catecholamine and exercise performance responses to varying doses of caffeine. *Journal of Applied Physiology* 78, 867–874.

Graham, T.E., Rush, J.W.E., & van Soeren, M.H. (1994). Caffeine and exercise: metabolism and performance. *Canadian Journal of Applied Physiology* 19, 111–138.

Graham, T.E., Helge, J.W., MacLean, D.A., et al. (2000) Caffeine ingestion does not alter carbohydrate or fat metabolism in skeletal muscle during exercise. *Journal of Physiology* 529(3), 837–847.

Graham, T., Sathasivam P., Rowland, M., et al. (2001) Caffeine ingestion elevates plasma insulin response in humans during an oral glucose tolerance test. *Canadian Journal of Physiology and Pharmacology* 79, 559–565.

Hulston, C.J. & Jeukendrup, A.E. (2008) Substrate metabolism and exercise performance with caffeine and carbohydrate intake. *Medicine & Science in Sports & Exercise* 40, 2096–2104.

Jenkins, N.T., Trilk, J.L., Singhal, A., et al. (2008) Ergogenic effects of low doses of caffeine on cycling performance. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 328–342.

Jentjens, R.L., Venables, M.C., & Jeukendrup, A. (2004) Oxidation of exogenous glucose, sucrose and maltose during prolonged cycling exercise. *Journal of Applied Physiology* 96, 1285–1291.

Kalmar, J.M. & Cafarelli, E. (2004) Caffeine: a valuable tool to study central fatigue in humans. *Exercise and Sports Science Reviews* 32, 143–147.

Keisler, B.D. & Armsey, T.D. (2006) Caffeine as an ergogenic aid. *Current Sports Medicine Reports* 5, 215–219.

Kovacs, E.M.R., Stegen, J.H.C.H., & Brouns, F. (1998) Effect of caffeine drinks on

substrate metabolism, caffeine excretion, and performance. *Journal of Applied Physiology* 85, 709–715.

Laurent, D., Schneider, K.E., Prusaczyk, W.P., et al. (2000) Effects of caffeine on muscle glycogen utilization and the neuroendocrine axis during exercise. *Journal of Clinical Endocrinology & Metabolism* 85, 2170–2175.

Lindinger, M.I., Graham, T.E., & Spriet, L.L. (1993) Caffeine attenuates the exercise-induced increase in plasma [K^+] in humans. *Journal of Applied Physiology* 74, 1149–1155.

Maughan, R.J. & Griffin, J. (2003) Caffeine ingestion and fluid balance: a review. *Journal of Human Nutrition and Dietetics* 16, 1–10.

Nehlig, A. & Debry, G. (1994) Caffeine and sports activity: a review. *International Journal of Sports Medicine* 15, 215–223.

Nehlig, A., Daval, J.-L., & Debry, G. (1992) Caffeine and the central nervous system: mechanisms of action, biochemical, metabolic, and psychostimulant effects. *Brain Research Reviews* 17, 139–170.

Pasman, W.J., van Baak, M.A., Jeukendrup, A.E., et al. (1995) The effect of different dosages of caffeine on endurance performance time. *International Journal of Sports Medicine* 16, 225–230.

Pedersen, D.J., Lessard, S.J., Coffey, V.G., et al. (2008) High rates of muscle glycogen resynthesis after exhaustive exercise when carbohydrate is coingested with caffeine. *Journal of Applied Physiology* 105, 7–13.

Roberts, S.P., Stokes, K.A., Trewartha, G., et al. (2010) Effects of carbohydrate and caffeine ingestion on performance during a rugby union simulation protocol. *Journal of Sports Sciences* 28, 833–842.

Spriet, L.L. (1995) Caffeine and performance. *International Journal of Sports Nutrition* 5, S84–S99.

Spriet, L.L. (2003) Caffeine. In *Performance-Enhancing Substances in Sport and Exercise* (eds. M.S. Bahrke & C.E. Yesalis), pp. 267–278. Human Kinetics, Windsor, ON.

Spriet, L.L. & Howlett, R.A. (2000) Caffeine. In *Nutrition in Sport—Encyclopaedia of Sports Medicine* (ed. R.J. Maughan), vol. VII, Chapter 28, pp. 379–392. Blackwell Science, Oxford.

Spriet, L.L., MacLean, D.A., Dyck, D.J., et al. (1992) Caffeine ingestion and muscle metabolism during prolonged exercise in humans. *American Journal of Physiology* 262, E891–E898.

Talanian, J.L. & Spriet, L.L. (2007) Low doses of caffeine late in exercise improve cycling time trial performance [abstract]. *FASEB Journal* 21, 107.

Tarnopolsky, M.A. (1994) Caffeine and endurance performance. *Sports Medicine* 18, 109–125.

Taylor, C., Higham, D., Close, G.L. et al. (2011) The effect of adding caffeine to postexercise carbohydrate feeding on subsequent high-intensity interval-running capacity compared with carbohydrate alone. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 410–426.

Van Nieuwenhoven, M.A., Brummer, R.M., & Brouns, F. (2000) Gastrointestinal function during exercise: comparison of water, sports drink and sports drink with caffeine. *Journal of Applied Physiology* 89, 1079–1085.

Van Soeren, M.H., Sathasivam, P., Spriet, L.L., et al. (1993) Caffeine metabolism and epinephrine responses during exercise in users and non-users. *Journal of Applied Physiology* 75, 805–812.

Vergauwen, L., Hespel, P., & Richter, E.A. (1994) Adenosine receptors mediate synergistic stimulation of glucose uptake and transport by insulin and by contractions in rat skeletal muscle. *Journal of Clinical Investigation* 93, 974–981.

Warren, G.L., Park, N.D., Maresca, R.D., et al. (2010) Effect of caffeine on muscular strength and endurance: a meta-analysis. *Medicine & Science in Sports & Exercise* 42, 1375–1387.

Yeo, S.E., Jentjens, R.L.P.G., Wallis, G.A., et al. (2005) Caffeine increases exogenous carbohydrate oxidation during exercise. *Journal of Applied Physiology* 99, 844–850.

Chapter 26

Buffering Agents

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Introduction

Fatigue is generally thought of as the inability to maintain the required or expected force or power output (Edwards, 1981). The causes of fatigue are multifaceted, with the limitations to exercise performance being influenced by mode, intensity, and duration as well as other factors such as diet and environment. Intense exercise of short duration results in a change in muscle biochemistry that stresses several physiological mechanisms that might subsequently contribute to fatigue. Energy demand on the muscle is increased by high-intensity exercise, with the link between chemical energy and mechanical work being mediated by adenosine-5′-triphosphate (ATP). To meet the increased energy demand, ATP is hydrolyzed to adenosine-5′-diphosphate (ADP), although the store of ATP is limited and must be continually resynthesized. ATP resynthesis is facilitated by the hydrolysis of phosphorylcreatine (PCr), glycolysis, and, as long as oxygen is available, oxidative phosphorylation in mitochondria, providing ATP from pyruvate or fat. However, during high-intensity exercise, the rate of ATP hydrolysis can exceed the rate of ATP resynthesis provided by aerobic metabolism, meaning that the shortfall in ATP must be met by the hydrolysis of PCr and anaerobic glycolysis (Hultman & Sjöholm, 1983), although meeting these energy demands comes at a cost. Simultaneously, these (particularly the fast action of the creatine kinase (CK)/PCr system) maintain ADP in the narrow concentration band consistent with continuation of muscle contraction.

Exercise performed at high intensities results in the formation of two carboxylic acid groups produced as a consequence of the oxidation of each glucose or glucosyl (from glycogen) unit metabolized. We now know that acidification in the muscle is the result of the formation of these carboxyl groups, most of which is accounted for by the increase in dissociated lactic acid. With a pK_a (the pH of a solution where the acid is 50% dissociated) of 3.86, lactic acid is almost totally dissociated into lactate anions (La^-) and hydrogen cations (H^+) across the physiological pH range (pH 7.1–6.0), as indicated by the Henderson–Hasselbalch equation. This describes the relationship between the pH of a solution, the pK_a of a weak acid group, and the concentration ratio of the acid and conjugate base. This is supported by the fact that ~94% of the increase in protons during exercise is linked with the accumulation of H^+ in the contracting skeletal muscle, with the remaining ~6% being accounted for by other acids and the accumulation of glycerol-1-phosphate and glucose-6-phosphate (Hultman & Sahlin, 1980). This suggests that anaerobic glycolysis is the primary metabolic process responsible for H^+ production (which initially occurs in the Embden–Meyerhoff pathway with the ionization

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of the carboxyl group of 1,3 diphosphoglycerate) in skeletal muscle during high-intensity exercise, although alternative sources of H^+ production have been suggested (Robergs et al., 2004).

Robergs et al. (2004) argued that a large portion of the metabolic acidosis occurring during exercise is a result of the breakdown of ATP to ADP. While it is clear that the breakdown of ATP to ADP does yield some H^+, the relative changes in ATP and ADP concentration are small during high-intensity exercise and the resynthesis of ATP from ADP would consume some of the H^+ produced in the reverse reaction. Furthermore, the pK_a of the terminal phosphate group of ATP (6.5) is close to the pK_a of inorganic phosphate (6.8) and that of the hexose and triose phosphates of glycolysis, which is the alternative fate of the phosphate released from ATP. Net loss of adenine nucleotides to inosine monophosphate, resulting in the release of two phosphate groups and also the release of one ammonia, occurs only in the low millimolar range. The impact of ATP breakdown on H^+ concentrations in the muscle is therefore likely to be small and the major cause of metabolic acidosis is the relatively much larger contribution of H^+ resulting from the accumulation of dissociated lactic acid.

In order to maintain homeostasis, the body must create a balance between the production and removal of H^+. Failure to do this during exercise will result in the accumulation of H^+ in the tissues and a reduction in the intracellular pH (pHi). There are several ways in which humans can regulate acid–base balance and minimize changes to pHi in the tissues, according to differing time frames. Chemical and metabolic buffers can alter the H^+ concentration in seconds, pulmonary ventilation can excrete H^+ over the course of minutes, and the kidneys can excrete H^+ over a longer time frame. In this chapter, we are primarily concerned with chemical and metabolic buffers in the intracellular (i.e., muscle) and extracellular (i.e., blood) environments which can assist with pHi control during exercise.

Under resting conditions, in healthy individuals, intramuscular pH is around 7.0, with arterial and venous blood pH being slightly higher at 7.4 and 7.3. However, during high-intensity exercise, muscle pH may fall to between 6.5 and 6.0 depending upon the method of determination, with the lower values only being reported using proton nuclear magnetic resonance spectroscopy (Pan et al., 1991). Higher values reported in muscle homogenates are almost certainly an artifact of the method employed, due to further post-sampling hydrolysis of PCr and the incorporation of extracellular proteins. Arterial and venous blood pH both decline to ~7.0. There are three potential sources of buffering in skeletal muscle, which help to determine the rate and extent of pHi decrease with the production of H^+ during exercise: physicochemical buffering, metabolic buffering, and dynamic buffering. Physicochemical buffering can occur in closed systems through, for example, the muscle content of histidine-containing dipeptides (of which carnosine is one) or in open systems through, for example, the intra- and extracellular levels of bicarbonate. Physicochemical buffers are dependent upon the equilibrium of their ionized/unionized forms across the physiological pH range and their effectiveness as a buffer is therefore dependent upon their pK_a. In order to be effective, intramuscular physicochemical buffers need to have a pK_a that is in the middle of the physiological pH range (6.1–7.0) and they also need to be present in high concentrations. Metabolic buffers act in a similar way to physicochemical buffers and include both PCr and weak acids within the muscle. PCr buffering is provided through the release of phosphate (which, as part of the PCr molecule, has a pK_a of 4.50) as inorganic phosphate (pK_a 6.8), even if some of this is subsequently metabolized to hexose or triosephosphates (pK_a's of the order of 6.1). Only relatively low levels of weak acids exist within the muscle at rest. However, the increased metabolic rate associated with high-intensity exercise can increase the concentration of these to levels at which they might be able to contribute to intracellular buffering.

Despite the role played by both physicochemical and metabolic buffering, intramuscular pH can still fall to as low as 6.0 (Pan et al., 1991), concomitant with the accumulation of La^-, which can exceed concentrations of 23 mmol/kg wet weight (~100 mmol/kg dry muscle). As a result, it is important to transport H^+ and La^- out of the muscle cell in order to prevent a further reduction in pHi and

to reduce the cellular concentrations of La^-. The removal of H^+ in this way allows chemical buffers (of which bicarbonate is the main protagonist) in the extracellular environment to assist in acid–base regulation.

La^- efflux from the muscle cell is mediated by specific monocarboxylate transporters (Juel & Halestrap, 1999), which co-transport La^- with H^+ across the cell membrane in a 1:1 ratio, with the rate of efflux primarily related to the La^- gradient, and to a lesser extent pHi (McDermott & Bonen, 1993). This enables the metabolism to be matched without causing an imbalance of H^+ in the cell, indicating that La^- transport is critical to muscle pHi regulation, perhaps even more so than either the Na^+–H^+ exchanger or the transport via bicarbonate (Juel, 1995). Despite this, however, the rate of co-transport increases only up to fourfold during high-intensity exercise (Juel & Halestrap, 1999), probably due to the relatively low (compared with the physiological range of intramuscular lactate) K_m of La^- of the monocarboxylate transporter (Watt et al., 1988). As a result, La^- and H^+ will still accumulate in the muscle during exercise. This provides additional evidence for the importance of intracellular buffers in dealing with abrupt changes in H^+ concentrations.

When the accumulation of H^+ in the muscle as a result of high-intensity exercise exceeds the rate of removal from the muscle cytoplasm, reductions in pHi will occur. Osnes and Hermansen (1972) examined the effect of running distances of between 100 and 5000 m on La^- accumulation in the blood. Blood La^- levels increased in line with the distance covered up to 1500 m, with no further increases in blood La^- concentration being shown as distance increased thereafter. In addition, the pH and blood bicarbonate levels were lowest following the 1500 m bout, suggesting that exercise durations of around 4 minutes provide the greatest challenge to the maintenance of acid–base control. Table 26.1 provides some examples of Olympic events which fall into a time frame that might be positively affected by the use of buffering agents.

Metabolic acidosis has been linked with a reduction in force production and the onset of fatigue. Acidosis has been shown to interfere with several metabolic processes that might explain an effect on force production and fatigue, including the accumulation of H^+ in skeletal muscle that might disrupt the recovery of PCr (Harris et al., 1976), the inhibition of glycolysis (Trivedi & Daniforth, 1966), or the direct disruption of the functioning of muscle contractile machinery (Fabiato & Fabiato, 1978). More recently, others have also suggested that H^+ accumulation in the blood might result in an increased perception of effort during high-intensity intermittent exercise (Price & Moss, 2007), which could also indirectly contribute to fatigue.

Table 26.1 Selection of Olympic events having exercise durations which suggest a potential benefit from supplementation with buffering agents

Olympic sporting event		Recent Olympics gold medal times	
		Men	Women
Running	400 m hurdles	47.63	52.70
	800 m	1.40.91	1.56.19
	1500 m	3.34.08	4.10.23
Swimming	100 m freestyle	47.52	53.00
	200 m freestyle	1.43.14	1.53.61
	400 m freestyle	3.40.14	4.01.45
Flat-water kayaking	500 m K1	N/A	1.51.46
	1000 m K1	3.26.46	N/A
Cycling	Track team pursuit	3.52.50	3.15.67
	BMX	37.58	37.71
Rowing	2000 m 8+	5.48.75	6.10.59
	2000 m 4 Sculls	5.42.48	6.35.93
Speed skating	1500 m	1.45.57	1.56.89
Alpine skiing	Downhill	1.54.31	1.44.19

Cooke and Pate (1985) have provided some evidence of an effect of pH on muscle force generation *in vitro*, showing a linear decrease of around 30% in the isometric tension of muscle fibers as pH decreases between 7.0 and 6.5. In addition, studies have shown an association between an increase in muscle buffering capacity and an improvement in high-intensity exercise performance and capacity (e.g., Bishop et al., 2004; Edge et al., 2006).

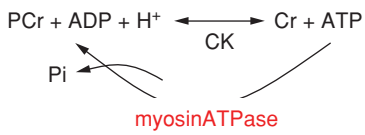
Given the potential for high-intensity exercise to result in metabolic acidosis as the result of increased H^+ production, coupled with La^- production, and given the effect that this might have on muscle

metabolism and function, any intervention capable of reducing the negative impact of H^+ accumulation would be of benefit to the athlete or athletic individual. Several dietary supplements might be of some use in this regard, including chronic supplementation with creatine monohydrate or β-alanine/carnosine to increase intracellular pHi buffering and acute supplementation with sodium bicarbonate/citrate to increase extracellular buffering.

Supplements to Increase Intracellular Buffering

Creatine/Phosphorylcreatine

Creatine (Cr) is a guanidino compound synthesized from arginine and glycine in the liver and kidney. Cr exists in high concentrations in the skeletal muscle with around 95% of the body content stored here. Approximately 65% of the intracellular store is in the form of PCr. Undoubtedly the most important physiological role for PCr is as a high-energy phosphate donor to facilitate ATP resynthesis during exercise. Maintenance of a high ATP to low ADP ratio is facilitated by the high activity of CK in muscle, and high concentrations of Cr and PCr. As a part of the CK-catalyzed reaction, H^+ are also utilized and hence buffered:



It is, therefore, possible that the breakdown of PCr could act as a suitable metabolic buffer during high-intensity exercise. Hultman and Sahlin (1980) estimated that hydrolysis of PCr, in the absence of any net change in ATP (resulting in the accumulation of phosphate (Pi)), could account for up to 30% of the total muscle buffering capacity. At rest, skeletal muscle concentrations of Cr plus PCr are between 100 and 150 mmol/kg dm, with concentrations of PCr being between 75 and 90 mmol/kg dm. Concentrations may be increased by up to 20% with supplementation of creatine monohydrate (Harris et al., 1992). As such, this approach potentially offers a means to increase muscle buffering capacity. However, Hultman and Sahlin (1980) estimated that an increase of around 20% in muscle PCr would only be sufficient to elevate muscle buffering capacity by around 3%, even with hydrolysis of most of the PCr, since it is only the hydrolysis of PCr to Cr and Pi that would add significantly to muscle buffering capacity. In terms of the direct buffering properties of PCr, it is unlikely to offer a significant contribution given a pK_a of 4.50, which is well below the physiological pH range.

Numerous studies have now shown a positive effect of Cr monohydrate supplementation at 20 g/day for 5–6 days on sprint and high-intensity exercise performance (for a recent review, see Tarnopolsky, 2010). However, for the reasons cited above, the majority of this effect is likely to be related to anaerobic energy delivery and not to an increase in muscle buffering capacity. As such, we will not discuss Cr supplementation further here but instead refer the reader to Chapter 24 for a more comprehensive discussion of this topic.

β-Alanine/Carnosine

Carnosine (β-alanyl-L-histidine) is a cytoplasmic dipeptide and is formed, mainly in muscle tissue, by bonding histidine and β-alanine in a reaction catalyzed by carnosine synthase. The concentrations of carnosine vary considerably in the muscles of different species and also between muscle fiber types. In humans, skeletal muscle concentrations (*m. vastus lateralis*) have been reported to be around 21 ± 4 mmol/kg dm in males and 18 ± 5 mmol/kg dm in females (Mannion et al., 1992), with the highest concentrations identified in fast-twitch fibers (Harris et al., 1998; Hill et al., 2007). There is an effect of diet, with vegetarians having significantly lower muscle levels than meat eaters (Everaert et al., 2011; Harris et al., 2007).

Studies of the chemical properties of carnosine demonstrated a pK_a of 6.83 for the imidazole ring of the histidine residue, making it a suitable buffer over the physiological pH range (Bate-Smith, 1938). Further support for the work of Bate-Smith (1938) comes from Tanokura et al. (1976) who used nuclear magnetic resonance spectroscopy to show that carnosine has pK_a's of 2.77 (carboxyl group),

9.66 (amino group), and 6.83 (imidazole ring). Further indirect evidence to support a role for carnosine in intramuscular buffering comes from comparative physiology which indicates that the highest muscle content of the histidine-containing dipeptides (of which carnosine is one) is found in those species whose muscles are frequently exposed to bouts of hypoxia, such as diving mammals, where an ability to buffer reductions in intramuscular pH would be a distinct advantage. Similarly, histidine-containing dipeptide contents are also high in the muscles of animals that depend upon hunting or escaping for survival and in several species involved in athletic competition, such as horses, greyhounds, and camels (Dunnett & Harris, 1997; Harris et al., 1990). Parkhouse et al. (1985) showed that sprinters and rowers, as examples of athletes performing high-intensity exercise, had higher muscle buffering capabilities and skeletal muscle carnosine levels than marathon runners and untrained subjects.

These findings support the assertion that pH buffering is a key physiological function of histidine-containing dipeptides in skeletal muscle and that evoking an increase in skeletal muscle carnosine levels would increase muscle buffering capacity and improve pHi regulation. While a role for carnosine in pH buffering is undisputable, since this is determined by its molecular structure and the fundamental laws of chemistry, it should be noted that other physiological roles for carnosine have also been suggested, which could contribute to enhanced exercise performance (for a review, see Sale et al., 2010). Taken together, these findings suggest that the elevation of muscle carnosine concentrations could provide a method of increasing intracellular buffering capacity during exercise, thus increasing high-intensity exercise capacity and performance.

Carnosine is synthesized in muscle from its constituent amino acids; the intact dipeptide is not transported into muscle. In humans, any carnosine appearing in blood (for instance as a result of leakage from damaged muscle fibers) is rapidly broken down to β-alanine and histidine by the enzyme carnosinase and these are then available for transport into muscle and other tissues. β-alanine transport into muscle is mediated by a specific β-amino acid transport protein that has a K_m of ~40 μM. The resynthesis of carnosine in muscle tissue is limited by the very low concentration of β-alanine and the relatively high K_m (1.0–2.3 μM) that β-alanine has for carnosine synthase. Histidine, on the other, hand is present in much higher concentrations in muscle and exhibits a lower K_m for carnosine synthase. This suggests that carnosine synthesis in the muscle is limited by the availability of β-alanine.

β-alanine is synthesized in the liver from the degradation of uracil (Mattews & Traut, 1987), a pyrimidine base which is also used in the synthesis of RNA. As yet there are no estimates of the hepatic output of β-alanine but this is clearly not saturating to the synthesis of carnosine in muscle, as indicated by the much lower muscle carnosine content in vegetarians. Meat eaters, on the other hand, obtain variable amounts of β-alanine from the ingestion of the muscle meat of animals and fish. Carnosine and related methyl derivatives (anserine and balenine) undergo hydrolysis during absorption and transport, with the β-alanine entering the general pool available to muscle and other tissues. Amounts available are highly variable according to the meat ingested (chicken and turkey breast meat being five times higher than pork, beef, or lamb), the quantity eaten, and the method of preparation (e.g., stewing results in the loss of carnosine into the stock which may or may not be consumed).

Direct supplementation with synthetic β-alanine is also available, with the peak in the plasma concentration occurring within 30–45 minutes. Supplementation with β-alanine has consistently been shown, in both muscle biopsy (e.g., Harris et al., 2006; Hill et al., 2007) and proton magnetic resonance spectroscopy studies (e.g., Baguet et al., 2009; Derave et al., 2007), to increase muscle carnosine concentrations by between 40% and 80% depending upon the dose given (between 3.2 and 6.4 g/day) and the duration of supplementation (between 4 and 10 weeks). Supplementation results in equal increases in both types I and II muscle fibers despite the fact that initially the carnosine content of type II fibers is 1.5–2 times higher than in type I (Harris et al., 1998; Kendrick et al., 2009; Tallon et al., 2007). Recently, Stellingwerff et al. (2012a) reported that it is the total amount of β-alanine consumed (dose × duration) that influences the absolute increase in

muscle carnosine and not the baseline muscle carnosine content, muscle fiber type, or the daily dose alone. An important point to note from the studies examining the effect of β-alanine supplementation on muscle carnosine content is that only one participant has failed to respond to β-alanine supplementation (see Harris et al., 2006). By comparison, there are reports suggesting that around 30% of subjects do not respond to creatine supplementation (Syrotuik & Bell, 2004).

When administered as a drink or in hard gelatine capsules, subjects have frequently reported symptoms of paresthesia above a dose of 10 mg/kg BM. At 40 mg/kg BM all subjects reported symptoms and recorded them as being uncomfortable. Paresthesia is a neuropathic pain with symptoms similar to extreme pins and needles. It is almost certainly caused by β-alanine-induced multiple action potentials of dorsal root ganglia neurons terminating in the skin (Crozier et al., 2007). To combat this, a sustained release formulation is now available (CarnoSyn™), which imposes a physical restraint on the rate of release of β-alanine. With sustained release tablets, the peak concentration in plasma achieved with a single dose is greatly attenuated, but release into plasma is maintained over 6 hours (Harris et al., 2008; Stellingwerff et al., 2012b). With cessation of supplementation, muscle carnosine returns to the pre-supplementation level, with a half time estimated to be between 5 and 9 weeks (Baguet et al., 2009; Harris et al., 2008; Stellingwerff et al., 2012a). Using sustained release tablets, each containing 800 mg β-alanine (CarnoSyn™, Natural Alternatives International, San Marcos, CA) symptoms of paresthesia are prevented even with single doses of 20 mg/kg BM (Sale et al., 2011). About 800 mg β-alanine is approximately the level that could be delivered from ingestion of ~175 g of chicken breast meat after hydrolysis of carnosine and anserine.

Hill et al. (2007) were the first to examine the effects of β-alanine supplementation on high-intensity exercise capacity using a cycling test performed at 110% of predetermined maximal power output (CCT110%). This test was designed to last between 2 and 4 minutes in order to induce a large accumulation of H^+ and a resultant drop in pHi. Subjects were supplemented with incremental doses ranging from 4.0 g/day in the first week to 6.4 g/day in the fourth week, which then continued for a further 6 weeks, meaning that subjects were supplemented for 10 weeks in total. Increases in muscle carnosine concentration were around 60% at 4 weeks and 80% at 10 weeks, with total work done during the CCT110% being increased by 13% at 4 weeks and 16% at 10 weeks.

The potential of β-alanine to enhance muscle buffering capacity through an elevation in muscle carnosine content and to subsequently increase exercise capacity has also been shown by our research group (Sale et al., 2012) during fixed exercise of the knee extensors at 45% of maximal voluntary contraction (MVC) which corresponds to the peak in La^- plus pyruvate accumulation in muscle (Ahlborg et al., 1972). Hold-time for the isokinetic exercise test was increased by 9.7 ± 9.4 seconds (13%) and impulse by 3.7 ± 1.3 kN/s (14%) following 4 weeks β-alanine supplementation at 6.4 g/day and this was significantly greater than the change in the placebo group. From the data of Ahlborg et al. (1972) it was estimated that the increase in hold-time would have resulted in the additional accumulation in muscle of ~11.5 mmol/kg dm La^- and H^+, matching the estimated increase in buffering capacity from carnosine increase. Endurance hold-times for both groups were not significantly different from values predicted by the Rohmert curve (Ahlborg et al., 1972); at 45% MVC the circulation to the quadriceps femoris is largely occluded by the increased intramuscular pressure with the result that the muscle exists as a closed system. Importantly, this study provides evidence that the mechanism by which β-alanine exerts its effect is through the increase in H^+ buffering capacity as a result of the elevation in muscle carnosine levels.

Derave et al. (2007) have also examined the effects of 4 weeks β-alanine supplementation at 4.8 g/day on isometric muscle endurance of the knee extensors at what was claimed to be 45% MVC in trained 400 m runners. In contrast to the results of Sale et al. (2012), Derave et al. (2007) reported no significant effect of β-alanine on isometric hold-time. However, the pretreatment times to fatigue reported by Derave et al. (2007) were 175 and 201 seconds for the placebo and β-alanine groups, respectively,

which brings into question the true intensity of the exercise used in this study given that the hold-time at 45% MVC would be expected to be ~80 seconds (Ahlborg et al., 1972). According to Ahlborg et al. (1972), the intensity of the exercise in the Derave et al. (2007) study was probably closer to 25% MVC, at which intensity circulation is maintained.

Derave et al. (2007) also examined the effects of β-alanine supplementation on 400 m running performance on an indoor athletic track. This was one of the first studies to examine the effect of β-alanine on exercise performance rather than capacity. The 400 m run times were faster following supplementation in both the active and placebo treatment groups (~0.7 seconds), but there was no additional benefit of β-alanine.

For a more complete review of the effects of β-alanine supplementation on exercise performance and capacity, readers are directed toward our review on this topic (Sale et al., 2010). A further summary is provided by a recent meta-analytical review (Hobson et al., 2012) which reports a significant positive effect ($p = 0.002$) of β-alanine supplementation on the outcome of 57 exercise tests conducted on a total of 360 subjects from 15 published papers. Much of this positive effect was most likely explained by the significant increase ($p = 0.013$) shown in exercise capacity tests, with no effect of β-alanine being shown on exercise performance tests ($p = 0.204$). Although no effect of β-alanine supplementation on exercise performance-based tests was shown, it should be noted that the number of performance tests conducted on β-alanine supplementation to date is comparatively low ($N = 12$).

In line with the purported mechanisms for an ergogenic effect of β-alanine supplementation, Hobson et al. (2012) also report that exercise tests lasting between 1 and 4 minutes were improved ($p = 0.001$) with β-alanine supplementation over placebo, as were those exercise tests lasting longer than 4 minutes ($p = 0.046$). In contrast, there was no benefit of β-alanine on exercise tests lasting less than 1 minute ($p = 0.312$). Analysis indicates that supplementation with a total of 179 g of β-alanine (the median dose across those studies included in the meta-analysis) would result in a median improvement of 3% in an exercise test compared with placebo. However, it should be noted that this calculation is based upon the median effect of numerous tests across several studies. In the only test to be independently repeated (a cycling test performed at 110% of maximal power output), on both occasions following 4 weeks of β-alanine supplementation, increases in exercise capacity of 13% (Hill et al., 2007) and 15% (Sale et al., 2011) were recorded.

Direct supplementation with carnosine offers no advantage over β-alanine, since this is fully hydrolyzed on absorption. However, by this means, meat and fish are a source for β-alanine supplementation, although the meat is best eaten uncooked where possible (the best dietary source is whale beef). Modern dietary practices–-with the possible exception of some communities in the United States and South America, and certain nomadic tribes in Central Asia where meat is still the main component of the diet–-favor a low meat intake, well below that of our Paleolithic forebears. It may be that β-alanine supplementation is the only means acceptable to modern man to achieve a dietary intake comparable to that during the major period of hominid evolution and capable of sustaining a high carnosine level in muscle (see also Harris et al., 2012).

Supplements to Increase Extracellular Buffering

Sodium Bicarbonate

For some time, acute sodium bicarbonate supplementation has been recommended as a buffering agent against the development of metabolic acidosis, with work in this area commencing in the early 1930s. This initial work suggested that supplementation with sodium bicarbonate could increase the systemic levels of bicarbonate, thus increasing the pH gradient between the intracellular and extracellular compartments, promoting the transmembrane transport of H^+. We now know this to be the mechanism, as the muscle cell membrane is impermeable to bicarbonate (Katz et al., 1984). Blood bicarbonate buffers H^+ by combining with it to form carbonic acid which then quickly dissociates to form carbon dioxide and water:

$$H^+ + HCO^-_3 \leftrightarrow H_2CO_3 \leftrightarrow H_2O + CO_2$$

It is now well accepted that supplementation with a large acute dose of sodium bicarbonate in the region of 0.3 g/kg BM (i.e., 24 g for an 80 kg individual) is sufficient to increase the blood bicarbonate pool. Indeed, pre-exercise alkalosis has been shown consistently following supplementation with sodium bicarbonate (e.g., Costill et al., 1984; Gaitanos et al., 1991; Katz et al., 1984) at sufficient levels to be of benefit to high-intensity exercise performance and capacity (for review, see McNaughton et al. 2008). A recent meta-analytical review on the topic reports that the ingestion of between 0.3 and 0.5 g/kg BM of sodium bicarbonate can result in an increase of 1.7% in mean power output during high-intensity exercise lasting around 60 seconds (Carr et al., 2011).

In addition to the studies examining the effects of acute sodium bicarbonate supplementation on exercise performance and capacity, others have also examined the effects of chronic sodium bicarbonate supplementation. The advantage of this approach is that subjects can take the supplement over a period of time without the requirement to take one large dose acutely prior to exercise, thus potentially preventing the gastrointestinal symptoms often associated with supplementation. Douroudos et al. (2006) examined the effects of 0.3 and 0.5 g/kg BM of sodium bicarbonate, given in four daily doses over 5 days, on performance during the Wingate test and reported a significant increase in mean power output in a dose-dependent manner. These findings concur with previous studies examining the chronic administration of sodium bicarbonate over 5 days at 0.5 g/kg BM (McNaughton et al., 1999). While these results are promising, the study did not use a standard crossover design and the choice of exercise test might be questioned given that others have indicated that reduced pH is not the cause of fatigue during a single bout of 30 seconds of maximal cycling (Bogdanis et al., 1998).

The reported effects of sodium bicarbonate supplementation on high-intensity exercise are not consistent, however, as several studies have shown either no effect, or a reduced effect, of supplementation on exercise performance and capacity. To summarize, the investigations showing no effect have tended to use lower doses (e.g., ≤0.2 g/kg BM; Katz et al., 1984), shorter exercise durations (e.g., 10 × 6 seconds; Gaitanos et al., 1991), and lower exercise intensities (e.g., 70% of 1 RM; Webster et al., 1993). That said, several studies have still shown significant improvements in exercise performance following sodium bicarbonate supplementation when using lower doses (Costill et al., 1984; McNaughton, 1992). The equivocal findings from these studies might be explained by individual responses to sodium bicarbonate supplementation (Price & Simons, 2010; van Montfoort et al., 2004) that in some cases, but by no means all, might be explained by the gastrointestinal discomfort often accompanying supplementation at this level.

Despite this, few studies have included sufficient subject numbers to adequately account for individual variability in responses, or to remove subjects suffering from gastrointestinal discomfort from the analysis. In general, sample sizes range between 5 and 10 subjects, although some published studies have exceeded this (Lavender & Bird, 1989: N = 23; van Montfoort et al., 2004: N = 15). Recently, Saunders et al. (2011) examined the effect of sodium bicarbonate supplementation on cycling capacity at 110% of maximum power output in 21 male subjects. In this study, sodium bicarbonate supplementation did not significantly improve high-intensity cycling capacity when all subjects were included in the analysis. However, when the subjects suffering from gastrointestinal discomfort (N = 4) were removed from the analysis (conducted with N = 17), exercise capacity was significantly improved. However, gastrointestinal discomfort did not explain all of the individual variability in exercise capacity outcomes. Similarly, the degree of alkalemia prior to exercise could not account for the individual differences in exercise capacity, although exercise affected blood markers differently between participants who improved and those who did not. It would seem that sodium bicarbonate has a degree of efficacy for some, but not all individuals.

Sodium Citrate

In addition to the use of sodium bicarbonate, several studies have also examined the effects of sodium citrate on high-intensity exercise performance and capacity (Kowalchuk et al., 1989; McNaughton

et al., 1990; Parry-Billings et al., 1986; Tiryaki & Atterbom, 1995), primarily because it is purported to act in a similar way to sodium bicarbonate but without the associated gastrointestinal distress. The utilization of protons during the oxidation of citrate effectively generates bicarbonate, so the ingestion of sodium citrate allows the indirect buffering of H^+ to occur in a very similar manner to that of sodium bicarbonate. The majority of studies do indeed report conversion of the citrate to bicarbonate prior to exercise testing.

Previous studies using sodium citrate to increase extracellular buffering have shown an increase in exercise performance and capacity in a dose-dependent manner between 0.3 and 0.5 g/kg BM (McNaughton, 1990). Subsequent studies have confirmed the efficacy of the supplement in exercise tests lasting between 2 and 4 minutes (McNaughton & Cedaro, 1991), which would support the purported mechanism of action. That said, Potteiger et al. (1996a) have shown beneficial effects of sodium citrate ingestion on exercise performance of longer durations (30 km cycling time trial performance), when subjects ingested doses of 0.5 g/kg BM. However, the same research group did not show any effect of either sodium citrate or bicarbonate in a further study of endurance running performance (Potteiger et al., 1996b). In line with the results relating to some of the other buffering agents reported earlier in this chapter, sodium citrate has been shown to be less efficacious during exercise performance of less than 30 seconds (McNaughton & Cedaro, 1991; Parry-Billings & Maclaren, 1986).

van Montfoort et al. (2004) compared the effects of sodium bicarbonate ingestion with the ingestion of other buffering agents (sodium citrate and sodium lactate) and placebo (sodium chloride) on sprint running capacity. In this study, subjects ingested 0.3 g/kg BM of sodium bicarbonate, with the doses of all the other buffering agents being provided in equal osmotic strength to this dose. Sodium citrate, sodium lactate, and sodium chloride were provided at doses of 0.525, 0.400, and 0.209 g/kg BM. Mean run times to exhaustion, at running speeds designed to result in fatigue between 1 and 2 minutes, were 82.3 seconds for sodium bicarbonate, 80.2 seconds for sodium lactate, 78.2 seconds for sodium citrate, and 77.4 seconds for sodium chloride. This study contradicts some of the previous work in this area and suggests that sodium citrate was not as effective as sodium bicarbonate at improving high-intensity running capacity. However, subjects in this study did not report any significant gastrointestinal symptoms following sodium bicarbonate ingestion, which might have explained why the majority of subjects performed better following the ingestion of sodium bicarbonate in contrast to many other studies.

The findings of van Montfoort et al. (2004) also seem to be supported by the findings of a recent meta-analytical review by Carr et al. (2011), who reported that the performance effect of sodium citrate was unclear. By comparison, sodium bicarbonate was reported to result in a clearly increased exercise performance, as highlighted above. The unclear performance effect of sodium citrate ingestion was reported despite the fact that sodium citrate was at least as effective in increasing blood bicarbonate concentrations and pH as sodium bicarbonate.

Summary

High-intensity exercise results in the production of lactic acid, when the rate of glycolysis in muscle is higher than the rate of pyruvate oxidation, with the resultant metabolic acidosis being caused by the intracellular accumulation of H^+. Despite the action of buffers in the muscle, H^+ can still accumulate and must be transported across to the blood, resulting in a reduction in blood pH. Acidosis can contribute to muscle fatigue, particularly during intense bouts of exercise lasting between 1 and 5 minutes. A potential strategy to delay the onset of fatigue during these intense activities is to increase the buffering capacity in muscle and/or in the blood. Several dietary supplements have some potential to achieve such an effect, including chronic supplementation with Cr monohydrate or β-alanine/carnosine to increase pHi buffering and acute supplementation with sodium bicarbonate/citrate to increase extracellular buffering. In particular, there seems to be a

small but significant effect of both β-alanine and sodium bicarbonate supplementation on high-intensity exercise performance and capacity, particularly in tests lasting between 1 and 4 minutes (Carr et al., 2011; Hobson et al., 2012). However, the effect of both Cr monohydrate (from an intracellular buffering perspective) and sodium citrate supplementation on high-intensity exercise capacity and performance is less clear.

At the point of writing, only one study (Sale et al., 2011) had examined the effects of combining chronic supplementation of β-alanine, to enhance intracellular buffering, with the acute supplementation of sodium bicarbonate, to enhance extracellular buffering, on high-intensity exercise capacity. The results showed that supplementation with 6.4 g/day of β-alanine for 4 weeks improved high-intensity cycling capacity at 110% of powermax by 15%, confirming the previous results observed by Hill et al. (2007). Despite a further increase in exercise capacity of around 4% with the addition of sodium bicarbonate, this did not reach statistical significance, although magnitude-based inferences suggested a ~70% probability of a meaningful positive difference. Clearly a gain of 19% would confer a major advantage to athletes if such results could be repeated in the field. Table 26.2 summarizes the effects of intracellular and extracellular buffering agents on exercise performance and capacity.

Table 26.2 Summary table for the main supplements that could be used to enhance intracellular and extracellular buffering

Supplement	Dosing regimen	Possible side effects	Effect on exercise performance
Creatine monohydrate	Acute: 20 g/day for 5–7 days Chronic: 3–5 g/day for more than 31 days	Increased body mass	Improves • Repeated sprint performance • Intense exercise of 1–5 min No difference • Single sprint of <15 s Impairs • Prolonged exercise (from only one study)
β-alanine	6.4 g/day for 28 days gives a 60% increase in muscle carnosine and 70 days gives an increase of 80% Over longer durations better to use 3.2 g/day for 28 days followed by a 1.6 g/day dose thereafter	Paresthesia, although slow release β-alanine tablets seem to remove this side effect	Improves • Sprinting after a prolonged preload • Intense exercise of 1–5 min No difference • Single sprint of <30 s • Prolonged exercise Impairs • Currently none reported
Sodium bicarbonate	Acute: ~0.3 g/kg BM pre-event Chronic: 0.3–0.5 g/kg BM over 5 days	Gastrointestinal distress, can be reduced by ingesting with water or splitting ingestion over the 2–4 h pre-event	Improves • Repeated sprints • Intense exercise of 1–5 min No difference • Single sprints of <60 s Impairs • Prolonged exercise
Sodium citrate	~0.3 g/kg BM pre-event	Gastrointestinal distress, although often less severe than with sodium bicarbonate	Effects are similar to those following sodium bicarbonate ingestion but not to the same magnitude

References

Ahlborg, B., Bergström, J., Ekelund L., et al. (1972) Muscle metabolism during isometric exercise performed at constant force. *Journal of Applied Physiology* 33, 224–228.

Baguet, A., Reyngoudt, H., Pottier, A., et al. (2009) Carnosine loading and washout in human skeletal muscles. *Journal of Applied Physiology* 106, 837–842.

Bate-Smith, E.C. (1938) The buffering of muscle in rigour: protein, phosphate and carnosine. *Journal of Physiology* 92, 336–343.

Bishop, D., Edge, J., & Goodman, C. (2004) Muscle buffer capacity and aerobic fitness are associated with repeated sprinted ability in women. *European Journal of Applied Physiology* 92, 540–547.

Bogdanis, G.C., Nevill, M.E., Lakomy, H.K.A., et al. (1998) Power output and muscle metabolism during and following recovery from 10 and 20s of maximal sprint exercise in humans. *Acta Physiologica Scandinavica* 163, 261–272.

Carr, A.J., Hopkins, W.G., & Gore, C.J. (2011) Effects of acute alkalosis and acidosis on performance: a meta-analysis. *Sports Medicine* 41, 801–814.

Cooke, R. & Pate, E. (1985) The effects of ADP and phosphate on the contraction of muscle fibers. *Biophysical Journal* 48, 789–798.

Costill, D.L., Verstappen, F., Kuipers, H., et al. (1984) Acid–base balance during repeated bouts of exercise: influence of HCO_3. *International Journal of Sports Medicine* 5, 228–231.

Crozier, R.A., Ajit, S.K., Kaftan, E.J., et al. (2007) MrgD activation inhibits KCNQ/M-currents and contributes to enhanced neuronal excitability. *Journal of Neuroscience* 27, 4492–4496.

Derave, W., Ozdemir, M.S., Harris, R.C., et al. (2007) β-Alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. *Journal of Applied Physiology* 103, 1736–1743.

Douroudos, I.I., Fatouros, I.G., Gourgoulis, V., et al. (2006) Dose-related effects of prolonged $NaHCO_3$ ingestion during high-intensity exercise. *Medicine & Science in Sports & Exercise* 38, 1746–1753.

Dunnett, M. & Harris, R.C. (1997) High-performance liquid chromatographic determination of imidazole dipeptides, histidine, 1-methylhistidine and 3-methylhistidine in equine and camel muscle and individual muscle fibres. *Journal of Chromatography, B, Biomedical Applications* 688, 47–55.

Edge, J., Bishop, D., Hill-Haas, S., et al. (2006) Comparison of muscle buffer capacity and repeated sprint ability of untrained, endurance-trained and team-sport athletes. *European Journal of Applied Physiology* 96, 225–234.

Edwards, R.H.T. (1981) Human muscle function and fatigue. In *Human Muscle Fatigue: Physiological Mechanisms* (eds. R. Porter & J. Whelan). Pitman Medical, London.

Everaert, I., Mooyaart, A., Baguet, A., et al. (2011) Vegetarianism, female gender and increasing age, but not CNDP1 genotype, are associated with reduced muscle carnosine levels in humans. *Amino Acids* 40, 1221–1229.

Fabiato, A. & Fabiato, F. (1978) Effects of pH on the myofilaments and the sarcoplasmic reticulum of skinned cells from cardiac and skeletal muscles. *Journal of Physiology* 276, 233–235.

Gaitanos, G.C., Nevill, M.E., Brooks, S., et al. (1991) Repeated bouts of sprint running after induced alkalosis. *Journal of Sports Sciences* 9, 355–370.

Harris, R.C., Edwards, R.H.T., Hultman, E., et al. (1976) The time course of phosphorylcreatine resynthesis during recovery of the quadriceps muscle in man. *Pflugers Archives* 367, 137–142.

Harris, R.C., Marlin, D.J., Dunnett, M., et al. (1990) Muscle buffering capacity and dipeptide content in the thoroughbred horse, greyhound dog and man. *Comparative Biochemistry and Physiology* 97A, 249–251.

Harris, R.C., Soderlund, K., & Hultman, E. (1992) Elevation of creatine in resting and exercised muscle of normal subjects by creatine supplementation. *Clinical Science* 83, 367–374.

Harris, R.C., Dunnett, M., & Greenhaff, P.L. (1998) Carnosine and taurine contents in individual fibres of human vastus lateralis muscle. *Journal of Sports Science* 16, 639–643.

Harris, R.C., Tallon, M.J., Dunnett, M., et al. (2006) The absorption of orally supplied β-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids* 30, 279–289.

Harris, R.C., Jones, G., Hill, C.A., et al. (2007) The carnosine content of *v. lateralis* in vegetarians and omnivores. *FASEB Journal* 21, 769.20.

Harris, R.C., Jones, G., & Wise, J.A. (2008) The plasma concentration-time profile of β-alanine using a controlled-release formulation (Carnosyn™). *FASEB Journal* 22, 701.9.

Harris, R.C., Wise, J.A., Price, K.A., et al. (2012) Determinants of muscle carnosine content. *Amino Acids* 43, 5–12.

Hill, C.A., Harris, R.C., Kim, H.J., et al. (2007) Influence of β-alanine supplementation on skeletal muscle carnosine concentrations and high intensity cycling capacity. *Amino Acids* 32, 225–233.

Hobson, R.M., Saunders, B., Ball, G., et al. (2012) Effects of beta-alanine supplementation on exercise performance: a review by meta-analysis. *Amino Acids* 43, 25–37.

Hultman, E. & Sahlin, K. (1980) Acid–base balance during exercise. *Exercise and Sports Science Reviews* 8, 41–128.

Hultman, E. & Sjoholm, H. (1983) Substrate availability. In *Biochemistry of Exercise* (eds H.G. Knuttgen, J.A. Vogel, & J. Poortmans), pp. 63–75. Human Kinetic Publishers, Champaign, IL.

Juel, C. (1995) Regulation of cellular pH in skeletal muscle fiber types, studied with sarcolemmal giant vesicles obtained from rat muscles. *Biochimica et Biophysica Acta* 1265, 127–132.

Juel, C. & Halestrap, A.P. (1999) Topical review: lactate transport in skeletal muscle—role and regulation of the monocarboxylate transporter. *Journal of Physiology* 517, 633–642.

Katz, A., Costill, D.L., King, D.S., et al. (1984) Maximal exercise tolerance after induced alkalosis. *International Journal of Sports Medicine* 5, 107–110.

Kendrick, I.P., Kim, H.J., Harris, R.C., et al. (2009) The effect of 4 weeks beta-alanine supplementation and isokinetic training on carnosine concentrations in type I and II human skeletal muscle fibres. *European Journal of Applied Physiology* 106, 131–138.

Kowalchuk, J.M., Maltais, S.A., Yamaji, K., et al. (1989) The effect of citrate loading on exercise performance, acid–base balance and metabolism. *European Journal of Applied Physiology* 58, 858–864.

Lavender, G. & Bird, S.R. (1989) Effect of sodium bicarbonate ingestion upon repeated sprints. *British Journal of Sports Medicine* 23, 41–45.

Mannion, A.F., Jakeman, P.M., Dunnett, M., et al. (1992) Carnosine and anserine concentrations in the quadriceps femoris muscle of healthy humans. *European Journal of Applied Physiology* 64, 47–50.

Mattews, M.M. & Traut, T.W. (1987) Regulation of N-carbamoyl-beta-alanine amidohydrolase, the terminal enzyme in pyrimidine catabolism, by ligand-induced change in polymerization. *Journal of Biological Chemistry* 262, 7232–7237.

McDermott, J.C. & Bonen, A. (1993) Lactate transport by skeletal muscle sarcolemmal vesicles. *Molecular and Cellular Biochemistry* 122, 113–121.

McNaughton, L. (1990) Sodium citrate and anaerobic performance: implications of dosage. *European Journal of Applied Physiology* 61, 392–397.

McNaughton, L. (1992) Bicarbonate ingestions: effects of dosage on 60 seconds cycle ergometry. *Journal of Sports Sciences* 10, 415–423.

McNaughton, L. & Cedaro, R. (1991) Sodium citrate ingestion and its effect on maximal anaerobic exercise of different durations. *Australian Journal of Science and Medicine in Sport* 23, 66–69.

McNaughton, L., Backx, K., Palmer, G., et al. (1999) Effects of chronic bicarbonate ingestion on the performance of high-intensity work. *European Journal of Applied Physiology* 80, 333–336.

McNaughton, L., Siegler, J., & Midgley, A. (2008) Ergogenic effects of sodium bicarbonate. *Current Sports Medicine Reports* 7, 230–236.

Osnes, J.B. & Hermansen, L. (1972) Acid–base balance after maximal exercise of short duration. *Journal of Applied Physiology* 32, 59–63.

Pan, J.W., Hamm, J.R., Hetherington, H.P., et al. (1991) Correlation of lactate and pH in human skeletal muscle after exercise by ^{1}H NMR. *Magnetic Resonance Medicine* 20, 57–65.

Parkhouse, W.S., McKenzie, D.C., Hochachka, P.W., et al. (1985) Buffering capacity of deproteinized human vastus lateralis muscle. *Journal of Applied Physiology* 58, 14–17.

Parry-Billings, M. & Maclaren, D.P.M. (1986) The effect of sodium bicarbonate and sodium citrate ingestion on anaerobic power during intermittent exercise. *European Journal of Applied Physiology* 55, 524–529.

Potteiger, J.A., Nickel, G.L., Webster, M.J., et al. (1996a) Sodium citrate ingestion enhances 30km cycling performance. *International Journal of Sports Medicine* 17, 7–11.

Potteiger, J.A., Webster, M.J., Nickel, G.L., et al. (1996b) The effects of buffer ingestion on metabolic factors related to distance running performance. *European Journal of Applied Physiology* 72, 365–371.

Price, M.J. & Moss, P. (2007) The effects of work:rest duration on physiological and perceptual responses during intermittent exercise and performance. *Journal of Sports Sciences* 25, 1613–1621.

Price, M.J. & Simons, C. (2010) The effect of sodium bicarbonate ingestion on high-intensity intermittent running and subsequent performance. *Journal of Strength and Conditioning Research* 24, 1834–1842.

Robergs, R.A., Ghiasvand, F., & Parker, D. (2004) Biochemistry of exercise-induced metabolic acidosis. *American Journal of Physiology* 287, R502–R516.

Sale, C., Saunders, B., & Harris, R.C. (2010) Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids* 39, 321–333.

Sale, C., Saunders, B., Hudson, S., et al. (2011) Effect of β-alanine plus sodium bicarbonate on high-intensity cycling capacity. *Medicine & Science in Sports & Exercise* 43, 1972–1978.

Sale, C., Hill, C.A., Ponte, J., et al. (2012) Beta-alanine supplementation improves isometric endurance of the knee extensor muscles. *Journal of the International Society of Sports Nutrition* 9, 26.

Saunders, B., Sale, C., Harris, R.C., et al. (2011) Effect of sodium bicarbonate supplementation on cycling capacity at 110% of maximum power output. *Medicine & Science in Sports & Exercise* 43, S588.

Stellingwerff, T., Anwander, H., Egger, A., et al. (2012a) The effect of two β-alanine dosing protocols on muscle carnosine synthesis and washout. *Amino Acids* 42, 2461–2472.

Stellingwerff, T., Decombaz, J., Harris, R.C., et al. (2012b) Optimizing human *in vivo* dosing and delivery of beta-alanine supplements. *Amino Acids* 43, 57–65.

Syrotuik, D.G. & Bell, G.J. (2004) Acute creatine monohydrate supplementation: a descriptive physiological profile of responder vs. nonresponders. *Journal of Strength and Conditioning Research* 18, 610–617.

Tallon, M.J., Harris, R.C., Maffulli, N., et al. (2007) Carnosine, taurine and enzyme activities of human skeletal muscle fibres from elderly subjects with osteoarthritis and young moderately active subjects. *Biogerontology* 8, 129–137.

Tanokura, M., Tasumi, M., & Miyazawa, T. (1976) 1H nuclear magnetic resonance studies of histidine containing di and tripeptides. Estimation of the effects of charged groups on the pK_a value of the imidiazole ring. *Biopolymers* 15, 393–401.

Tarnopolsky, M.A. (2010) Caffeine and creatine use in sport. *Annals of Nutrition & Metabolism* 57, S1–S8.

Tiryaki, G.R. & Atterbom, H.A. (1995) The effect of sodium bicarbonate and sodium citrate on 600 m running time of trained females. *Journal of Sports Medicine and Physical Fitness* 35, 194–198.

Trivedi, B. & Daniforth, W.H. (1966) Effect of pH on the kinetics of frog muscle phosphofructokinase. *Journal of Biological Chemistry* 241, 4110–4112.

van Montfoort, M.C.E., Van Dieren, L., Hopkins, W.G., et al. (2004) Effects of ingestion of bicarbonate, citrate, lactate, and chloride on sprint running. *Medicine & Science in Sports & Exercise* 36, 1239–1243.

Watt, P.W., MacLennan, P.A., Hundal, H.S., et al. (1988) L(+)-lactate transport in perfused rat skeletal muscle: kinetic characteristics and sensitivity to pH and transport inhibitors. *Biochimica et Biophysica Acta* 944, 213–222.

Webster, M.J., Webster, M.N., Crawford, R.E., et al. (1993) Effect of sodium bicarbonate ingestion on exhaustive resistance exercise performance. *Medicine & Science in Sports & Exercise* 25, 960–965.

Chapter 27

Alcohol, Exercise, and Sport

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Introduction

Alcohol is both a nutrient and a drug. It is not an essential part of the human diet, but various alcohols, of which the only one of quantitative significance is ethanol, are regularly consumed by a large part of the world's population. Annual per capita intake varies greatly between countries, exceeding 15 liters per adult in Russia and some of its former satellite states, while being close to zero in many of the predominantly Muslim states (World Health Organization, 2005). These values, though, are confounded by many factors, with many individuals abstaining entirely. Regular consumption in moderation may have some health benefits in terms of disease risk reduction, especially cardiovascular disease, but the association is not as convincing as is sometimes assumed (Piazza-Gardner & Barry, 2012). Even low intakes may substantially increase the risk of some cancers: a meta-analysis of 222 studies involving more than 92,000 drinkers and 60,000 nondrinkers suggests that one drink per day may increase the risk of esophageal cancer by about 30% (Bagnardi et al., 2013). High consumption, whether sustained or episodic, is undoubtedly associated with chronic diseases such as alcohol dependence, cancer, and liver cirrhosis, and acute health problems such as injuries (Gutjahr et al., 2001). Both current and lifetime alcohol consumption have been shown to be positively associated with overall and central obesity, in both women and men even after adjustment for age, education, smoking, energy intake, and regular physical exercise (Lourenco et al., 2012).

As well as being a significant source of energy, providing about 7 kcal (29 kJ) per gram, ethanol has a number of effects that have implications for athletic performance. There are reasons to believe that acute alcohol intake may impair the performance of endurance exercise because of effects on metabolic, cardiovascular, and thermoregulatory functions, and that its neurological actions may affect the performance of skilled tasks because of effects on reaction time, fine motor control, levels of arousal, and judgment. All of these elements are important components of sports performance, but there are relatively few well-controlled studies of performance itself. The limited amount of experimental evidence is at least in part because of the reluctance of institutional ethics review boards to condone the administration of high doses of ethanol to volunteers. There have been rather few studies in this area in recent years, and there is an urgent need for further studies, particularly investigations involving measurement of sport-specific performance. In the past few years, there appear to have been more review articles published on the effects of alcohol on sporting performance than original research papers. Recent reviews include those of El-Sayed et al. (2005), Maughan (2006), and Ferreira and Willoughby (2008).

Parra et al. (2012) have recently found evidence of a relationship between exercise participation and

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alcohol intake in both rats and undergraduate students. In spite of the potential for negative effects on performance and on health, there is ample evidence that alcohol features prominently in the lifestyles of many athletes at all levels of competition. There is evidence that at least some groups of athletes consume more alcohol than nonathletes, but separating fact from anecdote can be difficult. Data from a New Zealand student population show higher rates of hazardous drinking behaviors in elite sportspeople than in nonathletes (O'Brien et al., 2005). There are data from France to show a lower prevalence of alcohol use in athletic students (Lorente et al., 2003) and also data to show a greater prevalence of use (Lorente et al., 2004). Moore and Werch (2004) have highlighted some of the complexities that influence analyses of the relationship between sports participation and substance use: in their survey, school-sponsored, male-dominated sports were associated with increased alcohol use, but out-of-school, mixed-gender sports participation was associated with greater use in females. Reports that include only average intakes can be misleading, as binge drinking is commonly found in some team sports, and some athletes will abstain from alcohol in training but will drink copious amounts after competition (Burke & Maughan, 2000). The negative consequences of such excessive drinking may be greater for athletes than for their nonathletic peers (Martens et al., 2005).

There is also some evidence for a higher rate of alcohol consumption in physically active individuals who are not involved in competitive sports. In a cross-sectional population-based study of 34,653 adults in the United States, regular participation in vigorous physical activity was found to be positively associated with alcohol use in individuals under 50 years of age, but not in individuals over 50 years of age: the association between past-year moderate physical activity and alcohol use was stronger in men than in women (Lisha et al., 2011). The positive relationship between physical activity and alcohol consumption seems to be particularly strong in college student populations and appears not to be confounded by third variables including gender, ethnicity, age, college grade point average, fraternity/sorority membership, and sports participation (Musselman & Rutledge, 2010). In contrast to these findings, however, Ruiz-Juan and Ruiz-Risueno (2011) found a lower prevalence of alcohol use in Spanish adolescents who engaged in regular physical activity.

Understanding the reasons for alcohol use is crucial in providing help to those whose intake is excessive. Martens et al. (2006) have identified a number of reasons why athletes use alcohol and identified three main factors which they classified as positive reinforcement, team/group, and sport-related stress. It is interesting to note that, perhaps unexpectedly, the perceived drinking behaviors of high-profile athletes seem not to be significant predictors of sportspeople's drinking behaviors (O'Brien et al., 2010). O'Brien et al. (2011) have identified receipt of sponsorship from the alcohol industry as one factor associated with a higher level of drinking behavior in a large sample of Australian athletes.

Alcohol and Performance

Many of the studies on the effects of alcohol on athletic performance suffer from one or more defects. Subject numbers are often small, and the lack of a definite outcome may be the result of underpowered studies. The doses of alcohol that have been used in many studies are small relative to those reported to be consumed by athletes. The reluctance of investigators to induce inebriation in their subjects and the reluctance of ethics committees to sanction such studies because of safety concerns are understandable, but this nonetheless limits the application of the studies to the real world of sport. It is not always clear whether subjects have been fully familiarized with experimental protocols prior to measurements of performance, especially when tasks involving complex skills have been used. These factors may lead to performance changes that are of significance to the athlete being dismissed as being of no consequence (Hopkins et al., 1999). The older literature on alcohol and athletic performance, and of those aspects of physiological and metabolic functions that contribute to performance, has been reviewed by Reilly (2003) and Maughan (2006) and will not be repeated here.

Effects of Ethanol on Glycogen Metabolism

Resynthesis of the glycogen stores in liver and muscle is one of the key goals of athletes after intensive training or competition, and it is well recognized that ethanol has a variety of effects on carbohydrate metabolism in skeletal muscle and in liver. Much of the available evidence comes from older studies using animal models, showing that synthesis of glycogen in both liver and oxidative skeletal muscle is impaired in the presence of even relatively low levels of ethanol, though there seems to be no effect on type 2 muscle fibers. There is also evidence of impaired hepatic glucose output in the presence of even low doses of ethanol. This may be of particular concern during prolonged, moderate-intensity exercise when glucose output from the liver is an important source of energy. It is not entirely clear, however, that the animal data can be extrapolated to humans, especially to those habituated to moderate alcohol consumption.

Burke et al. (2003) reported the effects of alcohol intake on muscle glycogen storage in humans during recovery from a prolonged cycling bout that resulted in a substantial reduction of carbohydrate stores. Subjects undertook three different diets following their glycogen-depleting exercise: a high carbohydrate diet intended to optimize recovery, an isoenergetic alcohol displacement diet (reduced carbohydrate, in which about 210 g of dietary carbohydrate was replaced by about 120 g alcohol), and an alcohol + carbohydrate diet (about 120 g alcohol added to the high carbohydrate diet). Muscle glycogen storage was significantly reduced (by almost 50% at 8 hours and about 16% at 24 hours) on the alcohol displacement diet when the amount of carbohydrate provided by the diet was less than optimal (Figure 27.1). When the high carbohydrate diet was eaten,

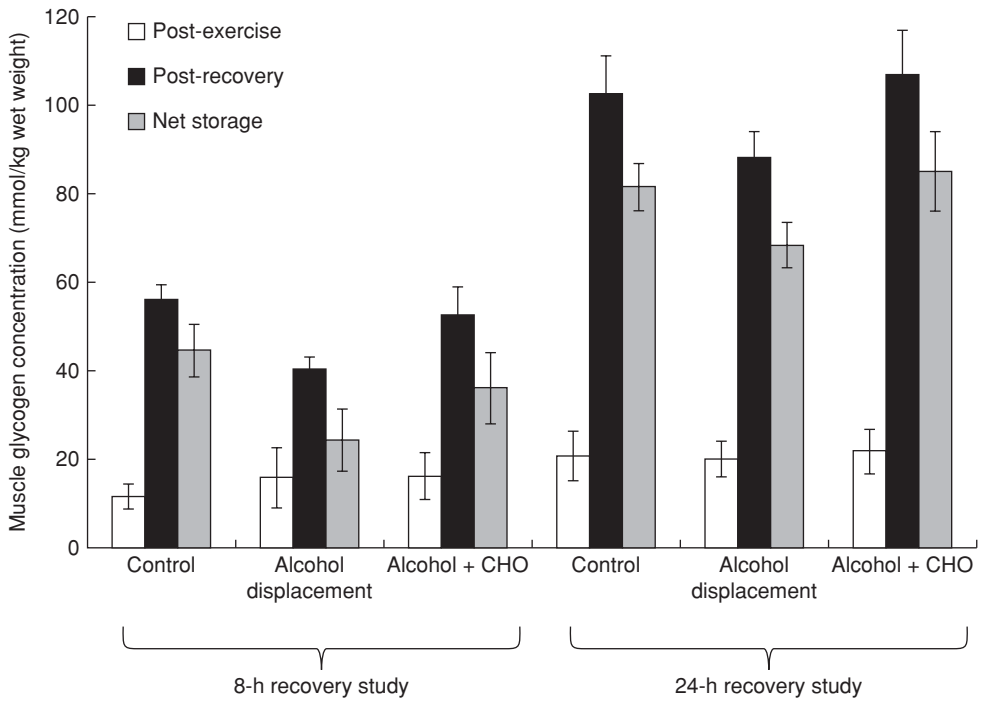

Figure 27.1 Muscle glycogen concentrations after exercise depletion and after a recovery period with different dietary interventions. Drawn from the data of Burke et al. (2003). Values are means ± SE for six subjects in the 8-hour study and nine subjects in the 24-hour study.

however, there was no clear evidence that alcohol intake caused a reduction in muscle glycogen storage: there was a small, not statistically significant reduction at 8 hours and no effect at all at 24 hours. It should be noted that there was a large variability in the responses of different subjects, and it may well be that some individuals will be unable to effectively replenish their glycogen reserves between daily training sessions if substantial amounts of alcohol are consumed.

Even if there is no direct metabolic effect of ethanol on glycogen storage when dietary carbohydrate intake is high, it is likely that athletes who consume large amounts of alcohol during the recovery period after training or competition will have a reduced carbohydrate intake, either as a result of a decreased total (nonalcoholic) energy intake or because of a failure to follow the recommended eating strategies at this time.

Alcohol, Exercise, and Oxidative Stress

Oxidative stress occurs when there is an imbalance between the rates of production and removal of reactive oxygen species (ROS) and free radicals leading to disturbances in cellular redox status. Free radicals are highly reactive chemical compounds by virtue of having one or more unpaired electrons in the outer orbit: they can damage components of the cell, including proteins, lipids, and DNA, by abstracting an electron. Some ROS, such as hydrogen peroxide, can cause similar damage to cells though they do not have an unpaired electron. Uncontrolled oxidative stress has been implicated in many diseases, including cardiovascular disease, Alzheimer's disease, and chronic fatigue syndrome, but ROS are also used by the immune system to kill invading pathogens and may have important roles in the process of adaptation to stress.

It is well established that physical exercise results in increased radical and ROS production in active skeletal muscles, though the mechanisms responsible remain under debate (Powers & Jackson, 2008). The metabolism of ethanol also generates ROS, including hydroxyl radicals, that may account for some of the harmful effects of chronic alcohol intake, including liver disease and myopathy (Ferreira & Willoughby, 2008). It is not clear whether the combination of exercise and alcohol intake may have synergistic effects on radical generation, with the possibility of adverse effects on both exercise and health. The issue of whether athletes should supplement their diet with antioxidant compounds that act as electron donors and thus reduce effects on cellular components is at present unresolved and is dealt with in more detail in Chapter 21. However, there is growing evidence that free radicals play an important role in some of the adaptations taking place in muscle in response to training (Powers & Jackson, 2008). There are also some indications that supplementation with high levels of the antioxidant vitamins E and C can attenuate the adaptations taking place in muscle in response to a short period of training (Gomez-Cabrera et al., 2008; Ristow et al., 2009).

The concept that modest levels of oxidative stress have beneficial effects on the organism may go beyond simply the issue of training adaptations. It has been proposed that similar effects may be involved in the health-promoting effects of exercise and, by extension, possibly also of regular moderate alcohol intake (Ristow & Zarse, 2010).

Hydration and Thermoregulatory Function

The diuretic action of ethanol is well recognized, and Shakespeare referred to this in the drunken porter's response to Macduff (Shakespeare, 1623). Eggleton (1942) quantified this effect and estimated an excess urine production of about 10 ml for each gram of ethanol ingested. She also reported that the diuretic action was greatly attenuated, or eliminated altogether, in individuals who were already hypohydrated. Subsequent studies showed that alcohol acts via suppression of the release of antidiuretic hormone from the pituitary. Shirreffs and Maughan (1997) confirmed the early report of Eggleton when they showed that alcohol has a negligible diuretic effect when consumed in dilute solution following a moderate level of hypohydration induced by exercise in the heat. There appears to be no difference in recovery from dehydration whether the rehydration

beverage is alcohol-free or contains up to 2% alcohol, but drinks containing 4% alcohol tend to delay the recovery process by promoting urine loss. Based on the data of Eggleton (1942), however, it is apparent that concentrated alcohol solutions will result in a net negative fluid balance: a 25 ml measure of spirits (40% ethanol) contains 10 ml of alcohol and 15 ml of water, resulting in a urine output of about 100 ml and a net negative water balance of 85 ml. Ingestion of large volumes of dilute alcohol will result in a water diuresis, but should promote restoration of fluid balance after sweat loss provided that there is also an intake of sodium which is essential for restoration of euhydration (Shirreffs & Maughan, 2005). This was confirmed in a recent study by Hobson and Maughan (2010) where the effect of consuming a dilute alcohol solution (weak beer) on urine production was assessed in euhydrated and hypohydrated individuals. Subjects exercised in hot, humid conditions to dehydrate by about 2% of body mass in the evening, and were then fed and rehydrated on two trials while on two other occasions they were fed the same meal but remained hypohydrated. The following morning they were given 1 liter of beer to drink. On two occasions, the beer was alcohol-free while on the other two occasions the same beer contained 4% ethanol. Urine output was followed for the subsequent 4 hours. Unsurprisingly, more urine was produced in both euhydrated trials than in either of the hypohydrated trials (Figure 27.2). When subjects were euhydrated, urine output was higher in the alcoholic than the nonalcoholic trials, confirming that a diuretic action was present, but there was no difference in the volume of urine produced between the alcoholic and nonalcoholic beer when hypohydrated. These results suggest that the diuretic action of alcohol is blunted when the body is hypohydrated.

The 1982 ACSM Position Stand on the use of alcohol in sports identified perturbations of thermoregulatory mechanisms, especially in the cold, as one of the reasons to abstain from alcohol prior to exercise. In warm environments, moderate doses of alcohol seem to have variable effects on thermoregulatory responses during exercise: varying patterns of thermal response are typically seen, with no consistent effect on the thermoregulatory

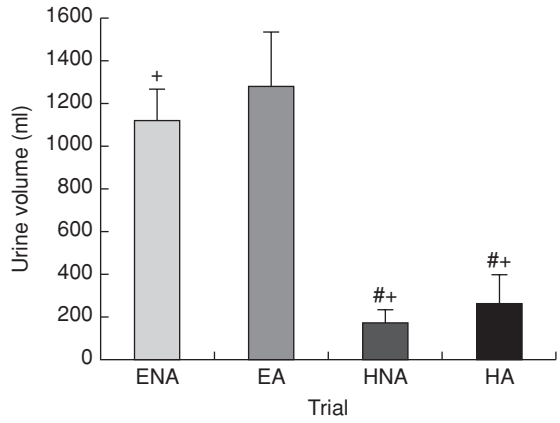

Figure 27.2 Total cumulative urine volume produced over the 4-hour observation period after drink ingestion in euhydrated or hypohydrated states. # denotes a significantly smaller volume produced than ENA. + denotes a significantly smaller volume than EA. ENA, euhydrated without alcohol; EA, euhydrated with alcohol; HNA, hypohydrated without alcohol; HA, hypohydrated with alcohol. Redrawn from Hobson and Maughan (2010), by permission of Oxford University Press.

response, suggesting that the capacity to regulate core temperature is not fundamentally altered by alcohol ingestion (Desruelle et al., 1996). With mild thermal stress in the absence of exercise, there may be a slight increase in skin temperature and in the sensation of warmth after alcohol ingestion, and this is followed by a fall in core temperature (Yoda et al., 2005). Small doses of ethanol, given to human volunteers at rest in the absence of a thermal stress, have very little effect on body temperature but large doses administered before exercise at low ambient temperatures result in increased peripheral vasodilatation and can precipitate a marked fall in core temperature. In combination with the concomitant fall in blood glucose concentration that is normally observed in this situation, there is clearly potential for an adverse effect on performance. Graham (1981) showed that ingestion of alcohol (2.5 ml/kg) prior to prolonged (3 hours) exercise in the cold resulted in increased heat loss, though this effect was somewhat attenuated by co-ingestion of glucose. In animal studies, administration of alcohol to animals exposed to ambient temperatures both above and below the

thermoneutral zone has shown that alcohol acts to impair adaptation to both heat and cold (Kalant & Lê, 1983). In another animal study, rats were given 0, 4, 8, 12, or 16% ethanol as the sole source of drinking water for 14 days. Time to fatigue in treadmill running in the heat (35°C) of rats drinking 4% ethanol was similar to that of rats consuming water (32 vs. 32.9 minutes, respectively), but the running time of rats drinking 16% ethanol was reduced (Francesconi & Mager, 1981). In humans, however, Johnston et al. (1996) showed that moderate alcohol consumption (1 g/kg body mass) had no effect on the rate of fall of core temperature during immersion in cool (28°C) water. There seem to be no more recent studies in this important area. In view of the potential for adverse health consequences for athletes engaging in training and competition at climatic extremes, there does seem to be an urgent need for further studies in this area.

The Aftermath of Alcohol Use

The acute intake of large amounts of alcohol is usually followed by a period of adverse symptoms that include general misery and more specifically headache, nausea, increased sensitivity to light and noise, lethargy, sweating, and thirst. These symptoms typically appear as the intoxicating effects of alcohol begin to wear off and may last from a few hours to a day or even two. There may also be psychological symptoms including heightened feelings of depression and anxiety.

There is limited and conflicting evidence of post-alcohol/hangover effects on functional capacity, but there is sufficient evidence of adverse effects the day after a heavy drinking session for such activities to be discouraged (Barker, 2004). O'Brien (1993) showed reductions in aerobic exercise performance of rugby players the day after an evening bout of drinking involving an intake of 1–38 units of alcohol, though anaerobic performance was unaffected. The negative effect on aerobic performance was apparent at even the smallest dose of alcohol. For obvious reasons, there are few studies of the effects of high alcohol intakes on sport-specific performance, and there appear to be no studies on elite team sports players even though binge drinking is known to occur in this population. In spite of substantial efforts on the part of the alcoholic drinks industry, the causes of the symptoms of hangover are not well understood, but are thought to include dehydration, acid–base disturbances, disruption of cytokine and prostaglandin pathways, and alterations in glucose metabolism via effects on circulating insulin and glucagon levels (Verster, 2008; Wiese et al., 2000). There are also disturbances of cardiovascular function during the hangover phase, including increased heart rate, decreased left ventricular performance, and increased blood pressure (Kupari, 1983).

Effects of Alcohol on Injury and Incapacity

The ingestion of alcohol is likely to have a number of behavioral and other effects that may influence the risk of injury and the recovery process after injury. There are many reported instances of athletes in various sports competing while under the influence of alcohol, even though prior alcohol consumption appears to increase the risk of sports-related injury. According to O'Brien and Lyons (2000), the injury prevalence in football players who habitually drink alcohol is higher (55%, $p < 0.005$) than in nondrinkers (24%). The mechanisms by which this association may be mediated are not entirely clear, but the increased risk of injury and the increased severity of injuries that do occur may be a consequence of increased risk-taking behaviors, as alcohol removes some of the restraints that normally operate (O'Brien, 1993). The available evidence suggests that prior exercise does not significantly influence the metabolism of ethanol, but it may reduce the individual's perception of the level of intoxication and may also increase the willingness to undertake risky behaviors such as driving (Irwin et al., 2012). Increased levels of aggression are also often displayed by those under the influence of alcohol. Intoxication during competition is probably rather rare, at least at the higher levels of sport, though it is not completely absent. It is perhaps not so unusual, though, for

athletes training the morning after a high alcohol intake the previous night to be still under the influence of alcohol, and several high-profile players from various football codes have publicly admitted to alcohol addiction.

Athletes often experience some degree of muscle damage, of either intrinsic or extrinsic origin, after hard training or competition. This is usually in the form of minor damage that results in some degree of pain and disability that may persist for hours or days (Clarkson & Hubal, 2002). This damage results in turn in an inflammatory response that involves an increase in local blood flow and macrophage infiltration of the damaged area. Though minor, the accompanying symptoms may interfere with subsequent training or competition. Recommended treatments include application of ice, compression, and elevation of the limb to reduce blood flow. Because alcohol can act as a peripheral vasodilator, it is often stated that alcohol intake should be avoided after any exercise that may have resulted in muscle damage. There appears, however, to be limited and conflicting experimental evidence to support these anecdotal observations. In a prospective randomized trial, the effects of alcohol ingestion on exercise-induced muscle damage and soreness were investigated by Clarkson and Reichsman (1990). In one trial, female subjects ingested alcohol (0.8 g/kg) 35 minutes before performing single-arm eccentric exercise, with a nonalcoholic drink being taken on the other trial. Exercise resulted in muscle damage, as assessed by leakage of muscle-specific enzymes into the circulation, pain, loss of strength, and decreased range of motion on both trials, with no measurable effect of alcohol on any of these responses. Barnes et al. (2010) looked at the effects of ingestion of a moderate amount of alcohol (1 g/kg body mass) on performance and muscle damage after a bout of eccentric exercise. All measures of muscle performance were significantly lower at 36 and 60 hours after exercise compared to pre-exercise measures, but peak strength loss was significantly greater when alcohol was given than on the control trial. There was no effect of alcohol ingestion on post-exercise plasma creatine kinase activity or on subjective ratings of muscle soreness. The same authors (Barnes et al., 2011) have used the same experimental model to show that a smaller dose of alcohol (0.5 g/kg body mass) had no effect on the exercise-induced loss of force-generating capacity in the recovery period. More recently still, these authors (Barnes et al., 2012) showed greater decrements in maximal voluntary isometric contraction at 36 and 60 hours after muscle-damaging exercise when alcohol was taken compared to a placebo trial where orange juice was given.

Although the evidence for a negative effect of moderate alcohol intake on muscle damage and soreness is not strong, it seems prudent to avoid intoxication as this is likely to result in inappropriate behaviors that may exacerbate existing muscle damage and delay the recovery process. With more serious injury or surgical intervention that results in longer term inability to train or play, athletes may face some special problems. There may be a temptation to drink more, perhaps because of the absence of a requirement to prepare for upcoming games or because of depression at absence from the game and from the routine of training. This may result in unwanted weight gain, apart from the potential for negative effects on the repair process in muscle and other tissues.

Summary

The use of alcohol is often intimately associated with sport. As well as providing a source of energy, alcohol (ethanol) has metabolic, cardiovascular, thermoregulatory, and neuromuscular actions that may affect exercise performance. Strength seems to be little affected, and performance impairments depend on the dose of alcohol and subject habituation to alcohol intake, exercise duration, environmental conditions, and other factors. Central nervous system function is impaired at high doses, resulting in decrements in cognitive function and motor skill as well as behavioral changes that may have adverse effects on performance. Recovery may be impaired if carbohydrate and nonalcoholic fluid intake are less than optimal. Effects may persist for some hours after signs of intoxication have disappeared.

References

Bagnardi, V., Rota, M., Botteri, E., et al. (2013) Light alcohol drinking and cancer: a meta-analysis. *Annals of Oncology* 24, 301–308.

Barker, C.T. (2004) The alcohol hangover and its potential impact on the UK armed forces: a review of the literature on post-alcohol impairment. *Journal of the Royal Army Medical Corps* 150, 168–174.

Barnes, M.J., Mundel, T., & Stannard, S.R. (2010) Acute alcohol consumption aggravates the decline in muscle performance following strenuous eccentric exercise. *Journal of Science and Medicine in Sport* 13, 189–193.

Barnes, M.J., Mundel, T., & Stannard, S.R. (2011) A low dose of alcohol does not impact skeletal muscle performance after exercise-induced muscle damage. *European Journal of Applied Physiology* 111, 725–729.

Barnes, M.J., Mundel, T., & Stannard, S.R. (2012) The effects of acute alcohol consumption and eccentric muscle damage on neuromuscular function. *Applied Physiology, Nutrition, and Metabolism* 37, 63–71.

Burke, L.M. & Maughan, R.J. (2000) Alcohol in sport. In *Nutrition in Sport* (ed. R.J. Maughan), pp. 405–416. Blackwell, Oxford.

Burke, L.M., Collier, G.R., Broad, E.M., et al. (2003) Effect of alcohol intake on muscle glycogen storage after prolonged exercise. *Journal of Applied Physiology* 95, 983–990.

Clarkson, P.M. & Hubal, M.J. (2002) Exercise-induced muscle damage in humans. *American Journal of Physical Medicine and Rehabilitation* 81, S52–S59.

Clarkson, P.M. & Reichsman, F. (1990) The effect of ethanol on exercise-induced muscle damage. *Journal of Studies on Alcohol* 51, 19–23.

Desruelle, A.V., Boisvert, P., & Candas, V. (1996) Alcohol and its variable effect on human thermoregulatory response to exercise in a warm environment. *European Journal of Applied Physiology and Occupational Physiology* 74, 572–574.

Eggleton, M.G. (1942) The diuretic action of alcohol in man. *Journal of Physiology* 101, 172–191.

El-Sayed, M.S., Ali, N., & El-Sayed, A.Z. (2005) Interactions between alcohol and exercise: physiological and haematological implications. *Sports Medicine* 35, 257–269.

Ferreira, M.F. & Willoughby, D. (2008) Alcohol consumption: the good, the bad and the indifferent. *Applied Physiology, Nutrition, and Metabolism* 33, 12–20.

Francesconi, R. & Mager, M. (1981) Alcohol consumption in rats: effects on work capacity in the heat. *Journal of Applied Physiology* 50, 1006–1010.

Gomez-Cabrera, M.C., Domenech, E., Romagnoli, M., et al. (2008) Oral administration of vitamin C decreases muscle mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. *American Journal of Clinical Nutrition* 87, 142–149.

Graham, T. (1981) Alcohol ingestion and man's ability to adapt to exercise in a cold environment. *Canadian Journal of Applied Sport Science* 6, 27–31.

Gutjahr, E., Gmel, G., & Rehm, J. (2001) Relation between average alcohol consumption and disease: an overview. *European Addiction Research* 7, 117–127.

Hobson, R.M. & Maughan, R.J. (2010) Hydration status and the diuretic action of a small dose of alcohol. *Alcohol and Alcoholism* 45, 366–373.

Hopkins, W.G., Hawley, J.A, Burke, L.M., et al. (1999) Design and analysis of research on sport performance enhancement. *Medicine & Science in Sports & Exercise* 31, 472–485.

Irwin, C., Goodwin, A., Leveritt, M., et al. (2012) Alcohol pharmacokinetics and risk-taking behaviour following exercise-induced dehydration. *Pharmacology Biochemistry and Behavior* 101, 609–616.

Johnston, C.E., Bristow, G.K., Elias, D.A., et al. (1996) Alcohol lowers the vasoconstriction threshold in humans without affecting core cooling rate during mild cold exposure. *European Journal of Applied Physiology and Occupational Physiology* 74, 293–295.

Kalant, H. & Lê, A.D. (1983) Effects of ethanol on thermoregulation. *Pharmacology and Therapeutics* 23, 313–364.

Kupari, M. (1983) Drunkenness, hangover, and the heart. *Acta Medica Scandinavica* 213, 84–90.

Lisha, N.E., Martens, M., & Leventhal, A.M. (2011) Age and gender as moderators of the relationship between physical activity and alcohol use. *Addictive Behaviors* 36, 933–936.

Lorente, F.O., Peretti-Watel, P., Griffet, J., et al. (2003) Alcohol use and intoxication in sport university students. *Alcohol and Alcoholism* 38, 427–430.

Lorente, F.O., Souville, M., Griffet, J., et al. (2004) Participation in sports and alcohol consumption among French adolescents. *Addictive Behaviors* 29, 941–946.

Lourenco, S., Oliveira, A., & Lopes, C. (2012) The effect of current and lifetime alcohol consumption on overall and central obesity. *European Journal of Clinical Nutrition* 66, 813–818.

Martens, M.P., Brown, N.T., & Donovan, B.M. (2005) Measuring negative consequences of college student substance use: a psychometric evaluation of the core alcohol and drug survey. *Measurement and Evaluation in Counseling and Development* 38, 164–175.

Martens, M.P. Dams-O'Connor, K., & Beck, N.C. (2006) A systematic review of college student-athlete drinking: prevalence rates, sport-related factors, and interventions. *Journal of Substance Abuse* 31, 305–316.

Maughan, R.J. (2006) Alcohol and football. *Journal of Sports Sciences* 24, 741–748.

Moore, M.J. & Werch, C.E. (2004) Sport and physical activity participation and substance use among adolescents. *Journal of Adolescent Health* 36, 486–493.

Musselman, J.R.B. & Rutledge, P.C. (2010) The incongruous alcohol-activity association: physical activity and alcohol consumption in college students. *Psychology of Sport and Exercise* 11, 609–618.

O'Brien, C.P. (1993) Alcohol and sport: impact of social drinking on recreational and competitive sports. *Sports Medicine* 15, 71-77.

O'Brien, C.P. & Lyons, F. (2000) Alcohol and the athlete. *Sports Medicine* 29, 295–300.

O'Brien, K.S., Blackie, J.M., & Hunter, J.A. (2005) Hazardous drinking in elite New Zealand sportspeople. *Alcohol and Alcoholism* 40, 239–241.

O'Brien, K.S., Kolt, G.S., Webber, A., et al. (2010) Alcohol consumption in sport: the influence of sporting idols, friends and normative drinking practices. *Drug and Alcohol Review* 29, 676–683.

O'Brien, K.S., Miller, P.G., Kolt, G.S., et al. (2011) Alcohol industry and non-alcohol industry sponsorship of sportspeople and drinking. *Alcohol and Alcoholism* 46, 210-213.

Parra, J., Neighbors, C., Bell, R.L., et al. (2012) Does exercise increase alcohol consumption? [abstract]. *Alcoholism: Clinical and Experimental Research* 36, 135A.

Piazza-Gardner, A.K. & Barry, A.E. (2012) Cautioning against causal alcohol–cardiovascular assumptions. *Drug and Alcohol Review* 31, 198–199.

Powers, S.K. & Jackson, M.J. (2008) Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. *Physiological Reviews* 88, 1243–1276.

Reilly, T. (2003) Alcohol, anti-anxiety drugs and sport. In *Drugs in Sport*, 3rd edn (ed. D.R. Mottram), pp. 256–285. Routledge, London.

Ristow, M. & Zarse, K. (2010) How increased oxidative stress promotes longevity and metabolic health: the concept of mitochondrial hormesis (mitohormesis). *Experimental Gerontology* 45, 410–418.

Ristow, M., Zarse, K., Oberbach, A., et al. (2009) Antioxidants prevent health-promoting effects of physical exercise in humans. *Proceedings of the National Academy of Sciences of USA* 106, 8665–8670.

Ruiz-Juan, F. & Ruiz-Risueno, J. (2011) Predicting variables of alcohol consumption among Spanish adolescents. *Anales de Psicologia* 27, 350–359.

Shakespeare, W. (1623) *Macbeth, Act II, Scene iii*. Heminges and Condell, London.

Shirreffs, S.M. & Maughan, R.J. (1997) Restoration of fluid balance after exercise-induced dehydration: effects of alcohol consumption. *Journal of Applied Physiology* 83, 1152–1158.

Shirreffs, S.M. & Maughan, R.J. (2005) Water-electrolyte balance. In *Encyclopaedia of Human Nutrition*, 2nd edn (eds. B. Caballero, L. Allen, & A. Prentice), pp. 100–105. Elsevier, Oxford.

Verster, J.C. (2008) The alcohol hangover—a puzzling phenomenon. *Alcohol and Alcoholism* 43, 124–126.

Wiese, J.G., Shlipak, M.G., & Browner, W.S. (2000) The alcohol hangover. *Annals of Internal Medicine* 132, 897–902.

World Health Organization (2005) *WHO Statistical Information System (WHOSIS): Per Capita Recorded Alcohol Consumption (Litres of Pure Alcohol) among Adults (≥15 Years)*. Available at: http://www.who.int/gho/alcohol/consumption_levels/adult_recorded_percapita/en/ (accessed May 10, 2013).

Yoda, T., Crawshaw, L.I., Nakamura, M., et al. (2005) Effects of alcohol on thermoregulation during mild heat exposure in humans. *Alcohol* 36, 195–200.

PART 4

PRACTICAL ISSUES

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Chapter 28

The Female Athlete

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Introduction

In 1896, the first modern Olympic Games were held in Athens, but, as in the Games of ancient Greece, women did not compete. Since those Games, women's participation in sport at both the elite and recreational levels has increased tremendously. As of 2012, female athletes now compete in all 35 Olympic sports, and women made up 42% and 40% of participants, respectively, in the 2008 Beijing Summer Games and 2010 Vancouver/Whistler Winter Games (Olympic.org, 2012). At the recreational level, they have moved from being prohibited from competing in marathons as recently as 1980 (there was no women's marathon at the 1980 Olympics) to making up the majority of the participants in half-marathon races and close to half the participants in marathons (Running USA, 2011).

When considering the topic of nutrition for the female athlete, it is clear that there are a number of issues that are relevant, and many of these are addressed in depth elsewhere in this volume. On average, women athletes are smaller than men and have lower energy requirements. Yet, their requirements for several key nutrients are either similar to those of men (e.g., calcium—see Chapter 20) or greater (iron—see Chapter 19). As a result, dietary quality may be of particular concern to female athletes. In addition, women appear more susceptible than men to the development of eating disorders and disordered eating, both of which may affect health and performance (see Chapter 42). They are also more likely than men to adopt vegetarian diets (see Chapter 31).

Perhaps the most fundamental biological difference, however, is that during their reproductive years—which encompass the timeframe when most women athletes train and compete at the highest level of intensity—women experience the cyclic exposure to estrogen and progesterone that manifest in the menstrual cycle. Accordingly, this chapter focuses on the menstrual cycle and its implications for the nutrition, performance, and health of women athletes. First, the physiology of the menstrual cycle is described, including an overview of the clinical and subclinical disturbances of ovarian function, a discussion of premenstrual syndrome, and the impact of the menstrual cycle on iron requirements. Next, the question of whether exercise performance is affected by menstrual cycle phase is addressed. Cycle disturbances are then addressed in greater depth, including factors that may contribute to their development as well as their implications for the health of the athlete.

The Menstrual Cycle

Overview of Normal Physiology

The female human menstrual cycle represents a complex interplay of hormones (Fritz & Speroff, 1983). Figure 28.1 illustrates the ovarian histology, changes in basal body temperature, hormonal patterns, and endometrial histology of a hormonally normal cycle. During menstrual flow, the onset of

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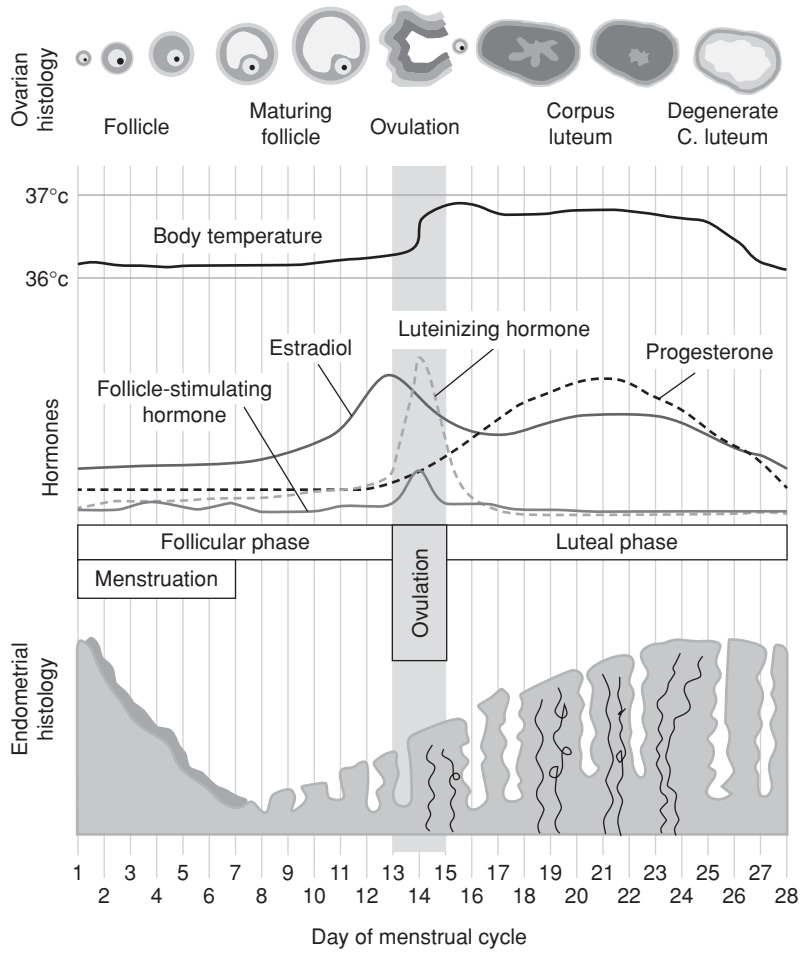

(Average values. Durations and values may differ between different females or different cycles.)

Figure 28.1 Hormonal fluctuations over the menstrual cycle. This Wikipedia and Wikimedia Commons image is from the user Chris 73 and is freely available at http://commons.wikimedia.org/wiki/Image:MenstrualCycle.png under the creative commons cc-by-sa 2.5 license.

which is used to define the first day of the cycle, circulating levels of both estradiol and progesterone are low. These low levels allow gonadotropin-releasing hormone (GnRH) from the hypothalamus to stimulate release of low levels of both follicle-stimulating hormone (FSH) and luteinizing hormone (LH) from the anterior pituitary. FSH stimulates the growth of a number of ovarian follicles, which begin to mature and secrete estrogen. Estrogen levels rise gradually during the follicular phase of the cycle, and cause thickening of the endometrial lining of the uterus. The increasing estrogen levels inhibit FSH secretion, and all but the most mature of the developing follicles undergo atresia. This dominant follicle produces large amounts of estrogen, which in turn appear to stimulate a major surge in LH. The LH peak has at least three consequences: it inhibits its estrogen production by follicular cells; initiates changes that result in the rupture of the dominant follicle and release of the ovum (ovulation); and brings about transformation of the ruptured follicle into the corpus luteum, which synthesizes both

estrogen and progesterone during the luteal phase of the cycle. If fertilization does not occur, estrogen and progesterone levels begin to fall and the corpus luteum undergoes luteolysis 10–16 days after its formation. The decline in ovarian hormones results in the shedding of the thickened endometrium as menstrual flow, beginning the next cycle. The characteristics of the cycle are such that it averages 28 days in length, with a normal range from 21 to 35 days.

Overview of Clinical and Subclinical Ovulatory Disturbances

The normal ovulatory cycle described above does not occur without fail between menarche and menopause. Deviations are more common during the periods following menarche and preceding menopause (Vollman, 1977), but alterations of cycle length and/or cycle characteristics can occur at any age in response to physiological or psychosocial stressors. Disorders of cycle length include secondary amenorrhea (absence of flow for at least 6 months in a nonpregnant woman) and oligomenorrhea (irregular cycles of 36–180 days). Disturbances of cycle characteristics include anovulation and luteal phase disturbances. Anovulation frequently occurs in women with oligomenorrhea, but may also occur in cycles of normal length. Finally, disturbances of the luteal phase include short luteal phase cycles (cycles in which the luteal phase lasts less than ~10 days), and luteal phase defects, in which luteal phase length may be normal but progesterone secretion is subnormal.

Amenorrhea and oligomenorrhea are apparent to women because of the absence or irregularity of menstrual flow. It is often assumed that a normal-length cycle reflects normal ovarian function, but this is not the case as normal-length cycles may also be anovulatory or have a short luteal phase. These subclinical disturbances of ovarian function are not recognized by women, and can be detected only if cycles are assessed by measuring hormone levels in blood, urine, or saliva, or by using methods such as quantitative analysis of basal body temperature. Additional information on the etiology and health implications of these cycle disturbances is presented later in this chapter.

Premenstrual Syndrome

Most women report experiencing one or more emotional or physical symptoms during the days preceding the onset of menses. In many women, these symptoms are mild and not troubling, but it has been estimated that about 15% meet the clinical criteria for premenstrual syndrome (PMS). According to the American College of Obstetricians and Gynecologists (2000), PMS is said to exist when a woman reports at least one affective symptom (e.g., depression, anger, irritability) and at least one somatic symptom (e.g., breast tenderness, bloating, peripheral edema) during the 5 days before the onset of menses. In addition, symptoms must be absent between days 4 and 13 of the cycle; occur reproducibly in two cycles of prospective recording; not be due to any drugs, endogenous hormones, or alcohol; and contribute to identifiable dysfunction. A smaller proportion, perhaps 5%, meets the criteria for premenstrual dysphoric disorder, a more severe condition.

Whether women athletes are less likely to experience PMS than normally active women is not clearly established. Daley (2009) reviewed the available literature and reported that many women use exercise to manage their symptoms, and that physicians frequently recommend exercise for women experiencing PMS. A number of cross-sectional studies also found that symptoms were less pronounced in active women, although this was not entirely consistent. Only four intervention trials were identified, and while they all reported a reduction in symptoms after exercise interventions, the sample sizes were small and the methodological quality was low. Daley (2009) concluded that more high-quality research is required to make evidence-based recommendations regarding exercise and PMS.

The other relevant issue for women with PMS is whether symptom occurrence and severity can be reduced by dietary means. In 2000, the American College of Obstetricians and Gynecologists reviewed information on popular nutrition-related approaches to PMS management. They concluded that there was consistent scientific evidence that evening primrose oil was not useful in

PMS management, and that vitamin B6 may have minimal effectiveness. Limited or inconsistent evidence was available regarding the potential of carbohydrate-rich foods to improve mood symptoms, and for the effectiveness of calcium supplementation. With regard to calcium, three randomized trials of calcium supplements demonstrated significantly greater reductions in symptoms with calcium supplements of ~1000 to 1200 mg/day than with placebo. More recently, higher intakes of calcium and vitamin D were associated with a lower risk of developing PMS symptoms in a nested case-control study of women who were initially symptom free (Bertone-Johnson et al., 2005). A similar study found that higher dietary intakes of thiamin and riboflavin were also associated with lower PMS risk, but no associations were observed with niacin, folate, vitamin B6, or vitamin B12 (Chocano-Bedoya et al., 2011).

In summary, the prevalence of PMS and the intensity of its symptoms may be lower in exercising versus sedentary women. Among those who experience PMS symptoms, diets rich in calcium and vitamin D may reduce symptom severity.

Direct Implications of the Menstrual Cycle for Women's Nutrition

Because of the loss of iron in menstrual flow, the iron requirements of reproductive-aged women are considerably greater than those of men or nonmenstruating women (Institute of Medicine, 2001). Menstrual iron losses can be estimated from the amount of blood lost per menstrual cycle (averaged over 28 days), the blood hemoglobin concentration, and the iron content of hemoglobin. Although losses appear to be quite consistent within an individual from one cycle to the next, they are highly variable among women. The distribution of losses is also right-skewed: it is 0.14 mg/day at the 5th percentile, 0.51 mg/day at the 50th percentile, and 2.32 mg/day at the 97.5th percentile. While most women have menstrual iron losses considerably below the 97.5th percentile, there is no easy or accurate way for an individual woman to determine whether her menstrual losses are light, average, or heavy. When combined with basal iron losses (in urine, skin, and through the gastrointestinal tract) and after accounting for absorption, the median requirement for dietary iron is 8 mg/day, but the Recommended Dietary Allowance (RDA, designed to meet or exceed the needs of almost all women, and therefore set at the 97.5th percentile) is much higher at 18 mg/day. This can be challenging for many women to obtain through the diet, as the iron content of the food supply averages about 6 mg/1000 kcal.

Iron requirements are also influenced by method of contraception, vegetarianism, and intense endurance training (Institute of Medicine, 2001). Oral contraceptives typically reduce menstrual blood losses by about 60%, meaning that the "RDA" for women who use oral contraceptives would be 11 mg/day rather than 18 mg/day. Conversely, certain intrauterine devices increase menstrual blood loss and therefore requirements. Because iron bioavailability is lower from plant-based foods than from meat, the bioavailability of iron in vegetarian diets is estimated to be about 10%, rather than about 18% from a mixed omnivorous diet. Thus, iron requirements for vegetarians are about 1.8 times higher than for omnivores. Finally, increased fecal losses and sporadic hematuria have been reported in endurance athletes (especially runners), leading to estimates that iron requirements of endurance athletes may be 30–70% higher than those of normally active individuals.

Because of the limited iron content in the food supply, the many variables that affect iron requirements, and the centrality of this nutrient to athletic performance, iron is a key nutrient for women athletes. Regular monitoring of their iron status with attention to diet and—as warranted—supplements, is therefore recommended. Chapter 19 provides an in-depth review of iron.

Does Menstrual Cycle Phase Affect Exercise Performance?

Estrogen and progesterone, the two main reproductive hormones in women, function primarily in the regulation of fertility and reproduction. They also have effects on a wide variety of cardiovascular, respiratory, and metabolic parameters. Some of these

effects, which have been reviewed elsewhere (Constantini et al., 2005; Oosthuyse & Bosch, 2010), have the potential to affect athletic performance. To the extent that the effects of estrogen and progesterone differ—and may be additive or antagonistic—performance could be differentially influenced in the follicular phase (when low levels of both hormones are present) versus the time around ovulation (when estrogen is high and progesterone is low) versus the luteal phase (when both estrogen and progesterone are high). For example, progesterone leads to increased ventilation during the luteal phase. It also affects thermoregulation, such that body temperature is about 0.3–0.5° C higher during the luteal phase, which could theoretically affect exercise tolerance at high environmental temperatures. Estrogen can influence the cardiovascular system and also substrate metabolism. It appears to spare glycogen stores by increasing free fatty acid metabolism, which could have implications for endurance performance.

While the effects on physiology and metabolism are well documented, the more relevant question for athletes is whether performance is influenced under "real-life" conditions. A number of review articles have concluded that menstrual cycle phase has little effect on exercise performance, although the available data are not entirely consistent (Constantini et al., 2005; Frankovich & Lebrun, 2000). Some of the discrepancies may be because the studies reviewed included both trained and untrained women, and that the menstrual cycle phase was not always documented or confirmed physiologically. While a comprehensive critique of studies that have examined aerobic, anaerobic, and strength performance is beyond the scope of this chapter, Table 28.1 presents data on studies that have assessed whether aerobic exercise performance differs by menstrual cycle phase in exercise-trained women. The rationale for focusing on aerobic exercise is that more research has been done in this area, and also that dietary manipulations are more likely to affect the performance of aerobic and endurance activity. All of the studies included in Table 28.1 had a number of strengths: they included trained subjects, confirmed whether the days of testing actually corresponded to the intended phase of the cycle (follicular versus luteal), and, in most cases, controlled for order effects by balancing the number of participants who had their first test during the follicular and luteal phases.

The primary limitation of these studies was the small number of participants, and the fact that, in most cases, power calculations were not provided. An exception was the study of Burrows and Bird (2005), and their power calculations provide insight into the challenges of conducting research in this area. These authors assessed the velocity at VO_2 max and peak treadmill velocity in 10 highly trained runners on 2 days within each of the follicular and luteal phases, as well as during two consecutive menses. They found no significant differences within or between the menstrual, follicular, and luteal phases of the cycle for either velocity at VO_2 max ($F_{5,35} = 0.72$, $p = 0.611$) or peak treadmill velocity ($F_{5,35} = 0.93$, $p = 0.472$). Based on their data, they estimated that 64 participants would be required to detect a difference in running speed of 0.5 km/h with power of 0.80 and $\alpha = 0.05$. For elite women runners running 17.5 versus 18.0 km/h, that translates to a difference in 10 km running time of 34.3 versus 33.3 minutes. Clearly, races are won and lost by far less than this margin; however, detecting a smaller difference in running velocity would require an even greater number of subjects.

Despite the inadequate power of the available studies, their results are largely consistent. Nine of the 12 studies reported no significant differences by cycle phase (and no consistent tendencies for insignificant differences). Among the other three studies, one (Nicklas et al., 1989) reported that time to exhaustion at 70% VO_2 max tended to be better during the luteal phase than the follicular phase, although this did not reach statistical significance ($p < 0.07$). In contrast, (Campbell et al., 2001) reported that time-trial performance following 2 hours of exercise at 70% VO_2 max was better during the follicular phase than the luteal phase when exercise was done in the fasted state and no carbohydrate was provided during exercise. However, when carbohydrate was provided during exercise (even though the exercise began after an overnight fast), no differences were detected. Finally, Lebrun et al. (1995) reported that VO_2 max was lower in the

Table 28.1 Aerobic exercise performance across the menstrual cycle in trained women

Study	Subjects	*n*	Exercise test	Cycle phases	Fed or fasted	Outcomes	Results
Vaiksaar et al. (2011a)	Competitive rowers Recreational rowers	9 8	Incremental test on rowing ergometer	MF (day 8) vs. ML (day 20); order balanced	~2 h post-meal	Max power, VO_2, peak lactate, RER	Competitive rowers had higher max power and VO_2 than recreational rowers; no differences by cycle phase
Vaiksaar et al. (2011b)	Competitive rowers	11	1 h rowing ergometer at 70% VO_2 max	MF (day 9) vs. ML (day 20); order balanced	~2 h post-meal	VO_2, lactate, RER, heart rate, CHO vs. lipid use	No differences by cycle phase
McLay et al. (2007)	Trained athletes	9	75 min intermittent cycle ergometer protocol; followed by 16 km TT	MF vs. ML on both normal diet and after 3-day CHO loading; order balanced by cycle phase and diet	1.5–2 h post-standard breakfast	Time to complete 16 km cycling TT	Menstrual cycle phase had no effect on TT performance during either normal diet or with CHO loading
Burrows and Bird (2005)	Trained runners	10	Incremental treadmill test	Menses 1, EF, LF, EL, LL, menses 2; order balanced	>4 h post-meal	Velocity at VO_2 max; peak treadmill velocity	No differences by cycle phase
Oosthuyse et al. (2005)	Trained cyclists	5	30 km cycle ergometer TT	EF, LF (~2 days before LH surge), ML; order balanced	Not specified but time of day kept constant	Time to complete TT; also heart rate, RER, % VO_2 max	No significant differences
Campbell et al.(2001)	Endurance trained	8	2 h on cycle ergometer trial at 70% VO_2 max; followed by 4 kJ/kg TT	Follicular vs. luteal each with 6% CHO (~1 g/kg/h) vs. placebo; order randomized	Fasted (overnight)	Time to complete 4 kJ/kg cycle TT	TT performance was faster during follicular vs. luteal phase with placebo (24.5 ± 5.9 vs. 28.4 ± 9 min, $p < 0.05$), but not with CHO (19.9 ± 2.4 vs. 20.9 ± 2.6 min; NS)
Bailey et al. (2000)	Moderately trained cyclists	9	Cycle ergometer trial at 70% VO_2 max	EF (days 1–8) vs. ML (days 19–24) each with 0.6 g/kg/h CHO vs. placebo; latin square design	3 h post-standard breakfast	Time to exhaustion	No effect of menstrual cycle phase on time to exhaustion (providing CHO vs. placebo increased performance in both menstrual phases)

Study	Subjects	*n*	Exercise test	Cycle phases	Fed or fasted	Outcomes	Results
Lebrun et al. (1995)	Trained athletes	16	Treadmill run at 90% VO_2 max	EF (day 6) vs. ML (day 23); order random	Fasted (overnight)	Time to exhaustion; VO_2; RER	Lower VO_2 max in ML (3.13 vs. 3.19 l/min in EF); no difference in RER or time to exhaustion
De Souza et al. (1990)	Trained runners	8	Incremental treadmill test; 40 min at 80% VO_2 max	EF (2–4 days after onset of menses) vs. ML (6–8 days after LH surge)	Fasted (>6 h post-meal)	VO_2 max and submax; time to exhaustion; RER, RPE, lactate	No differences by cycle phase in either maximal or submaximal exercise
Nicklas et al. (1989)	Moderately trained	6	Cycle ergometer trial at 70% VO_2 max	MF (days 7–8) vs. ML (7–8 days after ovulation); 4 of 6 did ML trial first	Not specified	Time to exhaustion; also VO_2, RPE	Time to exhaustion tended to increase in ML vs. MF (139.2 ± 14.9 vs. 126.5 ± 17.5 min, $p < 0.07$); no difference in VO_2 or RPE
Stephenson et al. (1982)	Active to competitive runners	6	5 min at each of 4 submaximal cycle ergometer workloads, followed by maximal load	Cycle days 2, 8, 14, 20, and 26; each subject began on a different day	Fasted (overnight)	VO_2 at submaximal workloads; time to exhaustion at max load	No differences relative to cycle day
Schoene et al. (1981)	Highly trained runners	6	Incremental cycle ergometer test	MF (6–10 day after onset of menses) vs. ML (4–9 days post-ovulation); order balanced	Fasted (overnight)	VO_2 max and time to exhaustion	Minute ventilation increased during the luteal phase, but no differences in VO_2 max or time to exhaustion

MF, mid-follicular phase; ML, mid-luteal phase; RER, respiratory exchange ratio; CHO, carbohydrate; TT, time trial; EF, early follicular phase; LF, late follicular phase; EL, early luteal phase; LL, late luteal phase; LH, luteinizing hormone; NS, not significant; RPE, rating of perceived exertion.

luteal phase than the follicular phase (although time to exhaustion was not different).

Thus, the available evidence does not suggest the aerobic exercise performance is influenced by menstrual cycle phase, particularly if current recommendations for pre-exercise and during-exercise nutrition are followed (see Chapters 8 and 9).

Does Exercise Cause Menstrual Cycle Disturbances?

The prevalence of menstrual disorders in athletes varies widely, but both clinical and subclinical disturbances are often reported as being higher than among the general population, especially in sports

that emphasize leanness (Nattiv et al., 2007). The question of whether exercise, *per se*, is a direct cause of menstrual disturbances in women athletes was initially examined in a number of exercise training studies conducted with untrained women. In general, when the volume and intensity of training increased abruptly and energy balance was not maintained, cycle disturbances were prevalent (e.g., Bullen et al., 1985), whereas when training was increased gradually and energy balance was maintained, they were much less common (e.g., Rogol et al., 1992).

Role of Energy Availability

The early work referred to above led to an examination of the role of adequate energy availability (defined as energy intake minus the energy expended in exercise), which is discussed in depth elsewhere in this volume (see Chapter 5). Briefly, young women assigned to even short periods (4 days) of severe energy deprivation (~10 kcal/kg lean body mass), either with or without exercise, experienced alterations in LH pulsatility that have the potential to disrupt the normal menstrual cycle. In contrast, under conditions of energy balance (~45 kcal/kg lean body mass), again achieved either with or without exercise, LH pulsatility remained normal. Subsequent studies revealed that these changes were more likely to occur when energy availability was in the range of 20 kcal/kg lean mass or lower. However, among gynecologically mature women (14 years of more after the onset of menstrual cycles) LH pulsatility was unaffected even by very low energy availability (10 kcal/kg lean mass).

This work demonstrated that exercise *per se* does not cause menstrual cycle disturbances, but that very low energy availability (induced by either dietary restriction and/or increased activity) can. Clearly, extremely low energy availability should be avoided, particularly among gynecologically immature female athletes. In this regard, it is not sufficient to simply assess energy intake, as athletes' energy expenditure can vary widely. A given energy intake could be inadequate, appropriate, or even excessive, depending on the athlete's body size and training volume.

While very low energy availability has the potential to disrupt menstrual function, particularly in younger women, it is also important to examine whether other factors contribute to menstrual disturbances. Although low energy availability is frequently reported in studies of women athletes (Nattiv et al., 2007), in many cases there are no differences between athletes with normal cycles and those with cycle disturbances. Moreover, studies conducted using doubly labeled water have raised questions about the plausibility of the athletes' reportedly low energy intakes. Doubly labeled water permits an objective assessment of energy expenditure over a period of time, and, under conditions of energy balance, also reflects energy intake. Hill and Davies (2001) reviewed the literature assessing the accuracy of self-reported energy intake as determined by doubly labeled water. Among other conclusions, they noted that underreporting was very common in women athletes (reported intakes averaged 13–43% below actual intakes), that exercise training may increase its magnitude, that it is more prevalent among groups in which a lean body build is considered desirable, and that it is also more common among those with high levels of dietary restraint. These findings are noteworthy, given that they describe many women athletes, and that another body of research has linked high levels of cognitive dietary restraint with disturbances in menstrual function.

Cognitive Dietary Restraint and Menstrual Disturbances

Cognitive dietary restraint reflects the *perception* that one is continuously monitoring and attempting to limit food intake in an effort to achieve or maintain a desirable body weight (Stunkard & Messick, 1985). The perceptual nature of dietary restraint is important to emphasize, as in many cases energy intake and body composition of women with low and high restraint scores are similar. Women with high restraint may *report* slightly lower energy intakes, but as indicated above, this may reflect an increased tendency to underreport, rather than a true biological difference. And in some studies, those with higher levels of restraint have reported

slightly higher energy intakes (Bedford et al., 2010). Yet women with high scores for cognitive dietary restraint have consistently been found to have more menstrual cycle disturbances, including irregular cycles, anovulatory cycles, and cycles with a short luteal phase (Table 28.2). Most studies have been conducted with normally active women, but some studies have also included athletes.

The biological mechanism underlying the higher frequency of ovulatory disturbances among these normal-weight women with high levels of dietary restraint may be related to the physiological stress response (Barr, 2004). It is generally accepted that stress-induced activation of the hypothalamic-pituitary-adrenal (HPA) axis can also disrupt the hypothalamic-pituitary-gonadal (HPG) axis, leading to ovarian disturbances (Meczekalski et al., 2008). Constant monitoring and mental efforts to control food intake (which ultimately may not result in lower intakes) could represent a subtle but frequently experienced stressor in women, and in support of this, higher levels of the stress hormone, cortisol have been reported in women with high levels of dietary restraint (Barr, 2004). This possible mechanism for an increased prevalence of menstrual disturbances is clearly not specific to dietary restraint (it can

Table 28.2 Associations between cognitive dietary restraint and menstrual disturbances

Author	Subjects	Results
Bedford et al. (2010)	123 nonobese women with TFEQ-R above ($n = 60$) or below ($n = 63$) the median	• High and low restraint groups similar in physical activity, but high restraint group had slightly higher BMI (22 vs. 21 kg/m^2) and reported energy intake • High restraint group had more cycles with subclinical disturbances (56% vs. 34%)
Vescovi et al. (2008)	Women with normal ($n = 46$) vs. high ($n = 38$) TFEQ-R	• Normal and high restraint groups were similar in age, BMI, physical activity, and resting energy expenditure • Prevalence of oligo/amenorrhea was higher in high restraint group (50% vs. 26%)
McLean and Barr (2003)	424 university women, nonusers of oral contraceptives	• Women with high, medium, or low restraint scores similar in age and BMI • Irregular cycles more common in high restraint group (34%) vs. low (18%) or medium (17%) restraint groups
Lebenstedt et al. (1999)	Weight-stable women athletes who were normally ovulatory ($n = 21$) or had subclinical ovarian disturbances ($n = 12$)	• Normal and ovarian disturbances groups similar in age, BMI, sport activity, and reported dietary intake • Ovarian disturbances group had higher TFEQ-R scores
Barr et al. (1994a)	27 women with a wide range of activity; compared upper ($n = 9$) vs. lower ($n = 9$) tertiles for TFEQ-R	• Women in upper and lower tertiles had similar age, BMI, % body fat, and physical activity • Women with high restraint had shorter luteal phase length
Barr et al. (1994b)	Healthy vegetarian ($n = 23$) and omnivorous ($n = 22$) weight-stable women	• Vegetarians had slightly lower BMI, but had lower restraint scores and longer luteal phase lengths than omnivores • When all subjects were pooled, those with high restraint scores had fewer ovulatory cycles and a shorter luteal phase length than those below the median
Schweiger et al. (1992)	22 young German women with TFEQ-R scores <50th percentile ($n = 13$) or >75th percentile ($n = 9$)	• High and low restraint similar in age, BMI, and activity level • High restraint had shorter luteal phase length and cycle length and lower progesterone

TFEQ-R, Three-Factor Eating Questionnaire Restraint subscale score; BMI, body mass index.

occur with all stressors) nor is it specific to exercising women. However, given the pervasive concern among women about body image, and the fact that these concerns may be stronger in certain groups of women athletes, it is clear that they may contribute to the menstrual disturbances in athletic women.

Polycystic Ovarian Syndrome and Menstrual Disturbances

In addition to severely limited energy availability and psychosocial stress, menstrual cycle function can be disrupted in women with polycystic ovarian syndrome (PCOS). This condition is characterized by oligo/amenorrhea and anovulatory cycles, androgen excess, and insulin resistance, and is much more common in overweight and obese women than in lean women (Teede et al., 2010). Up to 80% of women with PCOS have oligomenorrhea or amenorrhea, and at the population level, the majority of women with oligomenorrhea will have PCOS rather than hypothalamic dysfunction resulting from limited energy availability or psychosocial stress. Because of the key role of insulin resistance and obesity in PCOS, it appears to be relatively uncommon among women athletes, but should also be considered as a possible cause of menstrual dysfunction in this group (Awdishu et al., 2009). It may even confer some athletic advantages: in one small study of athletes with oligo/amenorrhea, the hyperandrogenic subgroup (about half of whom had polycystic ovaries) had a more anabolic body composition and the highest VO_2 max and physical performance (Rickenlund et al., 2003). Furthermore, the suggestion has been made that exercise may protect these individuals from the typical PCOS phenotype. It does not appear that exercise plays a causal role in the menstrual irregularities associated with PCOS.

Health Effects of Subclinical Menstrual Disturbances and Implications for Athletes

It is clear that overt ovarian disturbances such as hypothalamic amenorrhea have detrimental effects on bone in young women (e.g., Gordon & Nelson, 2003), but whether subclinical ovarian disturbances are associated with lower bone mineral density or increased bone loss has been controversial. Prior et al. (1990) were the first to examine this, and reported findings from 66 regularly menstruating women whose cycles were monitored over 1 year. Those who experienced subclinical disturbances, especially anovulatory cycles, had significant decreases in spinal bone density over the year. Two later studies failed to detect an association, but in one (De Souza et al., 1997), only three cycles were monitored and only a single cross-sectional measurement of bone density was obtained. In the other, no associations were detected between subclinical disturbances (based on an average of four monitored menstrual cycles) and change in bone measured over 17.5 months (Waller et al., 1996). However, in that study, the women with subclinical disturbances were significantly heavier; although BMI was not reported, mean body weight of those with luteal phase abnormalities was 82.5 kg (assuming average height, a BMI of about 30 kg/m^2). Heavier women are at a much greater risk of PCOS and thus of related menstrual dysfunction. However, likely due to the accompanying androgen excess and higher body weight, they generally have *increased* bone density (Kassanos et al., 2010). Subsequently, two larger prospective studies, both of which monitored a greater number of cycles, have confirmed that more negative changes in bone density occur in normal-weight women who experience a higher prevalence of subclinical disturbances (Bedford et al., 2010; Waugh et al., 2007).

Loss of bone mineral density likely predisposes women athletes to stress fractures (Lauder et al., 2000), and also places them at risk for developing osteoporosis in later life. Accordingly, disturbances in menstrual function in women athletes, whether profound or subtle, should be viewed as a sign that the athlete is not adapting well to her training program and/or is experiencing stress in other areas of her life. Possible contributing factors should be examined and steps taken to address them.

Summary and Conclusions

- The female human menstrual cycle reflects a complex interplay of hormones. Disturbances can be clinical (absence of cycles or irregular cycles of

longer than normal length) or subclinical and thus not apparent to women who experience them (normal length cycles in which ovulation does not occur or with an abnormal luteal phase).
- Some women experience premenstrual syndrome (PMS) in the days before the onset of menses. PMS prevalence and severity may be lower in exercising versus sedentary women, and a diet rich in calcium may reduce symptom severity.
- Menstrual blood loss increases women's iron requirements. Because of the many factors that influence iron requirements, its limited occurrence in the food supply, and its potential to affect athletic performance, monitoring the iron status of women athletes is warranted.
- Although most studies have inadequate statistical power, the available evidence indicates that athletic performance does not differ meaningfully in different phases of the menstrual cycle, particularly if current nutrition recommendations are followed.
- While exercise *per se* does not disrupt menstrual function, extremely low energy availability, excessive concern about food intake and body weight, or other psychosocial stresses can contribute to menstrual cycle abnormalities in women athletes. Whether clinical or subclinical, these disturbances can lead to bone loss.
- Maintenance of normal menstrual function should be a goal for the female athlete.

References

American College of Obstetricians and Gynecologists (2000) Premenstrual syndrome: clinical management guidelines for obstetrician-gynecologists. *American College of Obstretrics and Gynecologists Practice Bulletin* 15, 1–9.

Awdishu, S., Williams, N.I., Laredo, S.E., et al. (2009) Oligomenorrhoea in exercising women. A polycystic ovarian syndrome phenotype or distinct entity? *Sports Medicine* 39, 1055–1069.

Bailey, S.P., Zacher, C.M., & Mittleman, K.D. (2000) Effect of menstrual cycle phase on carbohydrate supplementation during prolonged exercise to fatigue. *Journal of Applied Physiology* 88, 690–697.

Barr, S.I. (2004) Cognitive dietary restraint, cortisol and bone density in normal-weight women: is there a relationship? In *Nutritional Aspects of Osteoporosis*, 2nd edn (eds. P. Burckhardt, B. Dawson-Hughes, & R.P. Heaney), pp. 165–177. Elsevier Academic Press, Amsterdam, The Netherlands.

Barr, S.I., Prior, J.C., & Vigna, Y.M. (1994a) Restrained eating and ovulatory disturbances: possible implications for bone health. *American Journal of Clinical Nutrition* 59, 92–97.

Barr, S.I., Janelle, K.C., & Prior, J.C. (1994b) Vegetarian vs. nonvegetarian diets, dietary restraint, and subclinical ovulatory disturbances: prospective 6-mo study. *American Journal of Clinical Nutrition* 60, 887–894.

Bedford, J.L., Prior, J.C., & Barr, S.I. (2010) A prospective exploration of cognitive dietary restraint, subclinical ovulatory disturbances, cortisol, and change in bone density over two years in healthy young women. *Journal of Clinical Endocrinology and Metabolism* 95, 3291–3299.

Bertone-Johnson, E.R., Hankinson, S.E., Bendich, A., et al. (2005) Calcium and vitamin D intake and risk of incident premenstrual syndrome. *Archives of Internal Medicine* 165, 1246–1252.

Bullen, B.A., Skrinar, G.S., Beitins, A.Z., et al. (1985) Introduction of menstrual cycle disturbances by strenuous exercise in untrained women. *New England Journal of Medicine* 312, 1349–1353.

Burrows, M. & Bird, S.R. (2005) Velocity at VO_2 max and peak treadmill velocity are not influenced within or across the phases of the menstrual cycle. *European Journal of Applied Physiology* 93, 575–580.

Campbell, S.E., Angus, D.J., & Febbraio, M.A. (2001) Glucose kinetics and exercise performance during phases of the menstrual cycle: effect of glucose ingestion. *American Journal of Physiology: Endocrinology & Metabolism* 281, E817–E825.

Chocano-Bedoya, P.O., Manson, J.E., Hankinson, S.E., et al. (2011) Dietary B vitamin intake and incident premenstrual syndrome. *American Journal of Clinical Nutrition* 93, 1080–1086.

Constantini, N.W., Dubnov, G., & Lebrun, C.M. (2005) The menstrual cycle and sport performance. *Clinics in Sports Medicine* 24, e51–e82.

Daley, A. (2009) Exercise and premenstrual symptomatology: a comprehensive review. *Journal of Women's Health* 18, 895–899.

De Souza, M.J., Maguire, M.S., Rubin, K.R., et al. (1990) Effects of menstrual phase and amenorrhea on exercise performance in runners. *Medicine & Science in Sports & Exercise* 22, 575–580.

De Souza, M.J., Miller, B.E., Sequenzia, L.C., et al. (1997) Bone health is not affected by luteal phase abnormalities and decreased ovarian progesterone production in female runners. *Journal of Clinical Endocrinology and Metabolism* 82, 2867–2876.

Frankovich, R.J. & Lebrun, C.M. (2000) Menstrual cycle, contraception, and performance. *Clinics in Sports Medicine* 19, 251–271.

Fritz, M.C. & Speroff, L. (1983) Current concepts of the endocrine characteristics of normal menstrual function: the key to diagnosis and management of menstrual disorders. *Clinical Obstetrics and Gynecology* 26, 647–689.

Gordon, C.M. & Nelson, L.M. (2003) Amenorrhea and bone health in adolescents and young women. *Current Opinions in Obstetrics and Gynecology* 15, 377–384.

Hill, R.J. & Davies, P.S.W. (2001) The validity of self-reported energy intake as determined using the doubly labeled water technique. *British Journal of Nutrition* 85, 415–430.

Institute of Medicine (2001) *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel,*

Silicon, Vanadium, and Zinc. National Academies Press, Washington, DC.
Kassanos, D., Trakakis, E., Baltas, C.S., et al. (2010) Augmentation of cortical bone mineral density in women with polycystic ovary syndrome: a peripheral quantitative computed tomography (pQCT) study. *Human Reproduction* 25, 2107–2104.
Lauder, T.D., Dixit, S., Pezzin, L.E., et al. (2000) The relation between stress fractures and bone mineral density: evidence from active-duty army women. *Archives of Physical Medicine & Rehabilitation*, 81, 73–79.
Lebenstedt, M., Platte, P., & Pirke, K.M. (1999) Reduced resting metabolic rate in athletes with menstrual disorders. *Medicine & Science in Sports & Exercise* 31, 1250–1256.
Lebrun, C.M., McKenzie, D.C., Prior, J.C., et al. (1995) Effects of menstrual cycle phase on athletic performance. *Medicine & Science in Sports & Exercise* 27, 437–444.
McLay, R.T., Thomson, C.D., Williams, S.M., et al. (2007) Carbohydrate loading and female endurance athletes: effect of menstrual-cycle phase. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 189–205.
McLean, J.A. & Barr, S.I. (2003) Cognitive dietary restraint is associated with eating behaviors, lifestyle practices, personality characteristics and menstrual irregularity in college women. *Appetite*, 40, 185–192.
Meczekalski, B., Podfigurna-Stopa, A., Warenik-Szymankiewicz, A., et al. (2008) Functional hypothalamic amenorrhea: current view on neuroendocrine aberrations. *Gynecological Endocrinology* 24, 4–11.
Nattiv, A., Loucks, A.B., Manore, M.M., et al. (2007) American College of Sports Medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise* 39, 1867–1882.
Nicklas, B.J., Hackney, A.C., & Sharp, R.L. (1989) The menstrual cycle and exercise: performance, muscle glycogen, and substrate responses. *International Journal of Sports Medicine* 10, 264–269.
Olympic.org (2012) *Official Website of the Olympic Movement*, *Sports*. Available at: http://www.olympic.org/sports (accessed February 2, 2012).
Oosthuyse, T. & Bosch, A.N. (2010) The effect of the menstrual cycle on exercise metabolism. Implications for exercise performance in eumenorrhoeic women. *Sports Medicine* 40, 207–227.
Oosthuyse, T., Bosch, A.N., & Jackson, S. (2005) Cycling time trial performance during different phases of the menstrual cycle. *European Journal of Applied Physiology* 94, 268–276.
Prior, J.C., Vigna, Y.M., Schechter, M.T., et al. (1990) Spinal bone loss and ovulatory disturbances. *New England Journal of Medicine* 323, 1221–1227.
Rickenlund, A., Carolstrom, K., Ekblom B., et al. (2003) Hyperandrogenicity is an alternative mechanism underlying oligomenorrhea or amenorrhea in female athletes and may improve athletic performance. *Fertility and Sterility* 79, 947–955.
Rogol, A.D., Weltman, A., Weltman, J.Y., et al. (1992) Durability of the reproductive axis in eumenorrheic women during 1 yr of endurance training. *Journal of Applied Physiology* 72, 1571–1580.
Running USA (2011) *2011 Marathon, Half-Marathon and State of the Sport Reports*. Available at: http://www.runningusa.org/node/76115#76116 (accessed November 21, 2011).
Schoene, R.B., Robertson, H.T., Pierson, D.J., et al. (1981) Respiratory drives and exercise in menstrual cycles of athletic and nonathletic women. *Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology* 50, 1300–1305.
Schweiger, U., Tuschl, R.J., Platte, P., et al. (1992) Everyday eating behavior and menstrual function in young women. *Fertility and Sterility* 57, 771–775.
Stephenson, L.A., Kolka, M.A., & Wilkerson, J.E. (1982) Metabolic and thermoregulatory responses to exercise during the human menstrual cycle. *Medicine & Science in Sports & Exercise* 14, 270–275.
Stunkard, A.J. & Messick, S. (1985) The Three-Factor Eating Questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of Psychosomatic Research* 29, 71–83.
Teede, H., Deeks, A., & Moran, L. (2010) Polycstic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. *BMC Medicine*, 8, 41. doi:10.1186/1741-7015-8-41
Vaiksaar, S., Jurimae, J., Maestu, J., et al. (2011a) No effect of menstrual cycle phase on fuel oxidation during exercise in rowers. *European Journal of Applied Physiology* 111, 1027–1034.
Vaiksaar, S., Jurimae, J., Maestu, J., et al. (2011b) No effect of menstrual cycle phase and oral contraceptive use on endurance performance in rowers. *Journal of Strength and Conditioning Research* 25, 1571–1578.
Vescovi, J.D., Scheid, J.L., Hontscharuk, R., et al. (2008) Cognitive dietary restraint: impact on bone, menstrual and metabolic status in young women. *Physiology and Behavior* 95, 48–55.
Vollman, R.F. (1977) *The Menstrual Cycle*. W.B. Saunders Company, Philadelphia, PA.
Waller, K., Reim, J., Fenster, L., et al. (1996) Bone mass and subtle abnormalities in ovulatory function in healthy women. *Journal of Clinical Endocrinology and Metabolism* 81, 663–668.
Waugh, E.J., Polivy, J., Ridout, R., et al. (2007) A prospective investigation of the relationships among cognitive dietary restraint, subclinical ovulatory disturbances, physical activity, and bone mass in healthy young women. *American Journal of Clinical Nutrition* 86, 1791–1801.

Chapter 29

The Young Athlete

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Introduction

Many children and adolescents engage in competitive sports that involve long and intense practices as well as demand competitive schedules. Training and games may be frequent, which does not allow sufficient time for recovery. Adequate nutrition is a key factor in ensuring optimum performance, recovery, and growth of the young athlete and can aid in preventing injuries and improving overall health.

The nutritional approach to young athletes has the unique challenge of dealing with a growing individual. In addition, the young athlete has other physiological and metabolic characteristics that distinguish them from adults. These factors may interact with their overall nutritional needs for energy, macro and micronutrients, and fluids. From a practical angle, we also have to consider the demands of school, athletic, domestic, and social activities, since busy lives may predispose adolescents to poor eating habits, such as skipping meals or eating at fast-food restaurants. Depending on the sport modality and psychological status, young athletes may be more susceptible to unhealthy body weight manipulations (or eating habits) and to the consumption of unnecessary supplements. Sociocultural issues and food preferences are also factors that influence nutrition habits.

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Focusing on the young athlete, this chapter will present topics in energy, macronutrient, and fluid needs, followed by micronutrients and dietary supplements. A more comprehensive rationale for each of these topics can be found in their respective chapter of this book. Finally, practical alternatives are presented to help young athletes, parents, coaches, athletic trainers, and health-care providers to achieve optimal nutritional practices on a more individual basis.

Energy, Macronutrients, and Fluids

Energy

Adequate energy intake is critical for growth, health, and performance of children and adolescents. Energy intake is expected to be higher in those who are athletes due to their greater energy expenditure, but there is a large variability between sports and between individuals. Table 29.1 summarizes some considerations regarding energy needs of young athletes. One feature is that, in nonathletic children, the energy requirement per kilogram of body mass during locomotion can be as much as 30% higher than in adults. This finding is explained by a few distinctive characteristics of children such as greater resting energy expenditure, greater stride frequency during walking and running, differences in kinematic variables, and greater co-contraction of antagonist leg muscles. Whether these characteristics are also prominent in children participating regularly in athletics is unknown, although it is possible that the energy cost of locomotion is lowered

Table 29.1 Considerations related to the energy needs of young athletes

- Growing process, especially during the period of peak height velocity (i.e., growth spurt)
- Maturational process
- Higher metabolic cost of locomotion
- Energy deficit, especially in aesthetic sport modalities, which emphasize leanness that could create health risks such as growth impairment, bone density, and anorexia
- Increased energy intake, especially in sports such as American football, following the general trend, with the risk of overweight/obesity and deleterious consequences for performance, musculoskeletal injuries, and health

as movements become more efficient with specific training, as happens in adults.

Depending on the intensity, duration, and frequency of the sport practice, energy expenditure may vary considerably. For example, daily energy expenditure was estimated to be as high as ~4000 kcal in adolescent male speed skaters using the doubly labeled water technique (Ekelund et al., 2002), while it was ~2600 kcal in adolescent female judokas using a 7-day factorial method (Boisseau et al., 2005). Within a given sport, sex also influences energy expenditure. In male adolescent distance runners, daily energy expenditure was estimated to be ~3600 kcal (57 kcal/kg) and in females, ~2500 kcal (50 kcal/kg) using the 3-day Bouchard activity diary (Eisenmann & Wickel, 2007). As described, energy expenditure measurements in these studies were made by different techniques which could account for variability in outcomes of these studies.

Traditionally, total energy expenditure has been evaluated based on physical activity levels (PAL) and resting energy expenditure (using classical equations). More recently, mathematical models using information from both accelerometers and heart rate monitors have been shown to improve the accuracy of estimates of energy expenditure in children and adolescents (Zakeri et al., 2010), but not specifically in young athletes, but it is likely that the energy cost of different movements and locomotion decreases as proficiency improves with training. The FAO/WHO/UNU (2005) has suggested PAL values for lightly to moderate active, but not competitive, adolescents. A recent study (Carlsohn et al., 2011) calculated the energy expenditure, measured by an activity protocol that had been validated by doubly labeled water, in 64 young athletes (12–18 year olds) performing various sports (track and field, soccer, handball, rowing, canoeing, swimming, and triathlon). The range in PAL was observed to be somewhat higher (1.75–2.05) than FAO/WHO/UNU's suggestion (1.5–1.85) for active adolescents. Considering that the energy demands and recovery patterns of athletic sports for youth may vary, it may be convenient to periodically evaluate energy needs on an individual basis though the lack of precision of such estimates likely precludes their use to prescribe energy intakes.

A careful assessment of height and weight and the use of follow-up growth charts (i.e., National Center for Health Statistics) can assist professionals in evaluating whether energy intake is sufficient to meet the energy demands of young athletes. It is also valuable to follow their maturational status and body composition, since adequate energy availability is essential to achieve optimum peak height velocity and lean tissue accrual once puberty begins (American Academy of Pediatrics, 2005; Bonci et al., 2008).

In sports that emphasize leanness or those that organize competitions by weight category, such as gymnastics and martial arts, young athletes may undergo restrictive diets. Negative energy balance may result in muscle loss and adversely affect strength and endurance performance as well as restricting growth and development (American Academy of Pediatrics, 2005). Of concern are the adverse health effects on various body functions such as immune, endocrine, and musculoskeletal systems. Low energy availability, which can be a result of disordered eating, may disturb the reproductive system causing menstrual irregularities and amenorrhea in the young female athlete. Also, bone mineralization could be impaired due to a decrease in estrogen levels (American Academy of Pediatrics, 2005). It is important, therefore, to investigate whether sufficient dietary energy is made available to these females to avoid such disorders. The National Athletic Trainers' Association developed

a comprehensive position statement (Bonci et al., 2008) with recommendations on early detection of disordered eating as well as prevention and management. More information on this topic is presented in Chapter 42.

In contrast, there appears to be an increased prevalence of obesity and overweight among young athletes that follows the overall tendency in the general population. Approximately 45% of American football high school athletes are overweight or obese (Malina et al., 2007). On the one hand, excess body mass may be perceived as advantageous in sports such as American football; on the other hand, excess fat mass may pose disadvantages in performance (speed, agility, and endurance) and thermoregulatory responses. Young athletes who are obese also have a higher incidence of injuries (Yard & Comstock, 2011), which may be related to postural and balance impairments as well as higher relative musculoskeletal strain (McHugh, 2010). In addition, they may be at a higher risk for other premature health issues related to excess body fat and elevations in systemic inflammatory markers and arterial pressure. In conclusion, energy imbalances in either direction should be prevented, by providing diets that satisfy the young athlete's actual need for macro and micronutrients.

Carbohydrate

Overall, children and adolescents should consume at least 50–55% of their total energy intake as carbohydrates, reflecting a range of about 6–10 g/kg body mass/day (Institute of Medicine, 2005). Complex carbohydrates (e.g., cereals, pasta, and rice) should be the predominant source of energy as opposed to simple sugars. As in adults, carbohydrate is a key fuel for optimum athletic performance and recovery in young athletes. Inadequate carbohydrate intake can result in suboptimal glycogen stores and premature fatigue. It is still uncertain whether young athletes obtain further benefits from a higher carbohydrate diet and from increasing glycogen stores prior to a competitive event (so-called carbohydrate loading), as do adult athletes. The classic method of "supercompensation" that occurs by previously depleting muscle glycogen has not been recommended due to the lack of evidence for efficacy in the young population. Instead, a concurrent decrease in training volume and increase in carbohydrate intake for the last 3–4 days prior to a competition is recommended. This alternative may be advantageous, since many youth sports are characterized by repetitive high-intensity efforts, which could be limited by muscle glycogen reserves.

Carbohydrate intake *during* exercise may also be advantageous for the young athlete. The relative oxidation of consumed (exogenous) carbohydrate during exercise has been shown to decrease with advancing maturation (Timmons et al., 2007b), and children appear to rely relatively more on exogenous carbohydrates than do adults, at least in males. As shown in Figure 29.1, the energy yield from exogenous carbohydrate oxidation during the late stages of exercise is between 25% and 30% in young boys compared to only ~20% in adult men. The reliance on exogenous carbohydrate during exercise is similar (~20% of the total energy expenditure) between preadolescent and adolescent girls and may reflect more advanced biological maturation for a given chronological age in the female groups. It might be that sex differences in substrate utilization start at an earlier age.

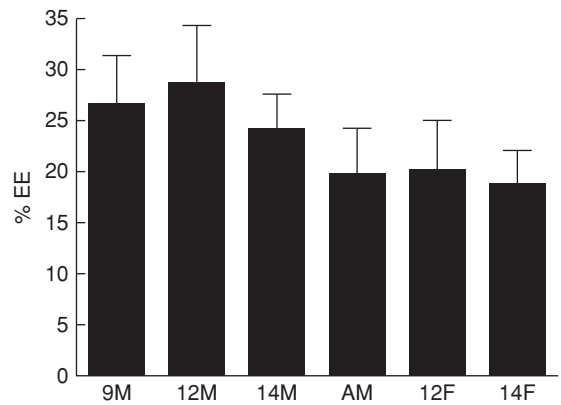

Figure 29.1 Exogenous carbohydrate oxidation expressed as a percentage of total energy expenditure (EE) during exercise. Exercise consisted of 60-minute cycling at 60–70% VO_2 max. Values were calculated over the last 10 minutes of exercise. 9M, 9- to 10-year-old boys ($n = 12$); 12M, 12-year-old boys ($n = 20$); 14M, 14-year-old boys ($n = 9$); AM, 20- to 25-year-old men ($n = 10$); 12F, 12-year-old girls ($n = 12$); 14F, 14-year-old females ($n = 10$). Data are redrawn from Timmons et al. (2003, 2007a, 2007b).

Notwithstanding the effects of carbohydrate intake during exercise on metabolism, it is still unclear whether carbohydrate consumption before or during exercise improves performance in young athletes. Variability in outcomes may be related to the mode of exercise, testing protocols, and maturational status. A study by Riddell et al. (2000) showed that ingestion of a ^{13}C-labeled glucose solution preserved endogenous carbohydrate (glycogen), which could offer extra energy needed for prolonged exercise. When active boys (10–14 year olds) ingested a 6% fructose/glucose-electrolyte drink during 90 minutes of intermittent cycling, an overall improvement of ~40% in a subsequent all-out performance test was observed in 9 of the 12 participants compared to when they ingested water; when a glucose drink was consumed, 7 of the 12 subjects went longer but this was not statistically significant (Riddell et al., 2001). However, ingestion of a similar drink had no effect on rating of perceived exertion when boys cycled 1 hour at 70% VO_2 peak (Timmons & Bar-Or, 2003). In a double-blinded and randomized counterbalanced study, Phillips et al. (2010) examined the effect of ingestion of a 6% carbohydrate-electrolyte drink on performance tests that consisted of 60-minute runs (4- to 15-minute intervals) followed by an intermittent 15-minute run to exhaustion with competitive young (12–14 years) players of football, rugby, and field hockey. Time to exhaustion increased by 24% in the 15-minute runs when carbohydrate was ingested prior to and during the test, but no difference was found in the 15-minute sprint time or peak sprint time.

Most young athletes are involved in sports such as soccer, basketball, tennis, and football, where the ability to perform brief sprints repeatedly is important. Recently, the effect of a 22% CHO beverage consumed 30 minutes prior to a sequence of anaerobic tests (two 30-s Wingate tests separated by ten 10-s sprints) was evaluated in active adolescent (14–16 years) boys (Lee et al., 2011). Mean power on the Wingate test was higher with carbohydrate ingestion (~330 W) than with placebo (~ 293 W), but only in the first test. No effect on heart rate, perception of effort, lactate, or catecholamine levels was observed with carbohydrate ingestion. In pre- and early pubertal boys, a previous study from that same laboratory (Marjerrison et al., 2007) observed no effect of carbohydrate ingestion 30 minutes prior to four Wingate tests with 2-minute intervals. Maturational differences may explain inconsistent findings on the effects of carbohydrate intake on anaerobic performance.

Protein

Protein is particularly important for children and adolescents to sustain growth and muscle development, although it is not a key fuel for exercise. Protein needs, when expressed relative to body mass, decrease from childhood to adulthood, and the dietary reference intakes (DRIs) (Institute of Medicine, 2005) are 0.95 g/kg for ages 4–13 and 0.85 g/kg for ages 14–18, for both boys and girls.

Limited convincing research exists to inform whether young athletes need more protein than their nonathletic peers. Boisseau et al. (2007) examined protein requirements in adolescent (~14 years) soccer players by evaluating the nitrogen balance of three diets with different amounts of protein: 1.4, 1.2, and 1.0 g protein per kg of body weight. Nitrogen balance became more positive with higher levels of protein intake and the calculated daily protein intake needed to maintain a "0" nitrogen balance (where losses equal intake) was 1.04 g/kg/day. This single study suggested that the protein requirement of a 14-year-old male soccer athlete is somewhat above that recommended for an average active male adolescent (1.0 g/kg/day). However, reports of dietary intake of protein in children and adolescents in the United States and Australia seem to exceed the DRI (for review, see Jeukendrup & Cronin, 2011). In cases where the young athlete is undertaking a more rigid diet of energy restriction or consumes a vegan/vegetarian diet, the need to monitor protein intake may be greater.

Fat

From 4 to 18 years of age, the American Heart Association (2005) recommends a daily energy intake from fat of 25–35% (not >10% from saturated fats) of total energy intake and that cholesterol intake does not exceed 300 mg. Therefore, a higher absolute amount of fat intake in young athletes should be explained only by their increased overall energy needs. Despite the universal trend toward increased fat intake, some

young athletes may avoid fats in their diet plan to achieve weight loss. As a consequence, they may face other nutrient deficiencies such as essential fatty acids, fat-soluble vitamins, calcium, and iron.

Using the respiratory exchange ratio and blood parameters, studies have clearly shown that children and adolescents use relatively more fat and less carbohydrate for energy during exercise, compared to adults, at a given relative intensity and during recovery. In a study, which grouped individuals by maturational status, it was shown that the development of an adult-like metabolic pattern occurs between mid- to late puberty and is completed by the end of puberty (Stephens et al., 2006). Hormonal and enzymatic responses could explain the higher fat oxidation rate in children compared to adults, but a clear mechanism remains unknown (for review, see Riddell, 2008). Moreover, from a practical standpoint, there is no evidence that young athletes involved in endurance activities will benefit from a higher fat content in their diet.

Table 29.2 summarizes considerations on carbohydrate, protein, and fat intake for young athletes.

Table 29.2 Considerations of carbohydrate, protein, and fat intake for young athletes

Carbohydrate	• Greater reliance on exogenous carbohydrates during exercise compared to adults • The classic method of "glycogen supercompensation" by previously depleting muscle glycogen is not recommended until additional evidence on safety and efficacy is generated
Protein	• Higher needs relative to body mass in order to sustain growth and optimum muscle development • No evidence for a greater dietary need than their nonathletic peers • Dietary intake usually exceeds the DRIs, except in young athletes undertaking an energy-restricted diet or who consume a vegan/vegetarian diet
Fat	• Greater relative use for energy during exercise, compared with adults, at a given relative intensity and at recovery • No evidence for a greater dietary need than their nonathletic peers

Fluids (Water and Sodium)

Young athletes may be at risk of hypohydration since fluid lost through sweating may not be fully compensated by fluid intake. Many intraindividual and sport-pattern factors influence both sweating rate and fluid intake, making it difficult to recommend a common volume to be ingested during the physical activities of young athletes. It is therefore advisable to periodically evaluate the athlete's hydration levels and fluid intake habits. A practical method to identify acute changes in hydration level is to measure body mass before and after exercise. A decrease in body mass of 2% or more from the initial body weight (or 2% hypohydration) seems to impair performance, as already observed in basketball skills of competitive adolescent players (Dougherty et al., 2006) and in endurance cycling performance of physically active boys (Wilk et al., 2002). Further hypohydration may exacerbate hyperthermia, which is a major concern in prolonged activities in the heat. Therefore, fluid replacement during exercise, as recommended for adults, should be aimed to prevent levels of hypohydration greater than about 2% (Sawka et al., 2007).

Another common finding that has been observed from urinary markers, such as specific gravity and color, is that young athletes often arrive at training sessions and competitions already hypohydrated (Meyer et al., 2012). Young athletes may not properly rehydrate after their athletic activities, especially in sports that carry successive competition events (i.e., basketball, soccer) and where insufficient time is available for recovery. For this reason, attention should be given to rehydration habits to ensure adequate fluid intake during recovery periods or between exercise bouts of same-day matches, especially in warm environments. This recommendation was recently included in a statement of the American Academy of Pediatrics (2011) as a policy to improve safety and performance. It was further advised that children, not specifically athletes, should have sufficient and appropriate fluid available at regular intervals before, during, and after all sports participation.

It should be noted that excessive fluid ingestion during exercise is undesirable. This is most relevant with sodium-free beverages accompanied by large sweating rates, because of the risk of hyponatremia (i.e., serum sodium concentration <135 mEq/l) (Montain et al., 2001). In young athletes, we know very little about the incidence of hyponatremia during sporting events. Episodes of muscle cramping, for example, could be due to hyponatremia, although there is no direct evidence in young athletes, but the true diagnoses will remain unconfirmed if a serum sodium analysis is not performed. Considering that some young athletes may be at risk of hyponatremia (see section on Sodium), the American Academy of Pediatrics (2011) has recently advised that sports drinks containing sodium may benefit child athletes.

Micronutrients and Dietary Supplements

Micronutrient deficiencies can affect physical performance and health. For the pediatric population, attention has been paid to calcium, vitamin D, iron, and sodium and, therefore, these are the elements underlined in the following sections. The roles of other vitamins and trace minerals are presented in Chapter 18.

Calcium and Vitamin D

In the pediatric population, adequate intakes of calcium and vitamin D are of major importance for optimum bone growth and density and prevention of osteoporosis, since ~90% of peak bone mass is attained by age 18 (Bailey et al., 1999). Table 29.3 shows the latest US daily recommended intakes for calcium and vitamin D for children and adolescents (Institute of Medicine, 2011). Requirements may vary due to dietary (low consumption of animal protein) and geographical reasons. Failure to meet adequate calcium and vitamin D intakes can lead to decreased bone mass and increase the risk for bone fractures during sports activities (Tenforde et al., 2010). Most of the calcium intake can be obtained from milk and dairy products, but other sources include dark green leafy vegetables. Major dietary sources of vitamin D are seafood, egg yolk, and various kinds of meat, but exposure of bare skin to sunlight is probably the best source (see Chapter 20). Milk is fortified with vitamin D in some, but not all, countries.

Vitamin D deficiency is detected by its serum concentration in the form of serum 25-hydroxyvitamin D (25(OH)D), recently established as <20 ng/ml by the Institute of Medicine (2011), with the support of the Pediatric Endocrine Society. A cross-sectional survey in Australia showed that 6 of 18 adolescent female elite gymnasts had serum 25(OH)D levels <20 ng/ml and coincidently, a higher prevalence of bone stress injuries was reported in these six females in the year prior to the study (Lovell, 2008). Another study in German gymnasts (Heaney et al., 2008) showed that ~40% had 25(OH)D levels <20 ng/ml. Ward et al. (2009) showed 25(OH)D levels were significantly associated with muscle power (measured by jumping exercise) and force in 12- to 14-year-old girls. Recently, Constantini et al. (2010) found a high prevalence of vitamin D insufficiency (in this study defined as vitamin D <30 ng/ml) among young athletes (mean age 14.7 years) who practiced dance (94%), basketball (94%), and Tae Kwon Do (67%). These findings indicate that screening for vitamin D deficiency in young athletes may be useful,

Table 29.3 Daily recommended intakes and tolerable upper intake levels (UL) of calcium and vitamin D for children and adolescents

Age (years)	Calcium (mg)		Vitamin D (IU)	
	Recommended intake[a]	Tolerable UL[b]	Recommended intake	Tolerable UL
4–8	1000	2500	600	3000
9–18	1300	3000	600	4000

[a]Meets or exceeds the requirement for 97.5% of the population.
[b]Intake level that is likely to pose no risk of adverse health effects to almost all individuals in the general population.

especially for those who experience inadequate sun exposure and/or vitamin D intake and are involved in sports, which require power and intense efforts.

Iron

Iron deficiency can cause fatigue and impaired immune and cognitive functions. Iron is also involved in energy metabolism and is a substrate for bone health. US-recommended daily intakes for iron are 8 mg for ages 9– 13 years; and 11 and 15 mg for boys and girls, respectively, for ages 14–18 years (Institute of Medicine, 2001). The increase in iron requirements during puberty is due to the growth acceleration, and in girls due to onset of menstruation. In the athletic population, additional iron losses may occur from endurance and regular intense exercise. Therefore, to achieve the required needs, the young athlete should ingest sufficient iron in their diets (the average American diet contains about 6 mg of elemental iron for every 1000 calories of food (Institute of Medicine, 2001). As iron status will also depend on intestinal absorption, some recommendations should be given to the young athlete to avoid iron inhibitors such as phytates (seeds, bran, and soy products) and polyphenolic compounds (strong coffee and tea, herb tea, cocoa). To optimize iron absorption, it is also recommended to ingest foods rich in vitamin C and consume iron as heme contained in lean red meats (Meyer et al., 2007). Therefore, iron deficiency is of concern among young athletes who avoid eating red meat and other iron-containing foods. A precise prevalence of iron deficiency among young athletes may depend on the interpretation of hematological parameters (ferritin levels) and it varies according to gender and sport modality (for reviews, see Zoller & Vogel, 2004). Low ferritin levels (<35 μg/l) were detected in 59% of 97 females and in 31% of 96 males who were German and elite adolescent (mean age 16 years) athletes (Koehler et al., 2011). In that study, the prevalence of low ferritin levels in endurance sports was similar among females (12%) and males (13%); however, in ball sports it was greater in females (14%) than in males (7%). Among 11 females involved in aesthetic sports, 8 had low ferritin levels, which could indicate some degree of inappropriate eating (Bonci et al., 2008). Unless iron deficiency and/or anemia are established, there is no evidence that iron supplementation increases athletic performance in young athletes.

Sodium

Sodium losses from the body of a young athlete through sweat may be high during exercise and losses must be replaced by dietary intake. For the pediatric population, emphasis has been given to the tolerable upper intake of dietary sodium of 2.2 g/day (American Heart Association, 2005) in light of the overall trend to consume larger amounts of sodium-rich snacks and foods. Young athletes are not excluded from this trend, but in certain situations attention should be given to the risk of hyponatremia (serum sodium level <135 mEq/l). In adults, hyponatremia has been reported during prolonged activities lasting longer than 2 hours, with most cases happening only when exercise lasts longer than 4–5 hours when there is a combination of high sweating rates and excessive intake of sodium-free drinks (Montain et al., 2001). Attention should, therefore, be given to those young athletes who follow a low-sodium diet and who lose a considerable amount of sodium from sweat (Meyer et al., 2012). Other risk factors for hyponatremia in young athletes are eating disorders (Bonci et al., 2008), renal disorders, and certain mutations of the cystic fibrosis gene (Montain et al., 2001).

Dietary Supplements

The use of dietary supplements is common among young athletes. In most cases, this practice is unnecessary and may present adverse health effects, apart from the risk of being contaminated with a prohibited substance and resulting in a doping offence. In this section, "dietary supplements" are defined as permitted substances (e.g., vitamins, minerals, amino acids or proteins, herbal elements), which will not be described individually as they can be found in various chapters of this Encyclopedia.

According to one study (Petróczi et al., 2008), dietary supplement use was reported by 48% of 403 UK young athletes (mean age 18 years). Besides energy drinks (consumed by 42% of athletes), the

other most consumed supplements were vitamin C and multivitamins (23%), whey protein (21%), and creatine (13%). In that study, supplement consumption was not analyzed by age group, but athletes as young as 12 years were included. Young athletes may start consuming dietary supplements with the expectation that they will improve health and performance, prevent illness, or compensate for any inappropriate diet (Meyer et al., 2007; Petróczi et al., 2008). External incentives, such as advertisements and marketing campaigns that use images of successful adult athletes, may also provide a stimulus to seek out a shortcut to success. Parents, as well as coaches, may also have misplaced beliefs and help supply dietary supplements to their children and/or athletes. Identifying individual motivations and misconceptions is a good strategy to educate the young athlete and prevent them from using unnecessary dietary supplements.

Practical Considerations

Young athletes may have distinct nutritional needs with regard to their energy, hydration, and micronutrient intake. However, to meet these needs with appropriately scheduled meals can be challenging because of the influence of individual, sport-related, and cultural factors. Understanding the basics of sports nutrition may be an initial step for young athletes, parents, and training personnel. Young athletes generally show interest in learning about sports nutrition, yet the information obtained from magazines, health food store personnel, coaches, gym owners, and other athletes may be unreliable. In addition, knowledge is not necessarily accompanied by adherence to appropriate nutritional behaviors. A comprehensive review (Heaney et al., 2011) reported no correlation between nutrition knowledge and dietary intake in studies of young athletes.

Timing of food consumption before, during, and after competition or training events can influence the ability to perform and recover from exercise. To systematically organize individual meal schedules, the assistance of a dietitian specialized in sports nutrition is advisable. Commonly, a "complete" meal is recommended to be ingested about 3–4 hours prior to competing/training. This meal composition should be low in fat and fiber and moderate in protein, but it can be rich in complex carbohydrates and fluids. This is to avoid any gastric distress, nausea, vomiting, and hypohydration. During exercise, it is recommended to ingest water according to amounts lost from sweat. If exercise is prolonged (>1.5–2 hours), or characterized by intermittent bouts at a high intensity and short recovery periods within the same day, as is the case with many youth tournaments, it may be beneficial to add some carbohydrate and sodium to the drinking solution to help maintain blood glucose and sodium levels and optimize fluid absorption (American Academy of Pediatrics, 2011). After the competition/training event, adequate energy in the form of carbohydrate and fluids must be consumed to replenish glycogen stores and to restore any previous and ongoing fluid losses from sweat and urine. Protein intake after exercise can provide amino acids to aid in muscle recovery. Following hard and prolonged exercise, well-balanced and regular (every 2–4 hours) meals are recommended.

Until more information is available concerning the safety of children and adolescents using dietary supplements, health-care providers should actively discourage their consumption. A close follow-up on the nutritional status of the young athlete along with good communication among the athlete and the parents/guardians, health-care professionals, and athletic trainers are needed to ensure optimum performance and health of young athletes.

References

American Academy of Pediatrics. Committee on Sports Medicine and Fitness (2005). Promotion of healthy weight control practices in young athletes. Committee on Sports Medicine and Fitness. *Pediatrics* 116, 1557–1564.

American Academy of Pediatrics (2011) Clinical report—sports drinks and energy drinks for children and adolescents: are they appropriate? *Pediatrics* 127, 1182–1189.

American Heart Association (2005) Dietary recommendations for children and adolescents: a guide for practitioners. *Circulation* 112, 2061–2075.

Bailey, D.A., McKay, H.A., Mirwald, R.L., et al. (1999) A six-year longitudinal study of the relationship of physical activity to bone mineral accrual in growing children: the University of Saskatchewan

Bone Mineral Accrual Study. *Journal of Bone and Mineral Research* 14, 1672–1679.
Boisseau, N., Vera-Perez, S., & Poortmans, J. (2005) Food and fluid intake in adolescent judo athletes before competition. *Pediatric Exercise Science* 17, 62–71.
Boisseau, N., Vermorel, M., Rance, M., et al. (2007) Protein requirements in male adolescent soccer players. *European Journal of Applied Physiology* 100, 27–33.
Bonci, C.M., Bonci, L.J., Granger, L.R., et al. (2008) National Athletic Trainers' Association position statement: preventing, detecting, and managing disordered eating in athletes. *Journal of Athletic Training* 43, 80–108.
Carlsohn, A., Scharhag-Rosenberger, F., Cassel, M., et al. (2011) Physical activity levels to estimate the energy requirement of adolescent athletes. *Pediatric Exercise Science* 23, 261–269.
Constantini, N.W., Arieli, R., Chodick, G., et al. (2010) High prevalence of vitamin D insufficiency in athletes and dancers. *Clinical Journal of Sport Medicine* 20, 368–371.
Dougherty, K.A., Baker, L.B., Chow, M., et al. (2006) Two percent dehydration impairs and six percent carbohydrate drink improves boys' basketball skills. *Medicine & Science in Sports & Exercise* 38, 1650–1658.
Eisenmann, J.C. & Wickel, E.E. (2007) Estimated energy expenditure and physical activity patterns of adolescent distance runners. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 178–188.
Ekelund, U., Yngve, A., Westerterp, K.R., et al. (2002) Energy expenditure assessed by heart rate and doubly labeled water in young athletes. *Medicine & Science in Sports & Exercise* 34, 1360–1366.
FAO/WHO/UNU (2005) Human energy requirements. Scientific background papers from the Joint FAO/WHO/UNU Expert Consultation. October 17–24, 2001. Rome, Italy. *Food and Nutrition Technical Report Series. 35ff*.
Heaney, R.P., Armas, L.A., Shary, J.R., et al. (2008) 25-Hydroxylation of vitamin D3: relation to circulating vitamin D3 under various input conditions. *American Journal of Clinical Nutrition* 87, 1738–1742.
Heaney, S., O'Connor, H., Michael, S., et al. (2011) Nutrition knowledge in athletes: a systematic review. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 248–261.
Institute of Medicine. Food and Nutrition Board (2001)*Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium and Zinc*. National Academies Press, Washington, DC.
Institute of Medicine. Food and Nutrition Board (2005) *Dietary Reference Intakes for Individuals, Macronutrients*. National Academies Press, Washington, DC.
Institute of Medicine. Food and Nutrition Board (2011) *Dietary Reference Intakes for Vitamin D and Calcium*. National Academies Press, Washington, DC.
Jeukendrup, A. & Cronin, L. (2011) Nutrition and elite young athletes. In *The Elite Young Athlete* (eds. N. Armstrong & A.M. McManus), Medicine and Sport Science, vol. 56, pp. 47–58. Karger, Basel.
Koehler, K., Braun, H., Achtzehn, S., et al. (2011). Iron status in elite young athletes: gender-dependent influences of diet and exercise. *European Journal of Applied Physiology* 12, 513–523.
Lee, J.D., Sterrett, L.E., Guth, L.M., et al. (2011) The effect of pre-exercise carbohydrate supplementation on anaerobic exercise performance in adolescent males. *Pediatric Exercise Science* 23, 344–354.
Lovell, G. (2008) Vitamin D status of females in an elite gymnastics program. *Clinical Journal of Sport Medicine* 18, 159–161.
Malina, R.M., Morano, P.J., Barron, M., et al. (2007) Overweight and obesity among youth participants in American football. *Journal of Pediatrics* 151, 378–382.
Marjerrison, A.D., Lee J.D., & Mahon, A.D. (2007) Preexercise carbohydrate consumption and repeated anaerobic performance in pre- and early-pubertal boys. *International Journal of Sport Nutrition and Exercise Metabolism* 17, 140–151.
McHugh, M.P. (2010) Oversized young athletes: a weighty concern. *British Journal of Sports Medicine* 44, 45–49.
Meyer, F., O'Connor, H., & Shirreffs, S.M. (2007) Nutrition for the young athlete. *Journal of Sports Sciences* 25(Suppl. 1), S73–S82.
Meyer, F., Volterman, K.A, Timmons, B.W., et al. (2012) Fluid balance and dehydration in the young athlete: assessment considerations and effects on health and performance. *American Journal of Lifestyle Medicine* 6, 489–501.
Montain, S.J., Sawka, M.N., & Wenger, C.B. (2001) Hyponatremia associated with exercise: risk factors and pathogenesis. *Exercise Sports Science Reviews* 29, 113–117.
Petróczi, A., Naughton, D.P., Pearce, G., et al. (2008) Nutritional supplement use by elite young UK athletes: fallacies of advice regarding efficacy. *Journal of the International Society of Sports Nutrition* 5, 22.
Phillips, S.M., Turner, A.P., Gray, S., et al. (2010) Ingesting a 6% carbohydrate-electrolyte solution improves endurance capacity, but not sprint performance, during intermittent, high-intensity shuttle running in adolescent team games players aged 12–14 years. *European Journal of Applied Physiology* 109, 811–821.
Riddell, M.C. (2008) The endocrine response and substrate utilization during exercise in children and adolescents. *Journal of Applied Physiology* 105, 725–733.
Riddell, M.C., Bar-Or, O., Schwarcz, H.P., et al. (2000) Substrate utilization in boys during exercise with [13C]-glucose ingestion. *European Journal of Applied Physiology* 83, 441–448.
Riddell, M.C., Bar-Or, O., Wilk, B., et al. (2001). Substrate utilization during exercise with glucose and glucose plus fructose ingestion in boys ages 10–14 yr. *Journal of Applied Physiology* 90, 903–911.
Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.
Stephens, B.R., Cole, A.S., & Mahon, A.D. (2006) The influence of biological maturation on fat and carbohydrate metabolism during exercise in males. *International Journal of Sports Nutrition and Metabolism* 16, 166–179.
Tenforde, A.S., Sayres, L.C., Sainani, K.L., et al. (2010) Evaluating the relationship of calcium and vitamin D in the prevention of stress fracture injuries in the young athlete: a review of the literature. *Journal of the American Academy of Physical Medicine and Rehabilitation* 2, 945–949.
Timmons, B.W. & Bar-Or, O. (2003) RPE during prolonged cycling with and without carbohydrate ingestion in boys and men. *Medicine & Science in Sports & Exercise* 35, 1901–1907.
Timmons, B.W., Bar-Or, O., & Riddell, M.C. (2003) Oxidation rate of exogenous carbohydrate during exercise is higher in boys than in men. *Journal of Applied Physiology* 94, 278–284.
Timmons, B.W., Bar-Or, O., & Riddell, M.C. (2007a) Energy substrate utilization during prolonged exercise with and without carbohydrate intake

in preadolescent and adolescent girls. *Journal of Applied Physiology* 103, 995–1000.

Timmons, B.W., Bar-Or, O. & Riddell, M.C. (2007b) Influence of age and pubertal status on substrate utilization during exercise with and without carbohydrate intake in healthy boys. *Applied Physiology, Nutrition, and Metabolism* 32, 416–425.

Ward, K.A., Das, G., Berry, J.L., et al. (2009) Vitamin D status and muscle function in postmenarchal adolescent girls. *Journal of Clinical Endocrinology and Metabolism* 94, 559–563.

Wilk, B., Yuxiu, H., & Bar-Or, O. (2002) Effect of hypohydration on aerobic performance of boys who exercise in the heat [abstract]. *Medicine & Science in Sports & Exercise* 34, S48.

Yard, E. & Comstock, D. (2011) Injury patterns by body mass index in US high school athletes. *Journal of Physical Activity and Health* 8(2), 182–191.

Zakeri, I.F., Adolph A.L., Puyau, M.R., et al. (2010) Multivariate adaptive regression splines models for the prediction of energy expenditure in children and adolescents. *Journal of Applied Physiology* 108, 128–136.

Zoller, H. & Vogel, W. (2004) Iron supplementation in athletes—first do no harm. *Nutrition* 20, 615–619.

Chapter 30

The Aging Athlete

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Introduction

This chapter provides an overview of the physiological benefits of sport to the aging individual, discusses nutrition recommendations for the aging athlete, and highlights special nutrition concerns including hydration, nutrient–medication interactions, and dietary supplements.

Aging is universal (everyone does it), intrinsic (even in a protective, toxin-free environment with the perfect balance of nutrients, aging occurs), and detrimental (there are negative changes in all physiological functions). However, participation in sport throughout life might be the only true anti-aging prescription. Masters athletes may be the best representatives for "successful aging" (Tanaka & Seals, 2008). The age at which one becomes an "aging" athlete or a master athlete varies with sport, but in general most champions are not older than mid-to-late thirties. Marathon times in older athletes have improved dramatically, as shown in Figure 30.1 of the world single age records in the marathon for men and women. In October 2011, an 80-year-old ran a 3:15:54 marathon…a remarkable feat when you consider that this 80-year old averaged less than 7.5 minutes/mi for the 26.2 mi course. Lepers and Cattagni (2012) analyzed running times in the New York City Marathon from 1980 to 2009 and found that during that period not only did the participation of masters runners increase, but that the times of older runners significantly decreased (for males older than 64 years and females older than 44 years). The authors conclude that masters runners have "probably not yet reached their limits in marathon performance." Most master or veteran athlete's organizations admit entry to competition at age 35 or 40 years. Athletes participating in skill sports like golf or baseball often compete into their late fourth or fifth decade of life; witness professional American golfer Tom Watson who finished second at the 2009 British Open at the age of 59, losing by one stroke in a playoff, or Randy Johnson, an American baseball pitcher who threw a perfect game at the age of 40–-the oldest player to do so.

Aging is a global phenomenon. In the not too distant future, people aged 65 and over will outnumber children under age five for the first time in history (Kinsella & He, 2009). In 2008, the global population over the age of 65 years was about 506 million or about 7% of the world's population; by 2040, the projection is 1.3 billion older people or doubling to about 14% of the population. Figure 30.2 shows the world's top 10 oldest populations in rank order. Keeping the older population in good health will be a big challenge and regular physical activity will be a cornerstone to keeping older people in good functional health. With an aging population there is likely to be a greater participation in competitions arranged for aging athletes.

It is not known how many athletes compete at the master level but many local, national, and international competitions for aging athletes are held annually. Table 30.1 identifies some of those organizations.

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Table 30.1 Examples of masters athletes organizations and competitions for aging athletes

Organization	Brief history	Web site	Sponsored activities	Drug testing?
National Senior Games Association (NSGA)	Originated as US National Senior Olympics in 1985 Held first National Senior Olympics in 1987 with 2500 competitors Changed name to US National Senior Sports Organization in 1990 Today known as National Senior Games Athletes must be 50 years or older and qualify through NSGA State Games	www.nsga.com	Summer National Senior Games (held odd number years) Winter National Games (held even number years but suspended in 2001; with interest in restarting winter games)	No
International Masters Games Association (IMGA)	First World Masters Games held in 1985 Reorganized in 1995 as IMGA from member International Federations to represent masters sport worldwide Athletes over the age of 35 are eligible to compete	www.imga.ch	European Masters Games held every 4 years World Masters Games held every 4 years Summer World and Winter Master Games held every other year	Yes, anti-doping policy adopted with drug testing at events
World Masters Athletics	Begun by middle-aged road runners and first organized event was held in 1975; it has evolved into an international masters organization for track and field for those 35 years of age and older	www.world-masters-athletics.org	Sponsors area and world championships in both stadia (events held within a stadium like track and field events) and non-stadia (events held on the road like cross-country and longer distance events.)	Yes, anti-doping policy adopted with drug testing at events
Huntsman World Senior Games	Began as World Senior Games in 1987 Today known as Huntsman Senior Games Open to any athlete 50 years and older	www.seniorgames.net	Yearly competitions held in St. George, Utah, USA	No

Source: Adapted with permission from Rosenbloom and Coleman (2012).

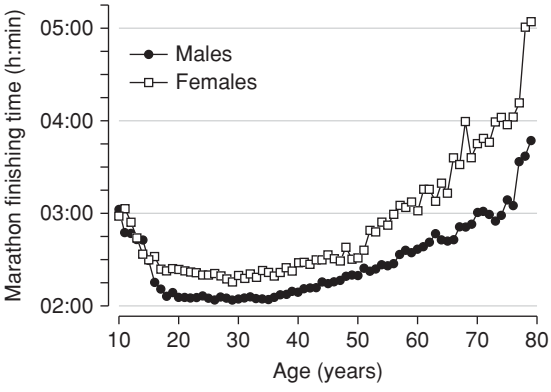

Figure 30.1 World male and female single age records marathon. (http://www.arrs.net/SA_Mara.htm)

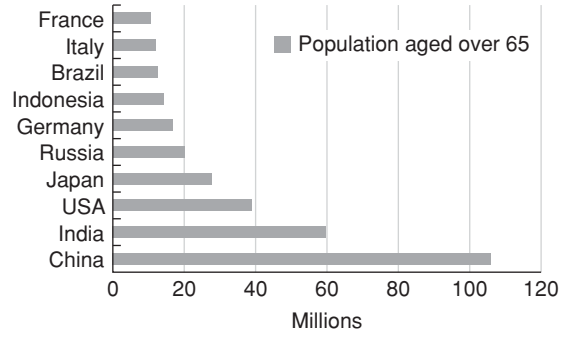

Figure 30.2 Rank order of the world's 10 largest older populations: 2008 (in millions) rank country population aged 65 and over. From US Census Bureau, International Data Base, accessed on October 2, 2011.

Table 30.2 Comparison of Paris Olympic Games (1900) gold medal winners with Masters Athlete Track and Field World Ranking by age group in athletics events

Event	1900 Olympic Games	35–39 years old	40–44 years old	45–49 years old	50–54 years old
100 m (s)	10.8	9.97	10.26	10.72	10.95
200 m (s)	22.2	20.11	20.64	21.80	22.53
400 m (s)	50.4	45.68	47.82	49.89	51.39
800 m (min:s)	1:59	1:43.36	1:48.81	1:54.18	1:58.65
1500 m (min:s)	4:06	3:32.45	3:42.65	3:48.53	4:05.20
Marathon (h:min:s)	2:59:45	2:05:59	2:08:46	2:14:16	2:19:29

Source: Data taken from International Olympic Committee, www.olympic.org/ and Masters Athletics Track and Field World Ranking, www.mastersathletics.net/

Much of what we know about the effect of aging on exercise performance comes from the study of master athletes. However, there is much that we do not know about nutritional status and altered nutrient needs of aging athletes therefore recommendations are extrapolated from younger athletes coupled with guidance on dietary recommendations from the Institute of Medicine (IOM).

What we do know is that an aging athlete of today could outperform the elite athlete of yesterday. Table 30.2 shows the results of the victors at the 1900 Olympic Games held in Paris for athletic events compared to the current master athlete's track and field world rankings by age group. Today's master athlete at age 49 years outperforms the 1900 Olympic gold medalist in every event and the 50- to 54-year olds recorded better times in three of the six events. Of course, today's athletes have an advantage in the choice of clothing, shoes, and running surfaces while at the same time training and nutrition knowledge have vastly improved since 1900.

Sports Participation and Effects on Physiological Systems

Cardiorespiratory Function and Cardiovascular Disease Risk

Aging results in changes to VO_2 max but it is hard to separate aging from disease and disuse. It is generally accepted that at about the age of 30 years VO_2 max declines about 10% per decade in healthy adults whereas in older endurance athletes the decline is about 5% per decade. Aging athletes who continue to perform endurance-type activities retain greater aerobic capacity than sedentary age-matched controls (Maron et al., 2001). Research on masters runners has shown that peak endurance is maintained until about the age of 35 years and then modest declines are seen until the fifth and sixth decade with more severe decreases in performance after the age of 60 (Tanaka & Seals, 2008). Wright and Perricelli (2008), studying participants over the age of 50 competing in the 2001 National Senior Olympic Games, found a 3.4% decrease in performance per year over 35 years of competition in both male and female athletes (50–75 years). Those who take up exercise in late middle age, however, can expect to see fitness levels improve over a number of years before the aging process takes over and the inevitable decline begins.

The decrease in VO_2 max is not fully understood but is believed to result from the reduction in training stimulus more so than changes to exercise economy or lactate threshold (Franklin et al., 2004; Tanaka & Seals, 2008). Aging athletes may lack the time to train at high levels as they did when they were younger; family and work responsibilities take time from training volume, and motivation may shift from setting personal best records to the health benefits of exercise (Roper et al., 2003).

Regular physical exercise is the main pillar of prevention for cardiovascular disease. Aerobic exercise seems to confer benefits on lipid profiles and aging athletes tend to have lipid profiles that put them at

lower risk for heart disease. The cardioprotective effect of exercise may be in part due to the increase in high-density lipoprotein cholesterol (HDL-C) and the lowered ratio of total cholesterol (TC) to HDL-C (Goldberg, 2000). However, it appears that regular continuous exercise is needed to maintain a favorable lipid profile. Giada et al. (1995) found that a 2-month break from training in older elite cyclists reversed desirable lipid profiles.

Muscle Function

Whereas VO_2 max starts to decline in the early fourth decade, age-related muscle atrophy usually begins at about age 50 when a loss of both muscle mass and strength of about 10–15% per decade ensues (Sallinin et al., 2008). Progressive, resistance strength training has been shown to increase muscle hypertrophy and strength but even lifetime physical activity cannot completely buffer the age-related declines in muscle fiber loss (Faulkner et al., 2008). Contributing to the loss of muscle mass and strength is the decrease in the number of functioning motor units. However, Power et al. (2010) estimated the number of functioning motor units in 65-year-old masters runners, 25-year-old recreational runners, and healthy sedentary 65-year-olds and found that motor units were lowest in the sedentary men with only small differences between the masters runners and young recreational runners. Lifelong high-intensity exercise could blunt the loss of motor units in masters runners.

When it comes to muscle strength, it is never too late to begin a strength training program. Fiatarone et al. (1994) have shown that even in sedentary, elderly nursing home residents (the oldest participant was 98 years old) muscle strength can be improved with regular resistance exercise. Improving muscle strength improves gait and balance with aging. Jannsen (2009) reviewed both randomized and non-randomized trials of strength training and increases in skeletal mass in previously sedentary individuals and concluded that there is a 1% increase in skeletal muscle size for each week of resistance training. Considering the usual decline in muscle mass, strength training for 6 weeks could reverse a decade's worth of muscle loss in aging individuals.

Bone Mineral Density

Preserving bone mass is especially important for women, who lose bone about a decade earlier than men begin to show bone loss. Estrogen losses at menopause results in an acceleration of bone loss due to resorption of bone and a decrease in bone mass accretion. Less is known about bone loss in men, but it is thought that declining androgens in elderly men contribute to loss of bone.

Master athletes tend to have better bone health, both greater bone mineral density and bone mineral content compared to sedentary individuals, probably related to the mechanical stress on bone through exercise. Wilks et al. (2009) conducted bone scans on the tibia and the radius in competitive female sprinters, race walkers, middle- and long-distance runners and compared the results to age-matched controls. The athletes ranged in age from 35 to 94 years and had been exercising for at least a decade prior to the study. As expected, the athletes had greater bone mass and bone mineral content in the tibia compared to controls. Male athletes had 14% larger tibias and female athletes 26% larger tibias compared to controls; there was also about 4% increase in cortical bone in the athletes. There was no difference between athletes and controls in the radius as this bone is not loaded during running or walking.

Even moderate impact exercise can improve bone health in aging athletes. Velez et al. (2008) studied bone mineral density in athletes competing in the National Senior Olympics in running and swimming events compared to controls; all subjects in the study were 65 years or older. The researchers found that total body bone mineral density of runners was significantly greater than controls and marginally improved in the swimmers, suggesting that impact sports have a greater positive effect on bone than non-impact sports.

Andreoli et al. (2012) conducted a retrospective study with post-menopausal women who were athletes (both weight bearing and non-weight-bearing sports) during their younger years and compared bone mineral density and bone mineral content with sedentary women. Both bone mineral density and mineral content were greater in all athletes than in sedentary controls. The researchers concluded that

exercise during younger ages conferred a benefit on bone mass and can prevent bone loss due to aging.

Body Weight

Obesity is a global concern. Among the 34 countries in the Organization for Economic Cooperation and Development (OECD), one in two adults is overweight and one in six is obese (Robb, 2010). Overweight and obesity rates are highest in the United States and Mexico and lowest in Korea and Japan, but obesity rates are growing in every country.

Lack of physical activity is a major contributor to the obesity epidemic and aging athletes are not immune to weight gain or body composition changes, but exercise is one way to temper age-related weight gain. While the absolute amount of exercise needed to prevent weight gain is not known, Lee et al. (2010) examined the association of physical activity with long-term weight change in over 34,000 US women who were eating a usual diet. The women (mean age of 54 years) were followed for 13 years and weight and physical activity levels (PAL) were measured at baseline and every 3 years during the study. At the end of the study, a mean of 2.6 kg was gained in all women. However, those who averaged 60 minutes a day of moderate-intensity exercise during the years of the study gained the least amount of weight (the two lesser active groups of women were significantly more likely to gain more than 5 pounds over a 3-year period than the higher active groups of women). The researchers concluded that it is possible to prevent overweight and obesity with aging by regular, moderate-intensity exercise. The amount of activity performed by women in this study is in line with the amount of exercise done by aging athletes.

Insulin Resistance

Aging brings about changes in disposal of blood glucose with the common result of increased fasting glucose levels due to insulin resistance. The end result could be type 2 diabetes. Amati et al. (2009) demonstrated that obesity coupled with chronic physical inactivity are likely the real cause of insulin resistance rather than aging per se. Measuring insulin sensitivity in younger and older endurance athletes at healthy weights, sedentary younger and older adults, and obese younger and older adults, the researchers demonstrated that regardless of age, athletes were more sensitive to insulin than either normal weight or obese individuals. The authors conclude that insulin resistance may not be a characteristic of aging but more likely it is associated with obesity and physical inactivity.

Emerging Health Benefit of Exercise

Aging athletes have lower cardiovascular disease risk, improved ability to maintain skeletal muscle, better bone architecture and mass, and less risk of obesity and diabetes. Emerging research also suggests that brain function is improved in masters athletes. Tseng et al. (2011) were the first to look at brain tissue integrity in masters athletes using MRI. Ten masters athletes (mean age 73 years, training for more than 15 years) matched with age and educational level sedentary elderly men underwent MRI to assess white matter in the brain. Masters athletes had greater white matter integrity in areas of the brain related to motor control, visuospatial function, and working memory, suggesting that these functions are better preserved in aging athletes.

Nutritional Considerations for Aging Athletes

Research on the physiological benefits of aging or masters athletes is available, but there is very little information on nutritional status, nutrient recommendations, or supplement use in this population. Much of what is known is extrapolated from younger athletes or from older adults who are recreational athletes but not highly competitive athletes.

Energy Needs

Energy requirements for aging athletes are not known with precision; energy needs usually decline with aging as reflected in a lower basal metabolic rate (BMR) due decreasing lean muscle mass. An aging athlete who maintains muscle mass through vigorous exercise probably has the same energy needs as a younger adult and many prediction equations

include age as a factor. Total energy expenditure (TEE) is comprised of BMR, thermic effect of food (TEF), and physical activity and all have been reported to decline with age. Physical activity is the most variable component in any age group so physical activity factors have been developed to be added to the energy expenditure equations to make adjustments for exercise. Therefore, using various prediction equations one can estimate energy needs for the aging athlete as these equations incorporate PAL to determine an energy intake level that should meet energy expenditure. However, none of these equations is a perfect predictor of energy expenditure and may over- or underestimate energy needs by as much as 20%; providing athletes with an energy range rather than a single number is more appropriate to meet their energy needs. Thompson and Manore (1996) studied energy expenditure in highly trained male and female endurance athletes (mean age of 26 for males and 31 for females) measuring RMR using a metabolic cart and then compared the results with various prediction equations. The Cunningham equation predicted measured RMR more accurately than the other equations (Harris and Benedict, Owen, and Mifflin). The Cunningham equation uses fat-free mass which is usually estimated from body fat percentage measurements. Therefore, this equation can only be used when body composition data are available.

Commonly used energy prediction equations include the following:

- Estimated Energy Requirements (EER) from the Food and Nutrition Board, IOM (Institute of Medicine, 2002)
- Mifflin–St Jeor equation (Mifflin et al., 1990)
- Cunningham equation (Cunningham, 1980)

Table 30.3 gives PALs from the IOM and Table 30.4 shows some of the prediction equations with examples for male and female aging athletes. The examples demonstrate the wide variability when using prediction equations for athletes.

Table 30.3 Physical Activity Level (PAL)

PAL	Description	Mean PAL factor	PAL range
Sedentary	Sedentary occupation; mostly sitting	1.2	1.0–1.4
Low active	Sedentary occupation with some walking for throughout the day	1.5	1.4–1.6
Active	Exercise for about 1 h a day or equivalent to about 12,000–16,000 steps per day	1.75	1.6–1.9
Very active	Exercise for several hours per day; either competitively or recreationally	2.2	1.9–2.5

Source: IOM (2002).

Carbohydrate Needs

The IOC Consensus Statement on Sports Nutrition (2010) advises athletes to match demands of training with adequate carbohydrate intake. Recommendations are not age-specific but because training volume may be different in aging vs. young athletes, matching carbohydrate intake with training needs will help fuel athletes for all activities. Insulin-regulated glucose transporter (GLUT4) is increased in older individuals who are exercise-trained and the levels are about the same as seen in young adults in response to training (Cartee, 1994) so it is logical to assume that older athletes have the same needs as younger athletes. The guidelines presented in Table 30.5 can be used for training and competition for aging athletes. Choosing whole grain breads and cereals, fruits, vegetables, and low-fat dairy foods will provide quality carbohydrates to fuel training and competition while at same time providing micronutrients.

While not universally accepted as the best way to select carbohydrate-rich foods, a diet that bases recommendations on the glycemic index (GI) is popular with consumers. For athletes, choosing carbohydrates based on GI may be a useful tool to select carbohydrates before, during, or after training. Low-GI foods may be better tolerated by some athletes before training and competition, whereas high-GI foods may help to restore glycogen after heavy exercise. Foods with a low GI include most whole fruits, milk, yogurt, and whole grain breads and cereals. High GI foods include sport drinks, potatoes, and refined grains.

Table 30.4 Examples of predictive energy equations and examples using male and female aging athletes

Equation	Example for male athlete: *A*, 50 years; *H*, 180 cm (1.8 m); *W*, 78 kg; FFM 70 kg; PA 2.2	Example for female athlete: *A*, 50 years; *H*, 165 cm (1.65 m); *W*, 59 kg; FFM, 48 kg; PA 2.0
Estimated Energy Requirements (Institute of Medicine, 2002)[a] Males: kcal/day = 662 − 9.53*A* + [PA × (15.9*W* + 540*H*)] Females: kcal/day = 354 − 6.91*A* + [PA × 9.36*W* + 726*H*)]	662 − 9.53(50) + [2.2 × (15.9 × 78 + 540 × 1.8)] = 5051 kcal/day	354 − 6.91(50) + [2.0 × (9.36 × 59 + 726 × 1.65)] = 3508 kcal/day
Cunningham equation (Cunningham, 1980)[b] Males and Females kcal/day = 500 + 22FFM × PA	500 + 22 (70) × 2.2 = 4488 kcal/day	500 + 22(48) × 2.0 = 3112 kcal/day
Mifflin–St Jeor equation (Mifflin et al., 1990)[c] Males: kcal/day = 10*W* + 6.25*H* − 5*A* +5 × PA Females: kcal/day = 10*W* + 6.25*H* − 4.92*A* − 161 × PA	10 (78) + 6.25(180) − 5(50) + 5 × 2.2 = 3652 kcal/day	10 (59) + 6.25(165) − 4.92(50) − 161 × 2.0 = 2428 kcal/day

[a]A, age in years; W, weight in kilograms; H, height in meters; PA, physical activity.
[b]FFM, fat-free mass; PA, physical activity.
[c]A, age in years; W, weight in kilograms; H, height in centimeters; PA, physical activity.

Protein Needs

The Recommended Dietary Allowance (RDA) for protein was established without taking physical activity into account. Research has demonstrated that protein needs are increased in both endurance and strength training athletes with the recommendation of 1.2–1.7 g protein/kg of body weight (Rodriguez et al., 2009). These recommendations come from studies using young, competitive athletes with a high volume of training and as Tarnopolsky (2008) points out, it is unlikely that older athletes will have the high training volume so using a range of protein of 1.0–1.2 g/kg/day is probably sufficient. It is important to note that protein needs are highly dependent on energy intake so protein needs could be higher when energy intake is low. Older athletes are encouraged to consume lean sources of protein to keep saturated fat intake low as risk for cardiovascular disease increases with age. Lean protein sources include lean beef and pork (cuts with "round" or "loin" in the name), chicken, turkey, eggs, low-fat milk, cheese, and yogurt, as well as plant-based proteins such as soy, ground and tree nuts and nut butters, dried beans and peas, and the grain quinoa.

Timing protein intake around exercise training has been shown to be beneficial to stimulate muscle protein synthesis in athletes, so consuming 15–25 g of high-quality protein (i.e., protein that provides all of the essential amino acids) is recommended within the hour after exercise (IOC Consensus Statement, 2010). Older athletes might consider consuming high-quality protein with every meal and add a progressive resistance exercise program to their routine exercise regimen to help maintain muscle mass and prevent atrophy.

Table 30.5 gives recommendations for carbohydrate and protein intakes for aging athletes.

Micronutrients

Little is known about the need for micronutrients in aging athletes, but a few researchers have tried to assess vitamin and mineral intakes through diet recalls. Nieman et al. (1989) collected food diaries from about 350 participants in the 1987 Los Angeles Marathon and most of the athletes consumed greater than two-thirds of the RDA for all nutrients, however the female marathoners had intakes of zinc and vitamin D less than two-thirds of the RDA. Chatard et al. (1998) collected food records

Table 30.5 Macronutrient recommendations for aging athletes

Carbohydrate	Protein	Fat
3–5 g/kg/day for low intensity or light training 5–7 g/kg/day for moderate training of 1 h/day 6–10 g/kg/day for moderate intensity training of 1–3 h/day 8–12 g/kg/day for ultraendurance training at high intensity for 3–4 h/day	1.0–1.7 g/kg/day with protein at the higher end when energy intake is restricted or a strength training program is in the initial stages	20–35% of total energy Linoleic acid 17 g/day for men and 12 g/day for women Alpha-linoleic acid 1.6 g/day for men and 1.1 g/day for women

Source: Burke et al. (2011), Rodriguez, et al. (2009), Tarnopolsky (2008), IOM (2002).

from older (mean age 63 years) French endurance and team sport athletes using multiple 3-day food records and found higher energy intakes than in the French general population but intakes of vitamin D and magnesium were lower than the French RDA (–70% and –16%, respectively). An intake of less than the RDA is, of course, not an indication of an inadequate intake and the authors noted that diet intake data were analyzed for 6 days which may not be representative of the typical diet.

Beshgetoor and Nichols (2003) collected food and supplement intakes in 25 female masters runners and cyclists (mean age 50 years) and compared micronutrient intakes in those who took supplements and those who did not. Although this was a small sample and relied on self-reported data, the researchers reported that average intakes of vitamins D and E, folic acid, calcium, magnesium, and zinc were lower than the US RDA in the women who did not take supplements.

The need for some vitamins and minerals increases with age and for two minerals (iron and chromium) decreased intakes are recommended. Table 30.6 summarizes the increased and decreased needs for micronutrients; most of the changes occur in the 50-year and older categories. The RDA or Adequate Intake (AI) is reported along with the Tolerable Upper Limit (UL) which can be used as guide for the UL for supplementation.

Two nutrients of particular interest to athletes are calcium and vitamin D. Both are closely tied to bone health and emerging research suggests that vitamin D may play a role in exercise. Inadequate calcium intakes are common in women, especially in western nations. Net bone formation occurs until about the age of 30 which is probably the time when peak bone mass occurs. During adulthood bone loss is very slow until the age of menopause in women (and probably about age 60 in men) when bone loss accelerates. Considering that women can now live one-third to one-half of their lives after menopause, preventing osteopenia and osteoporosis are global public health concerns. The International Osteoporosis Foundation (2011) reports that osteoporosis is estimated to affect 200 million women worldwide and they suggest three steps to "unbreakable" bones: adequate calcium, vitamin D, and protein. Strength training is also recommended, but having AI of nutrients to strengthen bones is critical. In addition to normal aging, several medications can contribute to bone loss. Corticosteroids (used as anti-inflammatory drugs and for the treatment of asthma, inflammatory bowel diseases, and rheumatoid arthritis) and aromatase inhibitors (used as breast cancer treatment), selective serotonin reuptake inhibitors (used as antidepressants), and proton pump inhibitors and aluminum-containing antacids (used for heartburn and other GI disorders) can all contribute to bone loss. Men and women should aim for 1000–1200 mg calcium/day, preferably from food. If supplements are used, older adults should consider taking calcium in the citrate form for better absorption.

Vitamin D is critical for calcium regulation but emerging research suggests that this vitamin also helps to regulate skeletal muscle and immune cell function, and may protect against cancer. At the current time, the IOM defines Vitamin D deficiency

Table 30.6 Micronutrient needs that are altered with age

Micronutrient	DRI[a]	Age-related change	Tolerable UL levels (ULs)	Reason for change
Vitamin D (cholecalciferol)	Males and females 31–70 years 15 μg/day >70 years 20 μg/day	⇧	4000 IU/day	Aging results in less cutaneous synthesis of Vitamin D and lower circulating levels of the vitamin
Vitamin B6 (pyridoxine)	Males 31–50 years 1.3 mg/day >51 years 1.7 mg/day Females 31–50 years 1.3 mg/day >51 years 1.5 mg/day	⇧	100 mg/day	Increased due to aging and gender effects of approximately 0.2–0.3 mg food B6/day
Vitamin B12 (cyanocobalamin)	Males and females >51 years 2.4 μg/day	⇧	Not determined	10–30% of older adults malabsorb food-bound vitamin B12 so fortified or synthetic B12 is recommended
Calcium	Males 31–70 years 1 g/day >71 years 1.2 g/day Females 31–50 years 1 g/day >51 years 1.2 g/day	⇧	2500 mg/day (ages 30–50 years) 2000 mg/day (ages >50 years)	Calcium absorption decreases with age and intakes greater than 1000 mg/day may reduce bone loss
Iron	Males >31 years 8 mg/day Females 31–50 years 18 mg/day >51 years 8 mg/day	⇩	45 mg/day	The RDA for women over the age of 50 is reduced to account for loss of menses
Chromium	Males 31–50 years 35 μg/day >51 years 30 μg/day Females 31–50 years 25 μg/day >51 years 20 μg/day	⇩	Not determined	The adequate intake (AI) for chromium is tied to energy intake; with lowered energy intakes in older adults, chromium needs decrease accordingly. However the DRI report indicates a need for research on chromium requirements in older adults.

Source: Dietary Reference Intakes, IOM, National Academy of Sciences, www.nap.edu
[a]RDA reported for all nutrients except chromium (AI).

when serum 25-OHD levels are below 20 ng/ml (Dietary Reference Intakes, 2010). No one knows how many aging athletes have low serum 25-OHD levels, but estimates from an older (≥65 years) large Dutch population found that half of the over 1200 men and women studied had levels below 20 ng/ml. Wicherts et al. (2007) found that low levels of vitamin D were associated with poor physical performance and a greater decline in physical performance when compared with age and gender matched controls over a 3-year period.

Houston et al. (2007) used data from the InCHIANTI study (a study of aging in the Chianti region) and found that vitamin D status was inversely associated with poor performance on hand grip strength. About half of the 976 participants in the study had vitamin D deficiency using the IOM criteria. While both of these studies looked at older adults and not aging athletes per se, the concern for decreasing vitamin D levels as aging occurs is real and aging athletes should take steps to insure adequate vitamin D intake. Very few foods are naturally good

sources of vitamin D (with cod liver oil and fatty fish being the exceptions) so choosing vitamin D fortified foods and a supplement that provides 400 IU of vitamin D could help maintain serum vitamin D levels with aging.

Special Considerations

Hydration

Fluid needs of athletes are discussed in Chapters 14–16, but aging athletes have a special challenge to ensure proper hydration. Kenney and Chiu (2001) have shown that when older individuals exercise in warm environmental conditions they experience a decrease in thirst sensation. Aging brings about changes in the systems related to thirst including a higher baseline osmolality and a decreased sensation of thirst in response to both hypo- and hypervolemia. Aging also brings about changes in sweating rates, renal adaptation to altered fluid and electrolytes, and altered blood flow response. In addition, alcohol, caffeine, and theophylline are all mild stimulants which could contribute to dehydration in aging athlete (Binkley et al., 2002). Therefore, aging athletes should not rely on thirst to stay hydrated but should adopt a drinking strategy to get adequate fluids.

Strategies to proper hydration include

- Monitor body weight before and after exercise to assess fluid losses
- Monitor urine volume and color to determine if dehydrated
- Drink fluids before activity and during activity, if exercising in hot humid conditions
- Replace fluid losses after exercise by drinking 1.2–1.5 l of fluids for each kilogram lost
- Eat foods with high water content (fruits and vegetables) and consume fluids with meals

Nutrient–Drug Interactions

It is not known how many aging athletes take over-the-counter or prescription medications but it is safe to say that many older adults take over-the-counter mild pain relievers and many may take prescription drugs for cholesterol-lowering, hypertension, or arthritis. Medication use is so common that many people do not think about the potential for nutrient–drug effects, including effects on hydration, micronutrient status, or exercise performance.

One class of drugs that has widespread use in athletes is the nonsteroidal anti-inflammatory drugs (NSAIDs). These drugs work by inhibiting the synthesis of prostaglandins involved in pain and inflammation. However, prostaglandins also work to protect the lining of the stomach and dilate arteries to the kidney in response to reduced kidney function caused by dehydration and exercise. NSAIDs have been shown to decrease glomerular filtration rate (GFR) during intense exercise (Walter & Lenz, 2011).

Statins are one of the most widely prescribed medications to lower blood cholesterol levels and lower risk of heart disease. Thompson et al. (2003) reviewed statin users' complaints of muscle pain and tenderness and found that a small percentage (1–5%) of patients reported muscle pain with statin use. Rhabdomolysis is even less common in statin users but can be exacerbated by diabetes, hypothyroidism, and disorders that affect the liver and kidney. Another concern with certain statins (atorvastatin, lovastatin, and simvastatin) is the consumption of grapefruit or grapefruit juice with the medication. Grapefruit contains the compound bergamottin which interacts with enzyme systems cytochrome P-450 and P-glycoprotein leading to an increase in the blood level of the statin. It is estimated that grapefruit interacts with over 50 medications so individuals should always learn about the medication prescribed, including how to take it, when to take it, and what to avoid when taking it.

Thiazide diuretics are frequently prescribed for hypertension and mild heart failure. Side effects that could have negative consequences for athletes include frequent urination, dehydration, and urinary potassium losses.

Adding to the concern about side effects of medications is drug testing that occurs in several masters athlete competitions. A tour of various websites reveals a myriad of comments about drug testing in this population; one side believes drug testing is needed and that cheating is rampant in master athletes and others who believe it is totally unnecessary and see it as a form of agism. The most recent

group of athletes to be drug tested occurred at the USMasters Track & Field competition in 2011. Banned substances are the same for younger athletes and a complete list can be found at the World Anti-Doping Agency (WADA) website (http://www.wada-ama.org/en/World-Anti-Doping-Program/Sports-and-Anti-Doping-Organizations/International-Standards/Prohibited-List/). An electronic list can also be send to a mobile phone for quick reference. Prohibited drugs that are of concern to masters athletes include:

- Aromatase inhibitors, including letrozole (Femara) and anastrozole (Arimidex)
- Selective estrogen receptor modifiers (SERMS) including tamoxifen
- Diuretics
- Beta blockers (for certain sports requiring fine motor skills and hand–eye coordination)

Aromatase inhibitors and SERMS are used by breast cancer survivors and breast cancer is the most common cancer in women worldwide (Parkin, 1999). Diuretics and beta-blockers are common medications used to treat hypertension and heart rhythm abnormalities in older adults. Athletes can apply for a therapeutic use exemption (TUE) but many may not think that taking a prescription medication for a diagnosed medical condition is "doping" to gain a competitive advantage over other athletes.

The Australian Anti-Doping Authority has a useful booklet for athletes, "Check Your Substances" available at http://www.asada.gov.au/publications/athlete_guides/asada_guide_Check_Your_Substances_2010.pdf

Dietary Supplement Use

Little is known about dietary supplement use in aging athletes but Striegel et al. (2006) assessed dietary supplement use at the 2004 World Masters Athletic Championships Indoors. About 600 questionnaires were returned from the 1560 distributed (38% return rate) from both male and female athletes. The supplements predominantly used were vitamins (35%), minerals (30%), protein (10%), carbohydrates (9%), and creatine (6%). Omega-3 fatty acids and herbals were reported but in much lower numbers.

Participants said they used supplements for treatment of injury (25%), other health reasons (20%), sports performance (18%), increased endurance (17%), and increased strength (10%). Also reported was to improve stagnating performance and body composition.

Creatine is often used by strength and power athletes to improve training, and research shows that it can enhance lean mass in older adults who undertake strength training. Older adults have lower muscle creatine than younger adults and Brose et al. (2003) studied creatine monohydrate supplementation in healthy men and women over the age of 65. Men and women were randomized to a creatine or placebo supplement (double blinded) and underwent 14 weeks of resistance training. As expected, both groups showed increases in all strength and functional tests, as well as showing increases in muscle fiber area. However, the creatine-supplemented group had significant improvement in several strength exercises compared to placebo and had greater improvement in body composition. The authors concluded that creatine is effective for aging athletes when combined with a progressive resistance program.

Growth hormone promoters sold as dietary supplements have not been systemically evaluated as over-the-counter drugs, but growth hormone is touted as an anti-aging treatment. Liu et al. (2007) conducted a thorough review of randomized control trials using growth hormone and found small changes in body composition but increased rates of adverse events including edema, arthralgias, carpal tunnel syndrome, gynaecomastia, and impaired fasting glucose. Therefore, growth hormone is not recommended as an anti-aging therapy.

Conclusion

Aging athletes can achieve personal best records into old age while improving functional health. While research on various body systems and health outcomes in aging endurance athletes has been published there is very little research on nutritional

Figure 30.3 Women athletes, ranging in age from 60 to 72 years, on a cross-country cycling trip through the Netherlands and Belgium. Courtesy of Christine A. Rosenbloom (2011).

status, nutrient needs, supplement use, and nutritional strategies that could improve performance. Aging athletes could be role models for others in society as a healthy lifestyle to be emulated (see Figure 30.3). It is recommended that masters athletes undergo screening for cardiovascular disease to assess risk and be sure there is no underlying heart disease that could manifest during competition. A statement by the American College of Sports Medicine (2010) recognized the need for medical screening in masters athletes as they are at risk for chest pain (angina), heart attack (myocardial infarction), and arrhythmia. They recommend that athletes with documented coronary artery disease (>50% narrowing as seen on angiography) should not participate in high-intensity sports without further consultation with a physician because of increased risk of myocardial infarction and sudden cardiac death.

However, risk of heart disease should not be a reason for not pursing an active life with competitive sports. As the pioneer of exercise physiology said,

> There is less risk in activity, than in continuous inactivity—it is more advisable to pass a careful physical examination if one intends to be sedentary in order to establish whether one's state of health is good enough to stand the inactivity.
>
> (Astrand, 1986)

References

Amati, F., Dubé, J.J., Coen, D.M., et al. (2009) Physical inactivity and obesity underlie the insulin resistance of aging. *Diabetes Care* 32, 1547–1549.

American College of Sports Medicine. (2010) Selected issues for the master athlete and the team physician: a consensus statement. *Medicine & Science in Sports & Exercise* 42, 820–833.

Andreoli, A., Celi, M., Volpe, S.L., et al. (2012) Long-term effect of exercise on bone mineral density and body composition in post-menopausal ex-elite athletes: a retrospective study. *European Journal of Clinical Nutrition* 66, 69–74.

Astrand, P.O. (1986) Exercise physiology of the mature athlete. In *Sports Medicine for the Mature Athlete* (eds. J.R. Sutton & R.M. Brock), pp. 3–13. Benchmark Press, Indianapolis, IN.

Beshgetoor, D. & Nichols, J.F. (2003) Dietary intake and supplement use in female master cyclists and runners. *International Journal of Sport Nutrition & Exercise Metabolism* 13, 166–172.

Binkley, H.M., Beckett, J., Casa, D.J., et al. (2002) National athletic trainers association's position stand: exertional heat illnesses. *Journal of Athletic Training* 37, 329–343.

Brose, A., Parise, G., & Tarnopolsky, M.A. (2003) Creatine supplementation enhances isometric strength and body composition improvements following strength exercise training in older adults. *Journals of Gerontolology: Biological Sciences* 58, 11–19.

Burke, L.M., Hawley, J.A., Wong, S., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Science* 29, S17–S27.

Cartee, G.D. (1994) Influence of age on skeletal muscle: response to exercise. *Medicine & Science in Sports & Exercise* 26, 577–585.

Chatard, J.C., Boutet, C., Tourny, C., et al. (1998) Nutritional status and physical fitness in elderly sportsmen. *European Journal of Applied Physiology* 77, 157–163.

Cunningham, J.J. (1980) A reanalysis of the factors influencing basal metabolic rate in normal adults. *American Journal of Clinical Nutrition* 33, 2372–2374.

Dietary reference intakes for calcium and vitamin D: report brief. (2010) Institute of Medicine, National Academies of Science, Washington, DC. Available at www.iom.edu/vitamind (accessed October 4, 2011).

Faulkner, J.A., Davis, C.S., Mendias, C.L., et al. (2008) The aging of elite male athletes: age-related changes in performance and skeletal muscle structure and function. *Clinical Journal of Sports Medicine* 18, 501–507.

Fiatarone, M.A., O'Neill, E.F., Ryan, N.D., et al. (1994) Exercise training and nutritional supplementation for physically frailty in very elderly people. *New England Journal of Medicine* 330, 1769–1775.

Franklin, B.A., Fern, A., Voytas, J. (2004) Training principles for elite senior athletes. *Current Sports Medicine Reports* 3, 173–179.

Giada, F., Vigna, G.B., Vitale, E., et al. (1995) Effect of age on the response of blood lipids, body composition, and aerobic power to physical conditioning and deconditioning. *Metabolism* 55, 161–165.

Goldberg, A.P. (2000) Cardiovascular fitness, body composition, and lipoprotein lipid metabolism in older men. *Journals of*

Gerontology: Biological Sciences and Medical Sciences 55, M342–349.

Houston, D.K., Cesari, M., Ferrucci, L., et al. (2007) Association between vitamin D status and physical performance: the InCHIANTI study. *Journals of Gerontology: Medical Sciences* 62A, 440–446.

Institute of Medicine. (2002) *Dietary Reference Intakes for Energy, Carbohydrates, Fiber, Fat, Protein and Amino Acids (Macronutrients)*. National Academies Press, Washington, DC.

International Olympic Committee Consensus Statement on Sports Nutrition (2010) Available at http://www.olympic.org/Documents/Reports/EN/CONSENSUS-FINAL-v8-en.pdf (accessed October 10, 2011).

International Osteoporosis Foundation (2011) Three steps to unbreakable bones on World Osteoporosis Day. Available at http://www.iofbonehealth.org/ (accessed May 8, 2013.

Janssen, I. (2009) Sarcopenia. In *Handbook of Clinical Nutrition and Aging*, 2nd edn (eds. C.W. Bales & C.S. Ritchie), pp. 183–205. Humana Press, New York.

Kenney, W.L., Chiu, P. (2001) Influence of age on thirst and fluid intake. *Medicine & Science in Sports & Exercise* 33, 1524–1532.

Kinsella, K. & He, W. (2009) An Aging World: 2008 International Population Reports. US Census Bureau, US Government Printing Office, Washington DC, http://www.census.gov/prod/2009pubs/p95-09-1.pdf (accessed September 28, 2011).

Lee, I.M., Djoussé, L., Sesso, H.D., et al. (2010) Physical activity and weight gain prevention. *Journal of the American Medical Association* 303, 1173–1179.

Lepers, R. & Cattagni, T. (2012) Do older athletes reach limits in their performance during marathon running? *Age (Dordrecht, The Netherlands)* 34, 773–781.

Liu, H., Bravata, D.M., Olkin, I., et al. (2007) Systematic review: the safety and efficacy of growth hormone in the healthy elderly. *Annals of Internal Medicine* 146, 104–115.

Maron, B.J., Araújo, C.G.S., Thompson, P.D., et al. (2001) Recommendations for preparticipation screening and the assessment of cardiovascular disease in masters athletes. *Circulation* 103, 327–334.

Mifflin, M.D., St Jeor, S.T., Hill, L.A., et al. (1990) A new predictive equation for resting energy expenditure in healthy individuals. *American Journal of Clinical Nutrition* 51, 241–247.

Nieman, D.C., Butler, J.C., Pollett, L.M., et al. (1989) Nutrient intake of marathon runners. *Journal of the American Dietetic Association* 89, 1273–1278.

Parkin, D.M., Pisani, P., & Ferlay, J. (1999) Global cancer statistics. *CA: A Cancer Journal for Clinicians* 49, 33–64. doi:10.3322/canjclin.49.1.33

Power, G., Dalton, B., Behm, D., et al. (2010) Motor unit number estimates in masters runners: use it or lose it? *Medicine & Science in Sports & Exercise* 42, 1644–1650.

Robb, D. (2010) Worldwide obesity stats—2010 and beyond. Available at http://www.hivehealthmedia.com/world-obesity-stats-2010 (accessed October 10, 2011).

Rodriguez, N.R., DiMarco, N.M., Langley, S., et al. (2009) Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *Journal of the American Dietetic Association* 109, 509–527.

Roper, E.A., Molnar, D.J., Wrisberg, C.A. (2003) No "old fool": 88 years old and still running. *Journal of Aging and Physical Activity* 11, 370–387.

Rosenbloom, C. & Coleman, E. (eds) (2012) *Sports Nutrition: A Manual for Professionals*. Academy of Nutrition and Dietetics, Chicago, IL.

Sallinin, J., Ojanen, T., Karavirta, L., et al. (2008) Muscle mass and strength, body composition and dietary intake in master strength athletes vs untrained men of different ages. *Journal of Sports Medicine and Physical Fitness* 48, 190–196.

Striegel, H., Simon, P., Wurster, C., et al. (2006) The use of nutritional supplements among masters athletes. *International Journal of Sports Medicine* 27, 236–241.

Tanaka, H. & Seals, D.R. (2008) Endurance exercise performance in masters athletes: age-associated changes and underlying physiological mechanisms. *Journal of Physiology* 586, 55–63.

Tarnopolsky, M.A. (2008) Nutritional consideration in the aging athlete. *Clinical Journal of Sports Medicine* 18, 531–538.

Thompson, J. & Manore, M.M. (1996) Predicted and measured resting metabolic rate of male and female endurance athletes. *Journal of the American Dietetic Association* 96, 30–34.

Thompson, P.D., Clarkson, P., & Karas, R.H. (2003) Statin-associated myopathy. *Journal of the American Medical Association* 289, 1681–1690.

Tseng, B.Y., Uh, J., Armstrong, K., et al. (2011) Life-long aerobic training preserves white matter integrity: a first look in the masters athlete's brain *Medicine & Science in Sports & Exercise* 43, 127.

Velez, N.F., Zhang, A., Stone, B., et al. (2008) The effect of moderate impact exercise on skeletal integrity in masters athletes. *Osteoporosis International* 19, 1457–1464.

Walter, A.N. & Lenz, T.L. (2011) Hydration and medication use. *American Journal of Lifestyle Medicine* 5, 332–335.

Wicherts, I.S., van Schoor, N.M., Boeke, A.J.P., et al. (2007) Vitamin D status predicts physical performance and its decline in older persons. *Journal of Clinical Endocrinology and Metabolism* 92, 2058–2065.

Wilks, D.C., Winwood, K., Gilliver, S.F., et al. (2009) Bone mass and geometry of the tibia and the radius of masters sprinters, middle and long distance, race-walkers and sedentary control participants: A pQCT study. *Bone* 45, 91–97.

Wright, V.J. & Perricelli, B.C. (2008) Age-related rates of decline in performance among elite senior athletes. *American Journal of Sports Medicine* 36, 443–450.

Chapter 31

The Vegetarian Athlete

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Introduction

All athletes, vegetarian or nonvegetarian, need to consume a diet that includes a wide variety of foods in moderation from all food groups in order to obtain the essential nutrients needed for health and sports performance. The risks of deficiency for any athlete becomes greater when foods groups are eliminated or when certain foods are restricted or avoided or if a few foods comprise a large part of the total intake. To date, there is no available evidence to support either a beneficial or a detrimental effect of a vegetarian diet on human performance. Some data do exist on elite athletes who consume a vegetarian diet but studies that include elite or Olympic caliber athletes are limited. Regardless, both vegetarian and nonvegetarian athletes will have poor sports performance when either group makes poor nutrition choices or fails to consume nutrients that are essential for both health and athletics.

Vegetarian Advantage

Although a vegetarian diet has not been found to benefit athletic performance, it does offer several health benefits which has caught the attention and interests of both athletes and consumers. Results from a 2011 telephone survey conducted by the Vegetarian Resource Group found that approximately 5% of the American population never eats meat, fish, seafood, or poultry. About half of these self-reported vegetarians are vegans, which means they do not consume eggs or dairy products either. Additionally, the survey found that nearly one-third of Americans do not eat meat, fish, seafood, or poultry at many of their meals or at more than half of their meals (Stahler, 2011). Clearly, initiatives such as Meatless Monday and the increase in vegetarian web sites, cookbooks, and the availability of vegetarian meals in grocery stores and restaurants are helping fuel the popularity of a vegetarian diet. Furthermore, many national and international organizations such as the World Cancer Research Fund, the Heart and Stroke Foundation of Canada, the American Cancer Society, and the American Heart Association are recommending the consumption of a plant-based diet focused on fruit and vegetables, whole grains, and limited animal products for a reduction in lifestyle diseases. Internationally, the incidence of vegetarianism is similar or higher than that in the United States. In Canada, it appears that 1 out of 25 is classified as vegetarian; the United Kingdom has reported rates between 7% and 11%, while India reports between 20% and 40% vegetarianism.

Classifications of Vegetarian Diets

Vegetarian diets range from the vegan diet (excludes all animal products), to the semi-vegetarian diet (may include animal products) (Table 31.1).

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Table 31.1 Classification of various vegetarian diets

Diet	Food pattern
Semi-vegetarian	Avoids some but not all groups of animal-derived food. Red meat is most often excluded or consumed in limited amounts
Lacto-ovo vegetarian	Consumes milk and milk products as well as eggs. Avoids meat, poultry, fish, and seafood
Ovo vegetarian	Eggs are included but meat, poultry, fish, seafood, and dairy products are avoided
Lacto vegetarian	Dairy products are included but does not eat meat, poultry, fish, seafood, or eggs
Vegan	Avoids all animal-derived foods including meat, poultry, fish, seafood, eggs, milk, and milk products
Macrobiotic	Avoids all animal foods and focuses on unprocessed, unrefined, natural, and organic foods

Whatever term is used, it appears that vegetarianism is a continuum of eating patterns, which range from the sole consumption of plant foods to a diet restricting certain kinds of animal products or limiting the frequency of animal protein consumption (ADA, 2009b). Recently, other terminology or definitions have been introduced to describe or define various vegetarian-eating styles. For example, Fuhrman and Ferreri (2010) define *vegetarian* as those who eat no animal flesh, but may consume eggs and dairy; *flexitarian* describes those who regularly follow a vegan diet but occasionally consume dairy, meat, fowl, or eggs; and lastly *nutritarian* describes individuals who follow an eating style high in micronutrients, based on unrefined plant foods and may or may not be vegans.

Vegetarian Diets and Performance

Several reviews have been published investigating the effects of a vegetarian diet on human performance (Barr & Rideout, 2004; Nieman, 1988, 1999; Venderley & Campbell, 2006). All four publications have basically concluded that a vegetarian diet is neither beneficial nor detrimental to training and human performance. Despite these conclusions, a popular belief exists that a meatless diet may be beneficial for athletes. The popularity of vegetarian diets in athletes has been fuelled by numerous professional and lay articles touting the benefits of vegetarian diets and highlighting the success stories of vegetarian athletes such as triathletes Dave Scott and Brendan Brazier, long-distance runner Pavo Nurmi, American football player Tony Gonzales, Olympic track athlete Carl Lewis, and body builder Kenneth Williams as evidence that an athlete can perform on a vegetarian diet. Despite great performances from these vegetarian athletes, data do not support a performance benefit from consumption of a vegetarian diet.

Early research on vegetarian athletes included studies done by Cotes et al. (1970), Hanne et al. (1986), and Raben et al. (1992). Cotes et al. (1970) investigated the effect of a vegan diet on physiological responses to submaximal exercise in 14 nonathletic females who had consumed a vegetarian diet for an average of more than 11 years and compared them with two different control groups: 66 sedentary, nonvegetarian females whose social backgrounds were similar to the vegans and a group of 20 active women who were nonvegetarian and had similar physical activity to the vegans. All subjects performed a submaximal test on a cycle ergometer in which they cycled for 3 minutes at 30 and 60 W. Ventilation and cardiovascular response to submaximal exercise on a cycle ergometer were measured as well as the width of muscles in the thigh. The authors found no statistical differences between the vegans when compared to the nonvegetarians.

Hanne et al. (1986) also investigated various fitness parameters of male and female vegetarian athletes and compared them with nonvegetarians. The vegetarian subjects include those who followed various vegetarian diets, including lacto, lacto-ovo, and vegan. Fitness parameters included anthropometric measurements, pulmonary function, aerobic and anaerobic capacity, and blood chemistries. The authors found no significant differences in body mass between the two groups, although the female vegetarians had a significantly (p >0.01) higher percentage body than their nonvegetarian counterparts. No differences were found in pulmonary

function, heart rate, and blood pressure or in the electrocardiogram. Furthermore, there were no differences in the subjects' aerobic capacity or anaerobic capacity as determined from a submaximal test and a Wingate test, respectively. Blood chemistries found that nonvegetarian controls had lower uric acid levels than the vegetarian males, but the nonvegetarian group was within the normal range. Vegetarian women had lower hematocrit values than controls, but hemoglobin, total protein, and glucose were similar in both groups. Again, the authors concluded that, as no differences were found between the groups, consuming a vegetarian diet does not impair human performance.

Lastly, Raben et al. (1992) studied the effects of endurance performance in male athletes who consumed a lacto-ovo vegetarian diet and compared them to endurance athletes who consumed meat on a mixed diet. The study consisted of eight trained athletes with a mean VO_2 max of 67 ml/kg/min. They were fed a lacto-ovo vegetarian diet for 6 weeks and mixed diet with meat for 6 weeks. Using a cycle ergometer, an endurance test was performed on weeks 0, 3, and 6 after consuming each diet. No significant changes in endurance performance were found on either diet.

While these studies have concluded that there is no performance benefit of a vegetarian diet, they have either used populations that are not representative of athletes or used subjects that are not habitual vegetarians. In theory, when athletes follow a vegetarian diet they should be consuming more carbohydrates and, if planned well, could meet or exceed recommendations for other macronutrients, micronutrients, and energy that would match the ideal or recommended diet for training and recovery. Clearly, more studies need to be performed in trained athletes who are habitual vegetarians to determine if any training or performance benefits exist.

Antioxidant and Exercise Performance

Vegetarian diets have been shown to offer a number of nutritional benefits, including lower levels of saturated fat, cholesterol, and animal protein as well as higher levels of carbohydrates, fiber, magnesium, potassium, folate, carotenoids, flavonoids, and other phytochemicals (ADA, 2009b). Furthermore, vegetarians have been reported to have a lower body mass and lower rates of death from heart disease. Vegetarians typically have lower blood cholesterol and blood pressure and are at less risk of type 2 diabetes and prostate and colon cancer than omnivore counterparts (ADA, 2009b).

Furhman and Ferreri (2010) proposed that serious athletes who adopt a vegetarian diet may avoid the immunosuppression that occurs in heavy training due to their consumption of more plant-based foods. Furthermore, the authors suggested that a vegetarian diet high in phytonutrients and antioxidants may attenuate the exercise-induced oxidative stress that occurs in athletes.

There is increasing evidence to show production of lipid peroxidation products and free radicals during physical exertion (Simon-Schnass & Pabst, 1988) and that reactive oxygen species (ROS) increase when the exercise is intense and prolonged (Leaf et al., 1997). While oxidative stress increases in response to training, studies have shown that well-trained athletes have endogenous adaptive mechanisms that improve the body's ability to defend against increased production of ROS during exercise (Watson, 2010).

Consumption of a diet rich in plant foods with high antioxidant content, such as a vegetarian diet, could minimize the potential for free radical damage and may offer advantages to an athlete's training and health. However, more data are needed to determine how antioxidants from food defend against oxidative stress induced by exercise and certainly more studies need to be conducted using habitual vegetarian athletes to determine if there is a performance benefit when compared to nonvegetarian athletes. Lastly, recommendations from scientific data confirm that athletes should consume more antioxidants in their diet, mostly from food as high dosages of antioxidant supplements may have a pro-oxidative effect and do more damage than good.

Creatine and Vegetarian Athletes

Creatine is a nitrogenous compound found in the muscle where it becomes phosphorylated producing phosphocreatine (PCr). The role of PCr is

to provide a rapid but brief source of phosphate to adenosine diphosphate (ADP) to form adenosine triphosphate (ATP) during maximal exercise. Thus, creatine is important for short bursts of high-intensity exercise like sprinting or during multiple bouts of high-intensity exercise such as soccer. Many studies (Branch, 2003; Rawson & Volek, 2003) have found that elevated levels of muscle creatine increase the rate at which phosphocreatine is resynthesized during recovery from exercise and may enhance exercise performance and recovery time during short, repeated bouts of maximal exercise. For this reason, many athletes may consider using creatine as a supplement.

The body turns over about 2 g of creatine per day and metabolizes creatine to creatinine, which is excreted by the kidneys. Vegetarians have been shown to have a lower body creatine pool than nonvegetarians, presumably due to their lower intake of dietary creatine (Lukaszuk et al., 2002). Nonvegetarians typically receive about 1 g of creatine/day through meat consumptions while both vegetarians and nonvegetarians synthesize about 1 g/day from the amino acids arginine, methionine, and glycine.

Several studies have investigated muscle creatine levels of vegetarians and compared them to their omnivore counterparts. Shomrat et al. (2000) investigated the effect of creatine supplementation on anaerobic performance by supplementing seven vegetarians and nine omnivores with 21 g of creatine per day for 6 days. Anaerobic power was measured during three 20-second maximal cycling tests. Both groups experienced an increase in anaerobic performance with no difference between groups. In contrast, Burke et al. (2003) compared changes in muscle creatine and resistance training by randomly assigning 18 vegetarian and 24 nonvegetarian men and women to receive either a placebo or a creatine supplement for 7 days with 0.25 g of creatine/kg of lean mass and 49 days of a maintenance dose of 0.0625 g of creatine/kg of lean muscle mass. The subjects participated in an 8-week resistance-training program. The researchers reported that total muscle creatine content was significantly lower in the vegetarian group than in the nonvegetarians at baseline. However, after supplementation those subjects who consumed the creatine supplement had a greater increase in resistance training and lean tissue than the placebo group. Additionally, they reported that vegetarians who took creatine had a greater increase in muscle creatine and work performance than nonvegetarians who took the creatine supplement. They concluded that vegetarians with initially low levels of muscle creatine might be more responsive to supplementation. Lastly, Lukaszuk et al. (2005) found that when subjects switched from an omnivorous diet to a lacto-ovo vegetarian diet, they had a reduction in muscle creatine. Subjects consisted of 16 men who consumed a lacto-ovo vegetarian diet for 3 weeks and 16 men who continued to consume an omnivorous diet. Both diets had comparable quantities of dietary protein. After establishing a lowered creatine level in the vegetarian group, the researchers then supplemented both groups with 0.3 g creatine/kg/day and 20 g of Polycose for 5 days. They found that both groups increased total muscle creatine with no difference between the vegetarian and nonvegetarian groups. The authors concluded that unlike glycogen supercompensation, lowering creatine levels by consuming a vegetarian diet and then supplementing with a high dose of creatine is not a viable strategy to achieve a higher than normal concentration of muscle creatine.

These studies show that vegetarian athletes may benefit from consuming a creatine supplement for high-intensity sports that rely heavily on ATP stores. While long-term consequences of creatine supplementation are unknown, there are anecdotal reports of muscle cramps, strains, and tears; however, scientific studies have not confirmed these concerns. Additionally, there is a weight gain associated with creatine supplementation for some individuals. Weight gain may be counterproductive in sports where a power to weight ratio is important or when an athlete needs to make a certain weight classification to compete. Finally, most athletes do not know how much creatine they should consume. They, along with most consumers, believe that if a little is good, more has got to be better. Higher doses of creatine do not further enhance creatine stores and the unintended consequences of consuming high doses of creatine are unknown. Further details of the effects of creatine supplementation can be found in Chapter 24.

Nutritional Adequacy of Vegetarian Diets

Protein Quality

A nutritionally sound vegetarian diet is possible, but there are certain nutrients that vegetarians must be aware of and plan for in order to ensure an adequate intake. The most obvious nutrient of concern is protein. Lacto-ovo vegetarians receive high-quality complete proteins and are unlikely to incur protein deficiencies. However, vegetarians who ingest only plant proteins need to consume a variety of plant foods in order to meet both their protein and energy requirements. Plant proteins are classified as incomplete proteins and lack one or more essential amino acids. While it was once thought that all the amino acids must be consumed in one meal, it is now agreed that the timing of the amino acid intake must be balanced over days rather than hours (Young & Pellet, 1994). It is recommended that vegetarians complement their proteins since they do not contain all the essential amino acids. For example, cereals are very low in the essential amino acid lysine, while legumes are slightly deficient in the sulfur-containing amino acids, such as methionine. By combining these two groups of foods, a vegetarian can provide a mixture of amino acids similar to that of a complete or high-quality protein food. Table 31.2 lists different combinations of incomplete proteins that will make complete proteins. Note that the amino acid lysine is low in two of the three plant sources of protein; therefore, vegetarians need to consume healthy servings of legumes and beans to meet their complete protein needs. Also, quinoa, usually considered to be a whole grain, is actually a seed that provides all nine essential amino acids. The same is true of soy. Soy, soybeans, and tofu contain all nine essential amino acids. Vegetarians who consume only plant proteins can easily add quinoa and soy to their meals for a great source of complete proteins.

Protein Requirements for Vegetarian Athletes

While most vegetarians meet or exceed recommendations for total protein intake, their diets tend to be lower in protein than those of omnivores (ADA, 2009a). Additionally, plant proteins tend be less bioavailable and several papers suggest that vegetarian athletes, who consume most of their protein from plant sources, should increase their protein intake by about 10% (ADA, 2009a; Barr & Rideout, 2004). The suggested range of protein intake for vegetarian athletes is 1.3–1.8 g/kg/day (ADA, 2009a). This recommendation is for vegetarian athletes who meet their energy requirements, as there is a relationship between energy balance and protein utilization. For example, if an athlete trains or competes in an energy-deficit state, then protein utilization is higher and protein intake should increase correspondingly. Therefore, it is critical that vegetarians consume sufficient energy to support not only training and performance, but growth and maintenance of lean tissue as well as immune and reproductive functions.

An interesting case study involving a young vegetarian was published by Borrione et al. (2009). This case involved a young vegetarian swimmer who

Table 31.2 Limiting amino acids in plant sources of protein

Food group	Primary limiting amino acid	To make a complete protein, combine with	Complementary food protein combinations
Legumes (peanuts, dry beans such as navy black, pinto, and kidney beans)	Methionine, tryptophan	Grains, nuts, or seeds	Hummus and pita bread, bean burrito and corn tortilla, barley and bean soup
Nuts and seeds (cashews, walnuts, almonds, and sunflower seeds)	Lysine, isoleucine	Legumes	Pasta with sesame seeds, peanut butter sandwich, bean soup with sunflower seeds
Grains (wheat, rice, oats, and corn)	Lysine, isoleucine, threonine	Legumes	Beans and rice, lentil soup, and cornbread

was diagnosed with rhabdomyolysis. The swimmer reported that he avoided eating meat, fish, eggs, legumes, cheese and he also limited his intake of dairy products. His only significant source of protein was 100 ml of milk in the morning and soy-derived products once or twice a week. Blood tests showed CK level of 9952 units/l, while AST and ALT levels were 159 units/l and 69 units/l, respectively, along with low ferritin and vitamin B_{12} levels. He was instructed to introduce protein into his diet and supplemental vitamin B_{12} was prescribed. At his 3-month follow-up, the patient had introduced meat into his diet four times a week and some dairy products, and his CK values were within the reference range as were ferritin and $B_{12.}$ Twenty months after the episode, the patient remained well and continued to swim at national level.

Vitamin B_{12}

Vegetarians who consume only plant-based diets tend to have low intakes of vitamin B_{12}. Concentrated sources of vitamin B_{12} are found in animal products and therefore vegetarians who restrict animal proteins do not get a reliable source of B_{12} in their diet. Deficiencies lead to microcytic anemia, which is associated with decreased oxygen delivery and therefore will impair athletic performance. Reliable sources of B_{12} for vegetarians who avoid animal protein include B_{12}-fortified foods such as fortified soy and rice products, fortified cereals, or fortified yeast products (ADA, 2009b). If these products are not consumed regularly then a B_{12} supplement is recommended.

Iron

Iron is classified as an essential nutrient and is required for the formation of hemoglobin and myoglobin, as well as cytochromes, which are components of the electron transport chain (see Chapter 19). Iron is also a cofactor for a number of enzymatic reactions including those involved in the synthesis of collagen, various neurotransmitters, and the Krebs cycle. The incidence of iron-deficiency anemia is similar to that of nonvegetarians (Messina et al., 2011), but due to the low bioavailability of nonheme plant sources, vegetarians have lower iron stores than omnivores (ADA, 2009b).

Since iron plays a critical role in oxidative metabolism, it is essential for athletes to have adequate stores. There has been some controversy in the past whether low iron stores without anemia impair exercise performance. Recent studies by Brownlie et al. (2002, 2004) and Hinton et al. (2000) using a relatively new, reliable, and precise marker of early iron depletion have confirmed that low iron stores reduced endurance and aerobic capacity in untrained women with iron depletion. Furthermore, Hinton and Sinclair (2007) found similar effects in iron-depleted recreational athletes after supplementation, although this does not seem to be the case with all studies. Peeling et al. (2007) recruited iron-depleted female athletes and found no effect of an injection of iron on aerobic capacity.

The low bioavailability of nonheme iron is most likely due to inhibitors such as phytates present in legumes, rice, and grains and polyphenols found in tea, coffee, herbs, and wine. Despite these limiting absorption factors, the absorption rate of nonheme iron can be increased when they are consumed with ascorbic acid or using food preparation techniques such as soaking or sprouting beans, grains, seeds, and the leavening in breads (rising of the bread dough) to reduce phytate (ADA, 2009b). The IOM and Food and Nutrition Board (2001) state that due to the decreased iron bioavailability from nonheme foods, the recommended iron intake is 1.8 times higher for vegetarians. Furthermore, vegetarian athletes should be monitored for iron status when taking iron supplements, especially during periods of rapid growth that occur during adolescence and pregnancy (ADA, 2009a).

Zinc

Zinc is a component of over 1000 enzymes and is involved in immune function, protein synthesis, and blood formation. Zinc absorption is similar to iron absorption in that phytates, oxalates, and tannins inhibit zinc absorption. The zinc intake of vegetarians appears to be variable. Some studies find zinc status from vegetarian diets to be similar

to nonvegetarian (Alexander et al., 1994; Davey et al., 2003), while others found zinc intakes to be significantly lower than nonvegetarians (Ball & Bartlett, 1999; Janelle & Barr, 1995). The variations in these studies are most likely due to the amount of phytate, fiber, calcium, or other inhibitors of zinc absorption in vegetarian diets (Hunt, 2003). In most of these studies, intakes of Zn and phytate were measured and the phytate to Zn molar ratio calculated and compared to plasma Zn levels. It appears that plasma Zn is inversely related to the phytate and Zn molar ratios, especially if the ratio is >15, which might account for the differences in results of the studies above. Additionally, others have found that those with diets high in refined grains (lower Zn concentrations) have lower plasma Zn levels than those who consume whole grains (higher Zn concentrations), but as the phytate to Zn molar ratio approaches or exceeds 15, Zn is less bioavailable. Some research says that at these ratios and above, vegetarians may require 50% more Zn than nonvegetarians. Nonetheless, food preparation such as soaking and sprouting beans, grains, and seeds as well as leavening in bread can reduce the binding of zinc by phytic acid.

Calcium

Lacto-ovo vegetarians appear to have similar if not higher intakes of calcium than nonvegetarians (Tesar et al., 1992), whereas vegans, who avoid dairy products, tend to be lower than both groups and often fall below the recommended intake (Haddad et al., 1999; Messina et al., 2011). As with iron and zinc, the absorption of calcium may be reduced by phytates, oxalates, fiber, and tannins (James et al., 1978; Weaver et al., 1999). Phytic acid is found in oatmeal and other whole grain cereals, while oxalates are commonly found in beets, spinach, and Swiss chard. Additionally, sesame seeds, almonds, and dried beans also have lower bioavailability of calcium than low-oxalate greens like bok choy, broccoli, Chinese cabbage, collards, and kale and fruit juices fortified with calcium, which are good sources of highly bioavailable calcium (Weaver et al., 1999). Research also points out that high-protein and high-sodium diets alter calcium balance negatively, while a diet high in fruits and vegetables and rich in potassium and magnesium tends to offset the calciuric effects of a diet high in meat and sodium and slow bone calcium turnover and urinary calcium loss (ADA, 2009a; Craig, 2010). A study of postmenopausal women found significantly lower urinary calcium excretion in those following a vegetarian diet compared to an omnivorous diet (3.2 ± 1.2 vs. 3.9 ± 1.3 mmol/24 h), despite similar dietary calcium intake (Ball & Maughan, 1997). Weaver et al. (1999), Messina et al. (2011), and Cox (2001) point out that vegans could meet calcium requirements if their diet regularly included foods fortified with calcium such as soy, soy milk, fruit juices, rice milk, and cereals as they can significantly contribute to dietary calcium.

Osteoporosis One consequence of not consuming adequate calcium is the risk of bone fractures and osteoporosis. Indeed, the EPIC-Oxford study (Appleby et al., 2007) found that the risk of bone fracture in meat eaters and lacto-ovo vegetarians was similar, but vegans had a 30% higher risk of fracture, possibly due to their low calcium intake. However, when vegans consumed more than 535 mg of calcium/day their fracture rate was not significantly different from that of omnivores.

All styles of vegetarian eating are encouraged to consume adequate levels of calcium to lower the risk of osteopenia and ultimately osteoporosis. Increased calcium consumption through low-fat dairy products, calcium-fortified food products, or a calcium supplement is strongly encouraged. Table 31.3 lists nutritional strategies for increasing vitamin $B_{12,}$ iron, zinc, and calcium for vegetarians.

Amenorrhea and Vegetarian Diets

A few studies have indicated that menstrual dysfunction may be higher in vegetarian athletes (Brooks et al., 1984; Pedersen et al., 1991; Slavin et al., 1984). Brooks compared the diets of amenorrheic runners (82% vegetarian) with those of regularly menstruating runners (13% vegetarian) and found that the runners who had regular menstrual cycles ate five times more meat and significantly more fat than the amenorrheic runners. Slavin et al. (1984) also found a high incidence of vegetarianism

Table 31.3 Nutritional strategies for vegetarians to improve micronutrient status

Nutrient	Physiological role	Food sources for vegetarians
Vitamin B_{12}	Activates folate, maintains the myelin sheath around nerve fibers, promotes growth, and functions as a coenzyme	Vegetarian support formula, nutritional yeast, fortified soy and rice products, and fortified cereals. Eggs and milk and milk products
Iron	Required for synthesis of hemoglobin and myoglobin, cofactor for many enzymatic reactions	Leafy green vegetables like bok choy, spinach, beans, fortified cereals like oatmeal, and fortified soy products. Consume a good source of vitamin C for better absorption
Calcium	Required for muscle contractions, blood clotting, nerve transmission, and blood pressure; major component of bones and teeth	Low-fat milk and dairy products, broccoli, kale, and spinach and calcium-fortified foods like orange juice, cereals, soy products, and soy and rice milk
Zinc	Part of over 1000 enzymes; involved in the synthesis of DNA and RNA; involved in the immune system, wound healing, and blood formation	Beans, nuts, seeds, peas, and fortified cereals and soy products

among amenorrheic athletes while Pedersen et al. (1991) reported that the prevalence of menstrual irregularities among 41 nonvegetarian women was 5%, compared to 27% among 34 vegetarian women. In contrast to these studies, Hanne et al. (1986) and Barr et al. (1994) reported no differences in menstrual irregularities among female lacto-ovo vegetarians, lacto vegetarians, and nonvegetarians.

Some female athletes who consume no meat and adopt a vegetarian diet may be attempting to mask their dietary behavior, which could be a warning sign of an eating disorder (Martins et al., 1999). However, not all women who become vegetarians have menstrual irregularities or are masking unhealthy weight-control behaviors. Clearly, menstrual dysfunction is a multifactorial issue and may include many factors such as adopting a vegetarian diet, negative energy balance, prolonged training, and emotional stress. More research is needed in this area.

Conclusions

To date there is no available evidence to support either a beneficial or a detrimental effect of a vegetarian diet on human performance. While some data do exist, no long-term studies have been conducted using habitual vegetarian athletes to determine if any training benefits exist. Despite the lack of performance studies, the scientific literature is replete with studies that show positive health benefits from consuming a nutritionally well-planned vegetarian diet. One of the benefits for vegetarian athletes is that they consume a higher percentage of energy from carbohydrates including more fruits and vegetables, which may minimize the potential for free radical damage and may offer advantages to an athlete's training and health.

Vegetarian athletes can meet their nutrient and energy needs by choosing a wide variety of plant foods, but several essential nutrients are of concern. Protein quality is a potential concern for vegetarians, especially for those who avoid all animal products. While most vegetarian athletes meet or exceed their protein requirements, some individuals may need to increase their overall protein intake. At the forefront of nutritional concern for vegetarians are vitamin B_{12}, iron, zinc, and calcium. Deficiencies in any of these nutrients will result in poor performance. Routine monitoring of these nutrients is recommended. Food records may show adequate intake but blood levels may be low due to the poor bioavailability of nonheme plant foods.

Vegetarians, in general, have lower stores of muscle creatine, thus high-intensity, sprint-like performances may be affected. Some studies have shown that vegetarians who were supplemented with creatine may see an improvement in performance, but performance was not better when compared to omnivores.

Clearly, more research is needed on habitual female vegetarians and the effects of diet on

hormonal status. Additionally, some women (and some men) may use a vegetarian diet as a means of restricting energy intake or to mask an eating disorder and so need to be monitored.

Lastly, the variability of a vegetarian diet makes assessing nutrient intake and nutrient status essential. Sport dietitians can play a key role in assessing dietary intake, educating vegetarians on menu planning, cooking, and food preparation for maximum performance.

Sources for More Information

The Vegetarian Resource Group
http://www.vrg.org

The Vegetarian Society of the United Kingdom
http://www.vegsoc.org/health

International Vegetarian Union
http://www.ivu.org/

References

Alexander, D., Ball, M.J., & Mann, J. (1994) Nutrient intake and haematological status of vegetarians and age-sex matched omnivores. *European Journal of Clinical Nutrition* 48, 538–546.

American Dietetic Association [ADA] (2009a) Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *Journal of the American Dietetic Association* 109(3), 509–527.

American Dietetic Association [ADA] (2009b) Position of the American Dietetic Association: vegetarian diets. *Journal of the American Dietetic Association* 109(7), 1266–1282.

Appleby, P., Roddam, A., Allen, N., et al. (2007) Comparative fracture risk in vegetarians and nonvegetarians in EPIC-Oxford. *European Journal of Clinical Nutrition* 61, 1400–1406.

Ball, M.J. & Bartlett, M.A. (1999) Dietary intake and iron status of Australian vegetarian women. *American Journal of Clinical Nutrition* 69, 944–952.

Ball, D. & Maughan, R.J. (1997) Blood and urine acid–base status of premenopausal omnivorous and vegetarian women. *British Journal of Nutrition* 78, 683–693.

Barr, S.I. & Rideout, C.A. (2004) Nutritional considerations for vegetarian athletes. *Nutrition* 20, 696–703.

Barr, S.I., Janelle, K.C., & Prior, J.C. (1994) Vegetarian vs nonvegetarian diets, dietary restraint, and sub-clinical ovulatory disturbances: prospective 6-mo study. *American Journal of Clinical Nutrition* 60, 887–894.

Borrione, P., Spaccamiglio, A., Salvo, R.A., et al. (2009) Rhabdomyolysis in a young vegetarian athlete. *American Journal of Physical Medicine & Rehabilitation* 88, 951–954.

Branch, J.D. (2003) Effect of creatine supplementation on body composition and performance: a meta analysis. *International Journal of Sport Nutrition and Exercise Metabolism* 13, 198–226.

Brooks, S.M., Sanborn, C.F., Albrecht, B.H., et al. (1984) Diet in athletic amenorrhoea [letter]. *Lancet* 1, 559–560.

Brownlie, T.I., Utermohlen, V., Hinton, P.S., et al. (2002) Marginal iron deficiency without anemia impairs aerobic adaptation among previously untrained women. *American Journal of Clinical Nutrition* 75, 734–742.

Brownlie, T.I., Utermohlen, V., Hinton, P.S., et al. (2004) Tissue iron deficiency without anemia impairs adaptation in endurance capacity after aerobic training in previously untrained women. *American Journal of Clinical Nutrition* 79, 437–443.

Burke, D.G., Chilibeck, P.D., Parise, G., et al. (2003) Effect of creatine and weight training on muscle creatine and performance in vegetarians. *Medicine & Science in Sports & Exercise* 35(11), 1946–1955.

Cotes, J.E., Dabbs, J.M., Hall, A.M., et al. (1970) Possible effect of a vegan diet upon lung function and the cardiorespiratory response to submaximal exercise in healthy women. *Journal of Physiology* 209, 30–32.

Cox, G.R. (2001) The vegetarian athlete. *International SportMed Journal* 2(2), 1–8.

Craig, W.J. (2010) Nutrition concerns and health effects of vegetarian diets. *Nutrition in Clinical Practice* 25, 613–620.

Davey, G.K., Spencer, E.A., Appleby, P.N., et al. (2003) EPIC-Oxford: lifestyle characteristics and nutrient intakes in a cohort of 33,883 meat-eaters and 31, 546 non-meat eaters in the UK. *Public Health Nutrition* 6, 259–268.

Fuhrman, J. & Ferreri, D.M. (2010) Fueling the vegetarian (vegan) athlete. *Current Sports Medicine Reports* 9(4), 233–241.

Haddad, E.H., Berk, L.S., Kettering, J.D., et al. (1999) Dietary intake and biochemical, hematologic and immune status of vegans compared with non-vegetarians. *American Journal of Clinical Nutrition* 70, 586s–593s.

Hanne, N., Dlin, R., & Rotstein, A. (1986) Physical fitness, anthropometric and metabolic parameters in vegetarian athletes. *Journal of Sports Medicine and Physical Fitness* 26, 180–185.

Hinton, P.S. & Sinclair, L.M. (2007) Iron supplementation maintains ventilator threshold and improves energetic efficiency in iron-deficient nonanemic athletes. *European Journal of Clinical Nutrition* 61, 30–39.

Hinton, P.S., Giordano, C., Brownlie, T., et al. (2000) Iron supplementation improves endurance after training in iron-depleted, nonanemic women. *Journal of Applied Physiology* 88, 1103–1111.

Hunt, J.R. (2003) Bioavailability of iron, zinc, and other trace minerals from vegetarian diets. *American Journal of Clinical Nutrition* 78, 633s–639s.

Institute of Medicine [IOM] & Food and Nutrition Board (2001) *Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc*. National Academies Press, Washington, DC.

James, W.P., Branch, W.J., & Southgate, D.A. (1978) Calcium binding by dietary fibre. *Lancet* 1, 638–639.

Janelle, K.C. & Barr, S.I. (1995) Nutrient intakes and eating behavior scores of vegetarian and nonvegetarian women. *Journal of the American Dietetic Association* 95, 180–189.

Leaf, D.A., Kleinman, M.T., Hamilton, M., et al. (1997) The effect of exercise intensity on lipid peroxidation. *Medicine & Science in Sports & Exercise* 8, 1036–1039.

Lukaszuk, J.M., Robertson, R.J., Arch, J.E., et al. (2002) Effect of creatine supplementation and a lacto-ovo-vegetarian diet on muscle creatine concentration. *International Journal of Sport Nutrition and Exercise Metabolism* 12(3), 336–348.

Lukaszuk, J.M., Robertson, R.J., Arch, J.E., et al. (2005) Effect of a defined lacto-ovo-vegetarian diet and oral creatine monohydrate supplementation on plasma creatine concentration. *Journal of Strength and Conditioning Research* 19(4), 735–740.

Martins, Y., Pliner, P., & O'Connor, R. (1999) Restrained eating among vegetarians: does a vegetarian eating style mask concerns about weight? *Appetite* 32, 145–154.

Messina, V., Mangels, R., & Messina, M. (2011) *The Dietitian's Guide to Vegetarian Diets: Issues and Applications*, 3rd edn. Jones & Bartlett, Burlington, MA.

Nieman, D.C. (1988) Vegetarian dietary practices and endurance performance. *American Journal of Clinical Nutrition* 48, 754s–761s.

Nieman, D.C. (1999) Physical fitness and vegetarian diets: is there a relation? *American Journal of Clinical Nutrition* 70, 570s–573s.

Pedersen, A.B., Bartholomew, M.J., Dolence, L.A., et al. (1991) Menstrual differences due to vegetarian and non-vegetarian diets. *American Journal of Clinical Nutrition* 53, 879–885.

Peeling, P., Blee, T., Goodman, C., et al. (2007) Effect of iron injections on aerobic exercise performance of iron depleted female athletes. *International Journal of Sports Nutrition and Exercise Metabolism* 17, 221–231.

Raben, A., Kiens, B., Richter, E.A., et al. (1992) Serum sex hormones and endurance performance after a lacto-ovo vegetarian and a mixed diet. *Medicine & Science in Sports & Exercise* 24(11), 1290–1297.

Rawson, E.S. & Volek, J.S. (2003) Effects of creatine supplementation and resistance training on muscle strength and weightlifting performance. *Journal of Strength and Conditioning Research* 17, 822–831.

Shomrat, A., Weinstein, Y., & Katz, A. (2000) Effect of creatine feeding on maximal exercise performance in vegetarians. *European Journal of Applied Physiology* 82(4), 321–325.

Simon-Schnass, I. & Pabst, H. (1988) Influence of vitamin E on physical performance. *International Journal for Vitamin and Nutrition Research* 58, 49–54.

Slavin, J., Lutter, J., & Cushman, S. (1984) Amenorrhoea in vegetarian athletes [letter]. *Lancet* 1, 1474–1475.

Stahler, C. (2011) How often do Americans eat vegetarian meals? And how many adults in the US are vegan? *The Vegetarian Resource Group*. Available at: http://www.vrg.org/journal/vj2011issue4/vj2011issue4poll.php (accessed December 30, 2011).Tesar, R., Notelovitz, M., Shim, E., et al. (1992) Axial and peripheral bone density and nutrient intakes of postmenopausal vegetarian and omnivorous women. *American Journal of Clinical Nutrition* 56, 699–704.

Venderley, A.M. & Campbell, W.W. (2006) Vegetarian diets: nutritional considerations for athletes. *Sports Medicine* 36, 293–305.

Watson, T. (2010) The science of anti-oxidants and exercise performance. In *Clinical Sports Nutrition*, 4th edn (eds. L. Burke & V. Deakin), pp. 295–303. McGraw Hill, Australia.

Weaver, C.M., Prouix, W.R., & Heaney, R. (1999) Choices for achieving adequate dietary calcium with a vegetarian diet. *American Journal of Clinical Nutrition* 70, 543s–548s.

Young, V.R. & Pellett, P.L. (1994) Plant proteins in relation to human protein and amino acid nutrition. *American Journal of Clinical Nutrition* 59, 1203s–1212s.

Chapter 32

The Special Needs Athlete

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Introduction

As is the case with scientific research in nutrition and exercise, the majority of the information presented in this Sports Nutrition text has been about able-bodied athletes. There are growing numbers of athletes with some level of impairment involved in a range of sports. At the elite level, over 4300 athletes competed across 20 sports at the 2012 London Summer Paralympic Games, and over 500 athletes competed across 5 sports at the 2010 Vancouver Winter Paralympic Games. Involvement in sport has progressed far beyond participation as a mode for rehabilitation, with athletes increasingly training at the same volume, frequency, and intensity as their able-bodied counterparts at full-time professional athlete standards. Demand for appropriate nutrition education and support for these athletes are increasing in line with this trend, but research has not kept pace. Limited funding, expertise, and the substantial dissimilarity between individuals within a local environment resulting in very small subject numbers and a lack of "power" in a scientific context, all contribute to this limitation.

Paralympic sport is subdivided into six major classes:

- *Spinal cord injury (SCI):* an injury to the spinal cord caused by trauma or can be acquired (such as spina bifida or polio). The impact of the injury depends on the site along the spinal column and whether the injury has caused complete or incomplete loss of motor or sensory function below the site.
- *Cerebral palsy (CP):* nonprogressive, incurable condition involving damage to the motor control areas of the brain. Symptoms can vary widely from mild to severe (confinement to wheelchair) and may be more predominant on one side of the body. Can coexist with deficits in cognition, speech, hearing, and visual impairments and approximately one-third of individuals with CP also have epilepsy.
- *Amputee:* acquired or inborn limb deficit and can include single or multiple limbs.
- *Visual impairment:* visual deficits ranging from partial visual impairment to complete blindness.
- *Les Autres:* a group of nonspecific physical disabilities including substantial leg length differences, club foot, muscular dystrophies, dwarfism, multiple sclerosis, and Freidrich's ataxia.
- *Intellectual impairment:* key components include an IQ of less than 75 with onset under the age of 18 years and limitations in adaptive behavior.

Interested readers are also referred to the International Paralympic Committee website (www.paralympic.org) for information regarding classification of athletes with an impairment, summer and winter paralympic sports, and the rule variations for sports, which should be considered when advising athletes with an impairment.

Many athletes with an impairment will be physiologically similar to able-bodied athletes, such as those with visual impairments, intellectual impairment, high-functioning CP, and forearm

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amputees/congenital malformations, and therefore sports nutrition principles can be directly applied to them, albeit with some practical modifications. Others present a greater challenge in how best to interpret guidelines, considering the limited sport- and athlete-specific research that is available to assist the practitioner. This chapter will focus on this latter group—specifically those with SCI, moderate to high dysfunction CP, and lower limb/multi-limb amputees. Wherever possible, only the literature relating to active individuals will be utilized, rather than considering literature in untrained/inactive populations or in a rehabilitation setting.

The Underpinning Science

Physiology of Spinal Cord Injury

The impact of SCI on muscular and neurological function depends on the location and completeness of the spinal cord lesion. Figure 32.1 outlines the spinal cord levels and their associated primary motor and neural control areas (more details are available on the ASIA website). The "normal" physiological response to exercise may be disrupted due to ineffective vasoregulation below the level of injury, reduced total active muscle mass, increased total peripheral resistance, and reduced venous return due to the lack of a musculoskeletal pump from the lower limbs (Glaser, 1985). Since spinal injuries can impair both sympathetic and parasympathetic responses, renal, intestinal, and liver function and catecholamine release may also be impacted.

The potential impact of SCI on exercise performance is well summarized by Bhambhani (2011). Peak anaerobic power and capacity, peak heart rate, and peak aerobic capacity are all inversely related to the level of the lesion. Gender differences are proportionally similar, and training appears to elicit similar proportional responses over time to those of able-bodied athletes. A well-trained athlete with low-level paraplegia (e.g., between lumbar level 4 (L4) and sacral level 3 (S3)) can generate peak anaerobic power three to four times greater than an athlete with quadriplegia with a lesion level above cervical level 6 (C6), which is a direct consequence of the total musculature available to generate power and their maximum cardiovascular capabilities. The peak heart rate attainable in quadriplegics ranges between 110 and 125 beats per minute because the sympathetic stimulation to the myocardium is disrupted. Peak blood lactate concentration is also somewhat suppressed, due to a blunted catecholamine response to exercise as well as the reduced functional muscle mass. An incomplete spinal cord lesion results in less impairment of functional capacity, and hence higher peak cardiorespiratory responses, at all levels of SCI. What is less well understood is the impact of SCI on catecholamine release and other components, which could influence substrate utilization during exercise.

Despite these functional limitations, the proportional physiological responses to exercise and variation in results between different sports are similar to what would be expected based on studies in an able-bodied population. For example, Leicht et al. (2012) determined that the blood lactate concentration and perceived exertion at submaximal stages of exercise were the same for a given percentage of peak aerobic capacity in a group of para- and quadriplegic athletes as they were for non-spinal cord-injured wheelchair athletes. Additionally, the maximum rate of fat oxidation during aerobic exercise is achieved at a similar percentage of maximum aerobic capacity (Knechtle et al., 2003).

Thermoregulatory capacity in SCI individuals is impaired due to the disruption of the autonomic and somatic nervous systems that control skin blood flow and sweat responses (Price, 2006). As with their cardiovascular responses, the degree of impairment is inversely related to the level and completeness of the spinal cord lesion. It is commonly believed that athletes with SCI do not sweat below the level of their spinal cord lesion, but there are variations in this response depending on the completeness of the spinal cord lesion, and it can be exacerbated by certain medications. In a practical setting, some athletes with complete spinal cord lesions report sweating below the level of their spinal cord lesion, albeit it is likely these sweat losses are not substantial nor may they contribute effectively to heat dissipation. As a consequence, in response to exercise and hot conditions, athletes with SCI will experience an increase in core body temperature and are

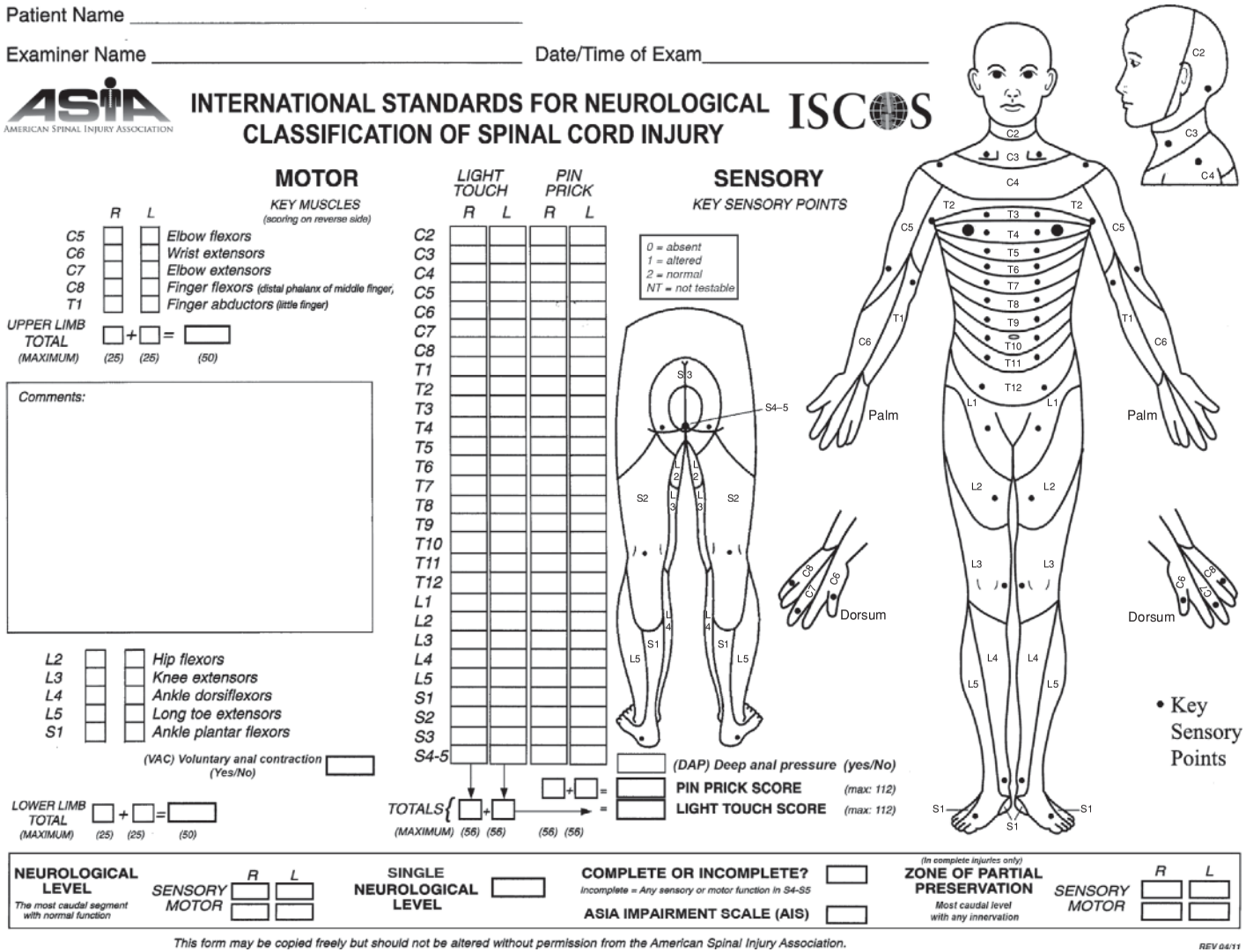

Figure 32.1 ASIA International Standards for Neurological Classification of Spinal Cord Injury. From the ASIA classification manual. Source: American Spinal Injury Association.

at a greater risk of developing heat stress responses than able-bodied athletes, and individuals with a higher level SCI may experience these effects both at rest in hot conditions and during exercise under any environmental conditions. Conversely, these athletes respond effectively to localized cooling (such as hand and head/neck cooling) before and during exercise in hot environments. Similarly, in cold conditions active heating may be required for a high-level SCI.

Physiology of Cerebral Palsy

The deficits in neuromuscular function experienced by CP athletes vary enormously and depend on the location and extent of the damage in the brain. The most common form of CP involves spasticity, resulting in jerky, uncoordinated movements due to increased muscle tone. Athetoid dyskinesis is another form of CP that results in slow, writhing, uncoordinated movements, which can affect all parts of the body, including face, mouth, and tongue. The third category is ataxic dyskinesis, which affects balance, coordination, depth perception, and the ability to undertake movements that require a great degree of control. Many individuals will have a combination of forms of CP, and the CP may affect only one limb or all muscles in the body. Physiological limitations (such as lower peak VO_2) are associated with reductions in peak power in the affected limbs and the earlier onset of local fatigue rather than deficits in attaining maximum heart rate (Bhambhani, 2011). The validity of lactate threshold in athletes with CP is questionable, possibly due to muscle spasticity (Bhambhani, 2011). Despite potential limitations, athletes with CP respond to training in a similar way to able-bodied individuals, and training itself may improve functionality through increased muscle strength.

The thermoregulatory capacity of athletes with CP has not been investigated. Theoretically, the reduced efficiency of movement in individuals with CP could increase their thermal stress during prolonged exercise, but their sweating response should not be impaired. Therefore, investigations of sweating responses and thermal load of exercise should be examined in CP athletes on a case-by-case basis.

Physiology/Biomechanics of Amputees

Little has been published regarding the impact of a limb deformity or amputation on physiology or biomechanics, most likely because the impact will depend on the activity being undertaken. During ambulatory activities, upper limb amputees may not exhibit physiology that is different from that of able-bodied individuals, while in contrast there appear to be some differences in lower limb amputees. Genin et al. (2008) showed that the maximum speed at which an individual can walk with a respiratory exchange ratio (RER) of less than one decreases with the level of amputation (1.4–1.7 m/s in transtibial amputees and 1.1–1.3 m/s in transfemoral amputees compared with up to 2 m/s in able-bodied individuals). Above their optimum walking speed of 1 m/s, the energy cost of walking is substantially higher in transtibial amputees than in able-bodied controls, whereas the energy cost of walking at any speed is higher in transfemoral amputees. Several reasons have been put forward for these changes, including asymmetry of movement patterns, the inability of the prosthetic limb to store elastic energy, and the lack of propulsive forces in the leg musculature. The technology of prostheses is continuing to evolve in order to minimize the likelihood that the prosthesis limits the performance of amputee athletes, but the majority of athletes continue to use less sophisticated prostheses in daily living situations.

Energy and Macronutrients

Energy Expenditure

To date, there are no validated prediction equations for energy expenditure in athletes with an impairment, and very few studies have measured any component of energy expenditure. It is reasonable to expect that both resting energy expenditure (REE) and energy expenditure during exercise are lower in SCI athletes than their able-bodied counterparts due to the reduced active muscle mass. Stuart (2007) found REE was lower in elite male SCI athletes than in age-, height-, and body mass-matched active able-bodied controls (6437 ± 769 kJ/day vs. 6964 ± 594 kJ/day). There was no difference

in energy expenditure when adjusted for body mass (92 ± 4 kJ/kg/day for both groups) but the REE was higher in the SCI athletes when adjusted for lean body mass (LBM) (146 ± 29 kJ/kg LBM vs. 125 ± 8 kJ/kg LBM). These results are similar to those reported in male SCI wheelchair ball game players whose REE ranged from 6300 to 6712 kJ/day (Abel et al., 2008), which are 9–15% lower than the estimated REE based on the Harris–Benedict equation. Contrary to expectations, no difference was found in the REE between wheelchair rugby players (all quadriplegics) and wheelchair tennis or basketball players (predominantly lower level SCI)—the standard deviations resulted in a within-group variation in resting metabolic rate (RMR) of up to 25%, which is not unexpected considering the spinal cord lesions of the group ranged from C5 to L3. On the other hand, energy expended during training (averaged over at least 10 training sessions) was significantly lower in the wheelchair rugby players (~1050 kJ/h) than in wheelchair tennis (~1360 kJ/h) or basketball (1550 kJ/h; Abel et al., 2008) players. No energy expenditure data have been published for female SCI athletes. Collins et al. (2010) have published a summary of the energy expenditures of activity in free-living SCI individuals, which represents the most comprehensive activity-related data to date on this group. While this could be a useful resource, it must be noted that many of the figures provided are based on very small sample sizes (two to three individuals), especially for females.

The energy expenditure of athletes with CP has been reported to be slightly lower than that of age-matched, able-bodied individuals and varies according to whether they have athetosis (uncontrolled, jerky movements that elevate RMR by approximately 15%; Johnson et al., 1996) and their ambulatory capabilities. Johnson et al. (1997) assessed the energy expenditure of free-living adults with CP using doubly labeled water and reported that REE was higher in ambulatory individuals (6663 ± 1296 kJ/day) than in nonambulatory ones (5870 ± 932 kJ/day), although when adjusted for fat-free mass (assessed via dual x-ray absorptiometry (DXA)) the reverse was true, most likely due to a higher proportion of athetosis in the nonambulatory individuals. Energy expenditure of daily activity, however, has been shown to be lower in those with athetosis (1346 vs. 2475 kJ/day), at least in a nonathletic CP population (Johnson et al., 1996). It has therefore been recommended that standard energy expenditure prediction equations are not appropriate for use in this population. To date, no studies have reported either RMR or total energy expenditure in athletes with CP.

The REE and total energy expenditure of amputee athletes have not been investigated. There is evidence to suggest that while REE may be reduced due to less muscle mass, the energy cost of ambulation and exercise may be increased by up to 60% (depending on the location of amputation) due to the balancing required and inefficiency of movement with prosthetic limbs (Genin et al., 2008). How each component affects total energy expenditure has not been determined and will depend upon the sporting activity undertaken by the athlete, training volume and type, and their degree of non-exercise-related mobility.

Only two studies have attempted to measure or report the dietary and energy intakes of athletes with an impairment. Goosey-Tolfrey and Crosland (2010) undertook 7-day food diaries with wheelchair sport athletes and found the average energy intake of males to be 8610 ± 3780 kJ/day (132 ± 52 kJ/kg/day) and females, 6353 ± 1430 kJ/day (100 ± 21 kJ/kg/day). Krempien and Barr (2011) reported similar intakes in male SCI athletes (9012 ± 1801 kJ/day, or 130 ± 33 kJ/kg/day) but higher intakes in their female SCI athletes (8322 ± 1601 kJ/day, or 150 ± 29 kJ/kg/day). As in able-bodied athletes, the males consumed higher energy intakes than the females. Neither study reported the variance in energy intakes according to the type of impairment or level of SCI, but the large standard deviation is indicative of the lack of homogeneity in this population. The lower reported energy intake in the wheelchair sport athletes confirms observations that many SCI/wheelchair-dependent athletes consume lower energy intakes than expected, which may be a result of efforts to prevent unwanted body fat gains. In practice, many athletes who report low energy intakes can respond well to an increased energy intake in regard to improved training capacity and exercise performance without detrimental effects to body composition.

In summary, it can be difficult to assess energy requirements of athletes with an impairment. A skilled practitioner will have to account for all the variables that could influence energy expenditure, but also consider current energy intakes, training loads, and body composition goals. It is important to maintain frequent reviews with the athlete in the initial stages of changing energy intakes in order to allow for adjustments to be made based on the response to change, as it can be easy to both over- and underestimate requirements.

Assessing Body Composition

The assessment of body composition in athletes with an impairment can be complicated and there are minimal comparative data to draw upon. As Chapter 6 outlines, numerous methods could be utilized to assess body composition, all of which have strengths and limitations. Consequently, it is important for the practitioner to understand what they want to assess, the limitations in the methods, and how this may be further compromised by the impairment itself, before deciding on whether and how to assess body composition in this athlete population. For example, long-term SCI results in muscle atrophy and reduced bone density in denervated areas. Because of this, methods that estimate body density will be invalidated since the assumptions underlying these calculations are not uniform across the SCI body. There are no validated equations to convert skinfold measures to percent body fat in SCI, and most skinfold equations substantially underestimated percent body fat in SCI athletes (Mojtahedi et al., 2009; Sutton et al., 2009). The appropriateness of whole-body versus segmental bioelectrical impedance (BIA) would need consideration in both SCI (due to potential differences in resistivity between innervated and non-innervated areas of the body) and amputee individuals (due to the lack of at least one limb compartment), and assumptions relying on body symmetry are violated in a number of paralympic populations. Percent body fat reported from BIA correlates poorly with percent body fat from DXA in SCI athletes (Mojtahedi et al., 2009) and with percent body fat from DXA and ^{18}O dilution in individuals with CP (Hildreth et al., 1997). The reproducibility of BIA measurements can be influenced by the degree of spasticity in the limbs; hence, athletes with CP or SCI who experience muscle spasm may require a longer time period for stabilization of measures (Hildreth et al., 1997). It appears that the more repeatable methods of assessing body composition are DXA and surface anthropometry measures (skinfolds, girths, bone lengths, and breadths).

Bone mineral density (BMD) below the level of a spinal cord lesion is lower in individuals with SCI due to bone demineralization, but appears to be positively influenced by an early involvement in sporting activities (Miyahara et al., 2008). In an SCI athlete population, there appears to be higher BMD in the arms, but lower BMD in the legs, compared with a reference able-bodied population (Miyahara et al., 2008; Stuart, 2007; Sutton et al., 2009). Similarly, LBM is lower in the legs due to muscle atrophy in those with SCI.

Stuart (2007) compared body composition of male SCI athletes using DXA and surface anthropometry. LBM was significantly lower in male SCI athletes than in active controls matched for age, body mass, and stretch stature (44.4 ± 7 kg vs. 56.0 ± 4.9 kg; p <0.01). Percent body fat on DXA was higher in the SCI athletes (33 ± 9% vs. 22 ± 5%; p 0.026) as were skinfolds, and the sum of eight skinfolds correlated strongly with percent body fat via DXA. Percent body fat (DXA) tends to be higher in the legs of SCI than able-bodied controls, whereas percent body fat in the arms tends to be lower (Stuart, 2007; Sutton et al., 2009).

In day-to-day practice, modifications may be required to the standard measurement protocols for surface anthropometry. For example, it is most relevant to measure upper body skinfolds (tricep, subscapular, bicep, and abdominal) and seated height in those with SCI, and measures may need to be undertaken on the left-hand side of the body in the case of right-sided amputees or right-sided hemiplegia in CP. Where changes are made, it is imperative that the practitioner records the changes so that the same protocol can be used each time. It is also important to have appropriate equipment (such as seated scales for SCI athletes) and to consider factors such as whether to take body mass with or

without prosthetics, since these can change over time. Combining skinfold measures with girths (especially waist girth for SCI, which is correlated with body fat; Sutton et al,. 2009) can be useful for assessing changes in body composition across time, as can the use of DXA (if available) for tracking segmental changes as a result of training. Athlete "normal values" are yet to be developed in the SCI population, whereas able-bodied athlete data can be a source of comparison for CP and amputees if the relevant sites can be taken.

Carbohydrate

The only study to look at muscle glycogen utilization during exercise in SCI individuals was undertaken 30 years ago in a non-exercising population. Resting muscle glycogen concentrations were lower than in able-bodied individuals, but muscle glycogen usage during exercise was reported to be similar (Skrinar et al., 1982). Peak fat oxidation (and conversely relative carbohydrate oxidation) during arm crank exercise has been found to be similar between SCI athletes and able-bodied cyclists (Knechtle et al., 2004b). Peak fat oxidation rates occurred at a lower percentage of maximal oxygen uptake (55% VO_2 peak) in wheelchair ergometry (Knechtle et al., 2004a) and hand cycling (Knechtle et al., 2004b) than in arm crank exercise (75% VO_2 peak) in well-trained SCI athletes. Whether these differences are due to the amount of active musculature involved, the SCI injury level, or the training state of the athletes has not been elucidated.

There are also limited data available regarding the response to carbohydrate feeding during exercise in athletes with an impairment. Temesi et al. (2010) investigated the response to carbohydrate intake during 60-minute steady-state arm cranking (65% VO_2 peak) followed by a 20-minute time trial (TT) in active SCI subjects (C6 to T7 complete lesions). No significant difference in TT performance was seen (51.8 ± 22.3 kJ for placebo, 54.2 ± 22.6 kJ for CHO). Of the six subjects, two improved in performance, one declined, and the other three remained stable with the carbohydrate ingestion. Spendiff and Campbell (2005) found a tendency toward improved TT performance in a similar exercise bout following consumption of an 11% compared to a 4% carbohydrate solution 20 minutes prior to commencing exercise in paraplegic athletes. With such limited research, it is difficult to determine the carbohydrate utilization and requirements of athletes with an impairment, relative to exercise intensity and duration and fitness levels. By the same token, it does not appear as though they differ substantially from able-bodied athletes and hence standard sports nutrition recommendations can still be used as a basis for education.

Carbohydrate intakes have been reported to vary from 3.4 to 4.9 g/kg BM/day in female athletes with an impairment and from 4.2 to 4.3 g/kg BM/day in males (Goosey-Tolfrey & Crosland, 2010; Krempien & Barr, 2011). Since carbohydrate requirements are dependent on exercise duration and intensity, it is important to understand the physical demands of the sport as well as the relative contributions of fat and muscle to the total body mass of the individual. Setting daily carbohydrate recommendations, and those specific to training, may require some trial and error in many athletes with an impairment.

As a percentage of total energy intake, the reported carbohydrate intakes of the female wheelchair sport players are the same as those of other athletes, so their lower intake is a consequence of a low total energy intake. The practitioner needs to work closely with athletes who display low total energy intakes in order for them to understand how better to match energy and carbohydrate needs to their training load. In addition, the types of foods contributing carbohydrate to the diet may need careful consideration in SCI athletes due to potential changes in fiber intake, which may affect carefully planned bowel management.

Protein

Protein requirements of athletes with an impairment have not been assessed. Currently, an absolute amount of protein is recommended immediately after exercise in able-bodied athletes, but the research upon which this is based involved athletes with a relatively homogeneous total body mass and LBM. It is yet to be determined whether recommendations for either daily or post-exercise recovery

protein intakes should be altered due to the reduced proportion of lean to total body mass in many paralympic athletes. The practitioner should therefore use some discretion, especially where athletes have a higher proportion of body fat compared with their able-bodied counterparts.

Dietary protein intakes have been reported to be within the recommended ranges for male athletes with an impairment (1.4 g/kg BM/day) but a wider variation has been reported for females (1.0–1.7 g/kg BM/day; Goosey-Tolfrey & Crosland, 2010; Krempien & Barr, 2011). As with carbohydrate, the protein intake of female wheelchair sport players is the same percentage of total energy intake as for the other athletes, so their lower protein intake is a consequence of a low total energy intake.

Fat

Dietary fat intakes have been shown to be approximately 27–29% of total energy intakes for male and female SCI and wheelchair sport athletes (Goosey-Tolfrey & Crosland, 2010; Krempien & Barr, 2011), which complies with general recommendations.

Water and Electrolyte Loss and Replacement in Training and Competition

Since athletes with SCI have a disrupted sweating response below the level of injury, it is logical to assume that their sweat losses during exercise are lower than for able-bodied populations. Price (2006) surmised that this is the case with lower level SCI, with the between-group differences increasing as environmental temperature increases. Higher level SCI individuals may appear to have higher localized sweat losses (e.g., forehead), but the overall sweat loss is lower and the sweat itself may be ineffective for cooling (Price, 2006). Personal observations in quadriplegic male wheelchair rugby players indicate sweat rates averaged 119 ml/h and fluid intakes of 279 ml/h during a 95-minute on-court training session. In contrast, male wheelchair basketball players (teams included athletes with SCI, CP, amputees, and other impairments) exhibited sweat rates ranging from 180 to 1045 ml/h and fluid intakes ranging from 360 to 1470 ml/h during games. Similarly, sweat rates were reported to range from 200 to 1300 ml/h in wheelchair tennis players undertaking intermittent sprint activity in the heat (30°C, males and females; Goosey-Tolfrey et al., 2008) while fluid intakes ranged from 500 to 1900 ml/h. To date, sweat rates and fluid intake practices of athletes with CP or amputees have not been reported. In regard to monitoring daily hydration status, the use of a refractometer to measure urine specific gravity is recommended as medications may influence the urine color.

It is important for the practitioner to remember that fluid intake recommendations should be provided according to estimated sweat losses. Athletes with SCI may require only very small fluid intakes during exercise, and yet be observed to drink more than they require. This may be due to elevated core body temperature creating a drive to drink or to inappropriate education. Active/passive cooling in these athletes (keeping athletes in the shade, ice vests, ice towels, cooling pieces around the head, neck, and hands, ingestion of iced or very cold beverages) has been shown to be effective in attenuating the increased core temperature during exercise (Price, 2006; Webborn et al., 2010). Indeed, the use of active cooling has been shown to reduce voluntary fluid intake in SCI athletes exercising in the heat, the outcome being that fluid intakes matched sweat losses (Goosey-Tolfrey et al., 2008). In addition, practical limitations in dealing with the consequences of over-hydration must be considered, such as being strapped into chairs, hands being taped, difficulty getting in and out of competition chairs, and accessing suitable toilet facilities.

Micronutrients and Dietary Supplements

Vitamins and Trace Minerals

Micronutrient requirements and intakes of athletes with an impairment have generally received very little attention. One recent report in 32 Canadian SCI athletes indicated that more than 25% of both males and females consumed less calcium, folate, magnesium, and vitamin D than the estimated average requirement, with males also consuming

less zinc, riboflavin, and vitamin B12 than the US-estimated average requirement (EAR) (Krempien & Barr, 2011). As 50% of the population might reasonably be expected to need less than the EAR, this does not necessarily suggest that the intake of any individual was inadequate. In contrast, iron was the primary micronutrient of concern in female wheelchair sports athletes in the United Kingdom, with 12 out of 14 athletes not achieving the UK-recommended nutrient intake whereas the males did (Goosey-Tolfrey & Crosland, 2010). There remains insufficient scientific evidence to indicate whether the dietary intakes of athletes with an impairment is compromised or differs from reports of able-bodied athletes.

Vitamin D

Vitamin D is one micronutrient which has been shown to be deficient in certain athletic populations (see Chapter 20) and has also been found to be insufficient during winter in 96% of individuals with chronic SCI (Oleson et al., 2010). As discussed in Chapter 20, vitamin D is essential not only for adequate bone mineralization, but also to influence muscle and immune function. Many athletes with SCIs compete in indoor sports (basketball, wheelchair rugby) or often cover their lower limbs, thereby minimizing the skin surface area exposed to sunlight. Consequently, it is important to assess vitamin D status of athletes with a SCI and limb amputees and manage appropriately (as outlined in Chapter 20).

Nutritional Ergogenic Aids

To date, there has been minimal research on the use of ergogenic aids in athletes with an impairment. While in theory there is a potential for ergogenic aids to improve performance in paralympic athletes, as with all athletes this should only be considered once an athlete has achieved a consistently high level of training/competition and consumes an appropriate dietary intake. In many instances, additional care should be taken regarding the potential impact of known side effects on the athlete due to functional, medical, and practical limitations, for example, the use of protein-containing supplements in athletes with renal impairment, the use of caffeine in athletes with neurological or cardiovascular disorders, the use of carbohydrate-containing products in athletes with diabetes, or the potential interaction of a supplement with medications.

Creatine

To date, only one study has been reported in trained athletes with an impairment. Perret et al. (2006) supplemented six SCI track athletes (four males, two females) with 20 g creatine monohydrate (4×5 g/day) for 6 days and found no difference in 800-m TT performance, heart rate, rating of perceived exertion, or lactate. No measures of muscle creatine concentrations were undertaken, and the creatine was consumed with water rather than with carbohydrate-containing liquid or food, so there is no indication as to the effect of the supplementation on muscle creatine concentration in this athlete population.

Practical Issues

The Traveling Athlete

Traveling with athletes with an impairment can present numerous challenges, primarily of a practical nature, such as baggage weight limitations and seating requirements on flights.

It is not uncommon for athletes with SCI, or other impairments which reduce mobility, to deliberately dehydrate ahead of long-haul flights. The reasons given include the limited accessibility of bathrooms on airplanes, the limited capacity of urine collection bags, and concerns regarding hygiene if catheters require replacement during flights. As a consequence, recovery times following flights tend to be more prolonged and the risk of developing urinary tract infections (UTIs) is increased. It is therefore important to explore ways to improve hydration status prior to and during long-haul flights, as well as rapid and effective rehydration upon arrival. It is recommended that athletes try the use of electrolyte-containing fluids (e.g., oral rehydration solutions, or electrolyte tablets), even in relatively small quantities (200–300 ml). Athletes should also be reminded to keep water bottles with them rather

than allow them to be placed in overhead luggage compartments.

Athletes who mobilize using a wheelchair need to pay particular attention to hygiene due to contact between the wheels of the chair and the ground and then the transfer to hands during ambulation. These athletes should be encouraged to regularly use hand-sanitizing solutions and take special care in avoiding touching the face/mouth.

Food and Nutrition Considerations at Major Competitions

Development of menus and utilizing caterers may also need consideration when working with paralympic athletes. Athletes with SCI, particularly those with higher level lesions, may have restricted capacity for torso and arm movement and limited elevation and arm reach for practical purposes. Staff may be required to assist in plating meals and carrying food to the table when athletes eat at buffet-style food services. Some athletes may not be able to hold a knife and fork; hence, the need to source foods that require minimal cutting can also be considered. Other athletes may have to eat using their feet to hold utensils, while visually impaired athletes often use a "clock" system to know where different components of food are on their plate, and several athletes may require assistance in serving and/or feeding themselves. If athletes are in self-catering facilities, consideration must be given to bench height and utensils. The time required to prepare a meal is often longer in athletes with an impairment due to their functional limitations, so the use of self-catering facilities and the timing of training sessions, team meetings, etc., should be carefully planned to ensure the capacity to eat is not compromised. Finally, some intellectually impaired athletes may have food aversions.

Health-Related and Clinical Sports Nutrition

Athletes with SCI and amputees are at risk of disruption to the skin surface via pressure or abrasions incurred through transferring between chairs or across different surfaces, or the interface between the skin and prosthetic limbs. The disrupted blood and nervous supply below the level of spinal cord lesion can lead to a reduced rate of healing and an increased risk of developing pressure ulcers (Brewer et al., 2010). If this occurs, it is important to consider not only the adequacy of total energy and protein intake to optimize healing, but also various micronutrients known to support the healing process, such as zinc, vitamin E, vitamin C, selenium, and copper. It is important that athletes not be supplemented with individual micronutrients unless a clinical deficiency is found, since excessive amounts of some nutrients may reduce the effectiveness of others. However, there may be benefits to supplementation with a broad-spectrum multivitamin/mineral supplement containing trace elements such as selenium and copper at times when wounds are present in order to promote healing. Furthermore, there is evidence that supplementation with arginine (>4 g/day) can speed the healing of grade 2 or higher pressure wounds (Brewer et al., 2010).

UTIs remain a common cause of morbidity and the most common infection in SCI individuals. For athletes, UTIs can disrupt training for at least several days due to pain, fever, and malaise, and in some athletes a UTI increases the likelihood of muscle spasm. A UTI occurring at the time of competition can substantially impair performance. There is evidence promoting the use of cranberry juice or tablets (2×500 mg tablets daily) to prevent UTI in SCI (Hess et al., 2008). Minimizing dehydration is an important adjunct to preventing UTI, and this is an area where education and practical advice regarding maintaining hydration, especially through travel, is often necessary. For travel, doctors may prescribe prophylactic antibiotics.

Autonomic dysreflexia is a reflex that is unique to individuals with SCI above T6 (Bhambhani, 2011). Autonomic dysreflexia can be spontaneously or deliberately induced via stimulation of nociceptive receptors below the level of spinal cord lesion resulting in an increased blood pressure and, consequently, an increased blood flow to muscles that can improve performance. Also commonly known as "boosting," this practice is banned by the International Paralympic Committee and World Anti-Doping Agency as the large increase in blood

pressure cannot be counterbalanced by vasodilation and, hence, it can result in serious conditions including cerebral hemorrhage and death. Some athletes will induce boosting by over-distending their bladder (e.g., clamping the urinary catheter), sitting on sharp objects, or using tight leg straps. It can also be induced as a result of UTIs, constipation, and pressure sores. It is important for all who work with athletes with SCIs to recognize the symptoms of autonomic dysreflexia (presence of "gooseflesh," increased sweating, skin blotching, anxiety, headaches, and tremors), as well as being aware of how they can reduce the likelihood of this occurring, such as avoiding large intakes of fluid in a short period of time and preventing UTIs and constipation.

In summary, each athlete with an impairment will present a unique set of circumstances that the practitioner needs to consider in preparing sports nutrition recommendations. The impact of "daily living" conditions, including medical and functional limitations, requires greater attention than in able-bodied athletes. Adapting sports nutrition recommendations to this population requires an intimate understanding of the individual, the sport they participate in and its physiological demands (specific to paralympic athletes), and the practical limitations you may face.

References

Abel, T., Platen, P., Rojas Vega, S., et al. (2008) Energy expenditure in ball games for wheelchair users. *Spinal Cord* 46, 785–790.

Bhambhani, Y. (2011) Physiology. In *The Paralympic Athlete* (eds. Y.C. Vanlandewijck & W.R. Thompson), pp. 51–73. Wiley-Blackwell, Chichester.

Brewer, S., Desneves, K., Pearce, L., et al. (2010) Effect of an arginine-containing nutritional supplement on pressure ulcer healing in community spinal patients. *Journal of Wound Care* 19, 311–316.

Collins, E.G., Gater, D., Kiratli, J., et al. (2010) Energy cost of physical activities in persons with spinal cord injury. *Medicine & Science in Sports & Exercise* 42, 691–700.

Genin, J.J., Bastien, G.J., Franck, B., et al. (2008) Effect of speed on the energy cost of walking in unilateral traumatic lower limb amputees. *European Journal of Applied Physiology* 103, 655–663.

Glaser, R.M. (1985) Exercise and locomotion for the spinal cord injured. *Exercise and Sport Science Reviews* 13, 263–303.

Goosey-Tolfrey, V.L. & Crosland, J. (2010) The nutritional profiles of competitive wheelchair games players. *Adapted Physical Activity Quarterly* 27, 47–59.

Goosey-Tolfrey, V.L., Diaper, N.J., Crosland, J., et al. (2008) Fluid intake during wheelchair exercise in the heat: effects of localised cooling garments. *International Journal of Sports Physiology and Performance* 3, 145–156.

Hess, M.J., Hess, P.E., Sullivan, M.R., et al. (2008) Evaluation of cranberry tablets for the prevention of urinary tract infections in spinal cord injured patients with neurogenic bladder. *Spinal Cord* 46, 622–626.

Hildreth, H.G., Johnson, R.K., Goran, M.I., et al. (1997) Body composition in adults with cerebral palsy by dual-energy X-ray absorptiometry, bioelectrical impedance analysis, and skinfold anthropometry compared with the ^{18}O isotope-dilution technique. *The American Journal of Clinical Nutrition* 66, 1436–1442.

Johnson, R.K., Goran, M.I., Ferrara, M.S., et al. (1996) Athetosis increases resting metabolic rate in adults with cerebral palsy. *Journal of the American Dietetic Association* 96, 145–148.

Johnson, R.K., Hildreth, H.G., Contompasis, S.H., et al. (1997) Total energy expenditure in adults with cerebral palsy as assessed by doubly labeled water. *Journal of the American Dietetic Association* 97, 966–970.

Knechtle, B., Muller, G., Willmann, F., et al. (2003) Comparison of fat oxidation in arm cranking in spinal cord-injured people versus ergometry in cyclists. *European Journal of Applied Physiology* 90, 614–619.

Knechtle, B., Muller, G., Willmann, F., et al. (2004a) Fat oxidation at different intensities in wheelchair racing. *Spinal Cord* 42, 24–28.

Knechtle, B., Muller, G., & Knecht, H. (2004b) Optimal exercise intensities for fat metabolism in handbike cycling and cycling. *Spinal Cord* 42, 564–572.

Krempien, J.L. & Barr, S.I. (2011) Risk of nutrient inadequacies in elite Canadian athletes with spinal cord injury. *International Journal of Sport Nutrition and Exercise Metabolism* 21, 417–425.

Leicht, C.A., Bishop, N.C., & Goosey-Tolfrey, V.L. (2012) Submaximal exercise responses in tetraplegic, paraplegic and non spinal cord injured elite wheelchair athletes. *Scandinavian Journal of Medicine and Science in Sports* 22, 729–736.

Miyahara, K., Wang, D., Mori, K., et al. (2008) Effect of sports activity on bone mineral density in wheelchair athletes. *Journal of Bone Mineral Metabolism* 26, 101–106.

Mojtahedi, M.C., Valentine, R.J., & Evans, E.M. (2009) Body composition assessment in athletes with spinal cord injury: comparison of field methods with dual-energy X-ray absorptiometry. *Spinal Cord* 47, 698–704.

Oleson, C.V., Patel, P.H., & Wuermser, L.A. (2010) Influence of season, ethnicity, and chronicity on vitamin D deficiency in traumatic spinal cord injury. *Journal of Spinal Cord Medicine* 33, 202–213.

Perret, C., Mueller, G., & Knecht, H. (2006) Influence of creatine supplementation on 800m wheelchair performance: a pilot study. *Spinal Cord* 44, 275–279.

Price, M.J. (2006) Thermoregulation during exercise in individuals with spinal cord injuries. *Sports Medicine* 36, 863–879.

Skrinar, G.S., Evans, W.J., Ornstein, L.J., et al. (1982) Glycogen utilization in wheelchair-dependent athletes. *International Journal of Sports Medicine* 3, 215–219.

Spendiff, O. & Campbell, I.G. (2005) Influence of pre-exercise glucose ingestion of two concentrations on paraplegic athletes. *Journal of Sports Sciences* 23, 21–30.

Stuart, N. (2007) Resting energy expenditure and body compositions in elite male athletes with spinal cord

injuries. Honours Thesis. University of the Sunshine Coast, Australia.

Sutton, L., Wallace, J., Goosey-Tolfrey, V., et al. (2009) Body composition of female wheelchair athletes. *International Journal of Sports Medicine* 30, 239–265.

Temesi, J., Rooney, K., Raymond, J., et al. (2010) Effect of carbohydrate ingestion on exercise performance and carbohydrate metabolism in persons with spinal cord injury. *European Journal of Applied Physiology* 108, 131–140.

Webborn, N., Price, M.J., Castle, P.C., et al. (2010) Cooling strategies improve intermittent sprint performance in the heat of athletes with tetraplegia. *British Journal of Sports Medicine* 44, 455–460.

Chapter 33

Overreaching and Unexplained Underperformance Syndrome: Nutritional Interventions

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The aim of this chapter is to identify nutritional strategies to optimize recovery from overreaching and provide a basis for the development of a symptom-based recovery plan from unexplained underperformance syndrome (UUPS).

Overreaching and UUPS in Athletes

The main goal for all athletes is to improve their performance and this is achieved by increasing frequency, volume, and intensity of training. Acute increases in training load, also known as overreaching, are often successful in inducing an improved sports performance. Paradoxically, there is anecdotal evidence cited in the literature that links excessive exercise, over and above the state of overreaching, with a chronic decrement in athletic performance.

To ensure that the athlete adapts favorably to the training load, subsequently improving sports performance, adequate rest and appropriate nutrition should be considered integral components of the program. If either rest or nutrition is not sufficient and the exercise load alone or combined with other stressors (physical, environmental, or psychological) is too great, the athlete may fail to adapt (maladapt) and become overreached. If insufficient rest and inappropriate nutrition continue when overreached, and the athlete is exposed to further stressors, then a state of chronic fatigue and non-recovery may occur resulting in UUPS (Figure 33.1).

UUPS is defined by a persistent decrement in athletic performance capacity despite 2 weeks of relative rest, which is acknowledged by both the coach and the athlete (Budgett et al., 2000). UUPS should not be confused with overreaching which causes a temporary deterioration in performance, from which with sufficient rest and appropriate nutrition, full recovery occurs within a short period of time (<2 weeks).

Cytokine Theory of UUPS

The universal and most debilitating symptom in UUPS is persistent fatigue reported by athletes. A convincing theory explaining this chronic fatigue is the "cytokine theory to overtraining" (Smith, 2000). The theory proposes that exercise-induced tissue trauma evokes a chronic inflammatory state resulting in raised blood cytokine levels and consequent "cytokine sickness." Cytokines communicate with the central nervous system and induce a set of behaviors referred to as "sickness

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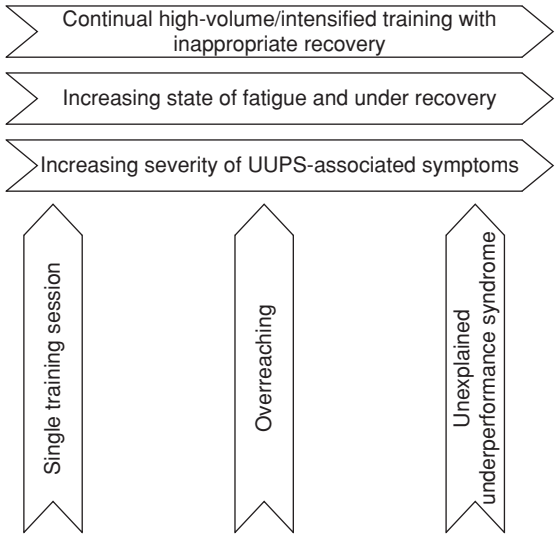

Figure 33.1 UUPS development continuum.

behavior," characterized by mood changes and persistent fatigue, until the dysregulated inflammatory response is resolved. This is thought to be a bioprotective mechanism, since it dampens the athlete's desire to expend energy in times of excessive physical and psychological stress.

Interleukin-6 Hypothesis to UUPS The cytokine theory has been further refined into the *"interleukin-6 (IL-6) hypothesis to UUPS"* (Robson, 2003) (Figure 33.2). This theory proposes that factors, aside from exercise-induced tissue trauma, trigger a dysregulated inflammatory response in UUPS causing either increased levels of the exercise-induced circulating cytokine IL-6 or an increased sensitivity to IL-6 by an upregulation of the soluble receptor for IL-6. The theory is primarily focused on IL-6's fatigue-inducing properties. IL-6 has many functions and a wide range of biological activities such as regulation of immune system responses and generation of acute phase reactions, and more recently, IL-6 has been identified as a glucose regulator during prolonged exercise (refer to Chapter 38 for further information on nutrition, exercise, and inflammation).

Concentrations of cytokines increase during strenuous exercise. In particular, IL-6 is produced in greater amounts than any other cytokine during

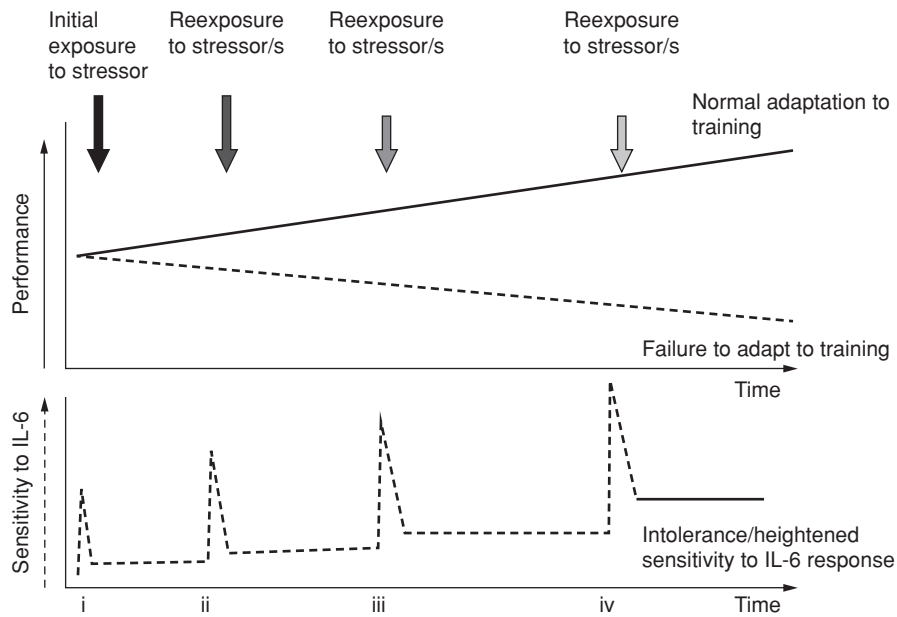

Figure 33.2 IL-6 hypothesis of unexplained underperformance syndrome (UUPS). i, initial exposure to a significant stressor, e.g., excessive exercise and poor recovery management; ii–iv, further exposure to stressor resulting in performance deterioration. Adapted from Robson (2003) with permission from Adis. (© Springer International Publishing AG, 2003. All rights reserved.)

prolonged exercise, with concentrations over 100-fold greater than resting levels reported following a marathon (Suzuki et al., 2002). Data obtained from a performance-related study (Robson-Ansley et al., 2004) suggest that IL-6 also plays a role in the sensation of fatigue during prolonged exercise. When a dose of rhIL-6 was administered to subjects prior to a 10 km time trial (to induce plasma concentrations equivalent to those found following prolonged exercise), subjects reported an increased sensation of physical and psychological fatigue during the exercise time trial, which ultimately resulted in a decrement in exercise performance. This suggests that IL-6 may also act as a circulating *"fatiguogen"* during exercise.

The IL-6 hypothesis suggests that a heightened sensitivity to or a dysregulated production of IL-6 during exposure to physical and/or psychological stress is likely mechanism for the development of UUPS in athletes. The absence of infection despite the flu-like symptoms reported by athletes following excessive exercise suggests a cytokine-mediated response to physical and psychological stress, and not necessarily the presence of pathogen-induced illness and infection.

Key Signs and Symptoms of UUPS

Over 200 signs and symptoms have been associated with UUPS (Fry et al., 1991), but the most prominent symptoms of UUPS are general fatigue and a heightened sense of effort during exercise/training. Other commonly reported symptoms include upper respiratory symptoms, e.g., sore throat, general malaise/flu-like symptoms; sleep disturbances and daytime sleepiness; unexplained/unusual muscle sensations, e.g., soreness, heaviness, and/or stiffness, slow wound healing; loss of appetite (anorexia) and/or unexplained/unintentional weight loss (Table 33.1).

Key Triggers for UUPS Athletes can often pinpoint an occasion after which UUPS developed. Triggering events include periods of overreaching, an infection, exertional heat stresses, and/or periods of hypoglycemia (exercise or diet-induced). These are frequently encountered by competitive athletes during training and competition programs.

Table 33.1 Commonly reported symptoms by athletes with UUPS

Increased fatigue during exercise
Underperformance with an inability to increase the pace at the end of a race
Increased fatigue and sleepiness during actual daily living
Increased upper respiratory tract symptoms
Reduced sleep quality—waking unrefreshed
Slow wound healing and muscle soreness

Nutritional information on maintaining a normal immune response, coping with environmental stressors, and optimizing carbohydrate intake to meet training and competition needs is comprehensively covered in other chapters. The following section will specifically address the role of nutrition for recovery from overreaching as well as providing nutritional advice for addressing key symptoms associated with UUPS.

Nutrition for Recovery from Overreaching

Overreaching is a critical period during training, whereby sufficient rest and nutrition will promote favorable training adaptation to the prescribed exercise load and subsequent improvement in sports performance. During periods of overreaching, it is essential to develop nutritional strategies to provide sufficient quantity and quality of total energy and endogenous carbohydrate energy substrate (refer to Chapters 7–9 for further information on carbohydrate and exercise) and offset the potential for developing UUPS (Robson-Ansley et al., 2009).

A simple and generally reliable way to identify whether an athlete is consuming sufficient energy to meet exercise demands is to record body mass (BM) on a weekly basis, at the same time and day of the week (preferably in the morning after waking). Small fluctuations in BM should be expected but a consistent, unexplained, and unintentional BM loss represents an unknown energy deficit (providing that dehydration has been eliminated as a potential cause—see Chapters 14 and 16). Consecutive weeks of consistent BM loss would indicate a chronic

energy deficit: if this is unintentional, it requires attention (Seagle et al., 2009).

Guidelines suggest that most dietary energy for athletes should come from carbohydrate sources, with other energy sources being consumed mainly to supply other essential nutrients (amino acids, essential fatty acids, vitamins, minerals, water; Burke et al., 2004). High levels of carbohydrate intake are associated with optimum sport performance, while low levels of carbohydrate intake generally impair performance. Suboptimal carbohydrate intake can result in altered stress hormone responses, worsened mood state, general fatigue, and immune system depression and potentially lead to the development of UUPS (Robson-Ansley et al., 2009). General carbohydrate recommendations for exercise include ≤6 g/kg BM/day for those completing up to 2 hours of strenuous exercise; 6–8 g/kg BM/day for 2–4 hours of strenuous exercise; and 8 g/kg BM/day (up to 12 g/kg BM/day) for >4 hours of strenuous exercise (quantity dependent on training load; Burke et al., 2004). If carbohydrate needs for training and competition are not met, fat will become the predominant fuel at the point when glycogen stores become depleted, resulting in excessive production of ketone bodies. Ketone bodies are formed as a metabolic by-product of fat oxidation in the presence of inadequate carbohydrate. Regular monitoring of urinary ketone bodies at rest may provide a useful noninvasive test to monitor adequacy of carbohydrate and energy availability, with higher levels indicating that the body is using fat as the major energy source due to carbohydrate deprivation.

The following section will address some of the practical issues reported by athletes when attempting to maintain a high carbohydrate intake during high volume training:

There is a reduced time for food purchase, preparation, and consumption. As previously discussed, when exercise training loads increase, the demand for exogenous carbohydrate energy substrate also increases. Coupled with increased exercise training is a reduction in non-exercising free time which can have a significant impact on actual time spent on buying and sourcing food, as well as for the preparation and consumption of food. Poor time management could result in an athlete consuming less carbohydrate than required for optimum recovery and performance.

During periods of high training loads, carbohydrate requirements can only realistically be met through consumption of predominantly simple carbohydrate forms (especially if two strenuous training sessions are performed per day). It is therefore recommended, to increase consumption of simple carbohydrates in "an easy to consume" texture, e.g., carbohydrate-rich fluids. This should be *in addition* to habitual dietary intake. Supplementation with additional carbohydrates, e.g., glucose and/or maltodextrin, may be required to meet extremely high carbohydrate needs.

Large carbohydrate meals can lead to post-consumption discomfort. The consumption of bulky solid forms of carbohydrates is also another barrier to preventing athletes from meeting high carbohydrate requirements during periods of heavy training. Athletes may not feel like eating large amounts of bulky foods that may result in gastrointestinal discomfort if training shortly after ingestion. Therefore, the use of carbohydrate-rich fluids, e.g., milk shakes, fruit smoothies, soft drinks, fruit juices, and other hypertonic solutions, during these periods provides a practical way of consuming sufficient carbohydrates. Depending on the type of carbohydrate-rich fluid, e.g., milk shakes and fruit smoothies, they can also provide a good source of other nutrients (proteins, micronutrients, water). Chapter 43 provides further information on gastrointestinal function and dysfunction during exercise.

There is an increased risk of dental caries with frequent high carbohydrate ingestion. Caution should be paid to dental hygiene with the frequent consumption of foods and especially with the continual sipping of high carbohydrate fluids. Frequent brushing and regular 6-monthly checkups with a dentist and oral hygienist are recommended to avoid dental problems.

A high carbohydrate diet can lead to an unbalanced intake of protein and fats. In general, an athlete's protein requirements can easily be met though normal

daily dietary habits if energy requirements for training and/or competition programs are being met from a variety of food groups. Protein or amino acid supplements during periods of overreaching are not required if athletes are consuming a varied diet to meet their energy demands (Tipton & Wolfe, 2004). The amount of dietary fat that an athlete requires is dependent on their body composition, sport, and training/competition objectives. Dietary fat intake recommendations should be developed on an individual basis in consultation with a trained dietitian.

During periods of overreaching where proinflammatory and oxidative processes may be highly active, it is advised to incorporate foods rich in monounsaturated fats into the diet, e.g., olive products, rapeseed oil, avocado, eggs, almonds, hazelnuts, and macadamia nuts, as well as omega-3 polyunsaturated fats, e.g., oily fish, as these foods also have antioxidant and anti-inflammatory properties. Additionally, athletes should avoid nonessential fats such as saturated fat products, e.g., animal fats, high-fat dairy, coconut and palm oil products, as well as products with a high content of trans fats (hydrogenated, processed foods), e.g., some cakes, biscuits, and low-fat spreads, as these can have proinflammatory effects.

A high carbohydrate diet can lead to an unbalanced intake of other micronutrients. There is currently no research evidence indicating that athletes are at risk of vitamin or mineral deficiencies if they are consuming a varied diet and consuming sufficient energy to meet their energy requirements (Lukaski, 2004). During periods of overreaching, energy balance may not be met consistently. An increased exercise load can result in suppressed appetite, gastrointestinal discomfort, general fatigue/malaise/lethargy, as well as reduce motivation for food preparation potentially resulting in a restricted intake of specific foods and consequent vitamin/mineral deficiencies. For these high-risk athletes, an individualized dietary assessment is required, prior to any dietary recommendation or advice on micronutrients. However, during overreaching if an athlete is not consuming a varied diet, a daily intake of a multivitamin/mineral supplement that meets 100% of the recommended nutritional intakes is suggested in case of any short-term deficiencies.

Nutritional Strategies for an Athlete with UUPS Using a Symptomatic Approach

Frequent Infectious Episodes

Epidemiological studies have concluded that excessive exercise increases the risk of self-reported upper respiratory symptoms (Gleeson, 2000), the incidence of which is notably related to periods of heavy or intensified training as well as following prolonged exercise bouts (Figure 33.3). Furthermore, athletes with UUPS report an increased frequency of infection-type symptoms. Temporary modulations in innate and adaptive immune system function have been alleged to be the basis for the relationship between susceptibility to infection, exercise training, and UUPS (refer to Chapter 39 for further information on exercise, nutrition, and immune function). Indeed, excessive and intensified exercise training can transiently suppress markers of immune function (Walsh et al., 2011a, 2011b). However, studies demonstrating a relationship between the temporal suppression of immune function following excessive exercise and incidence of upper respiratory symptom have not been forthcoming. Furthermore, while athletes with UUPS may report greater frequency of symptomatic infectious episodes, these have not been directly linked to immune system suppression.

While a proportion of reported respiratory tract symptoms will be infectious in origin, other etiological factors include localized non-infective inflammation in the airways due to breathing high volumes of air during exercise, viral reactivation, inhalant allergies, and/or exercise-induced asthma (Walsh et al., 2011a). Some athletes may appear prone to colds and upper respiratory tract symptoms but this may be due to an increased exposure to viruses and bacteria in their everyday life, e.g., having young children, work and social life locations, and traveling on public transport. In

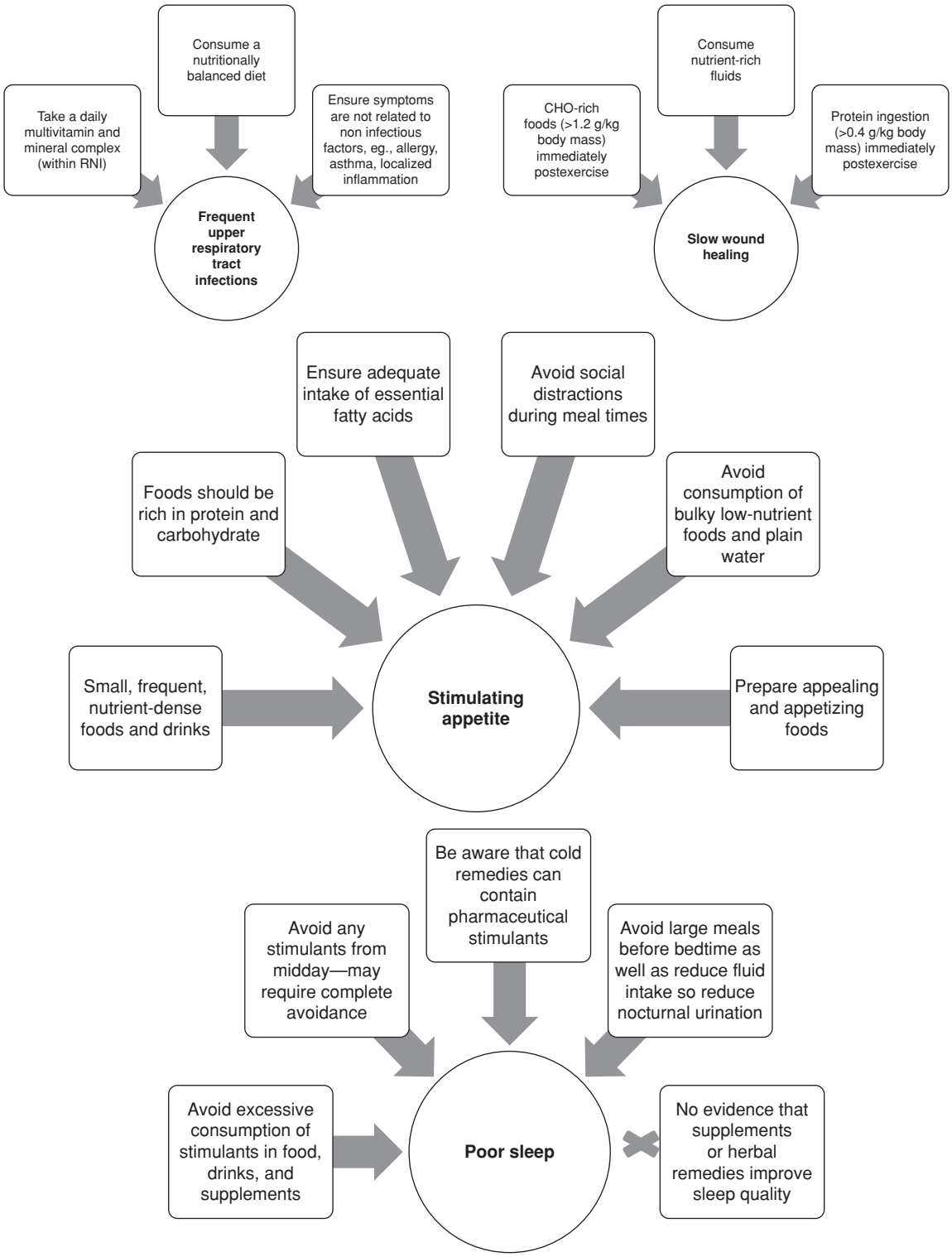

Figure 33.3 Recommended strategies for treatment of unexplained underperformance syndrome (UUPS) using a symptomatic approach. (*continued*)

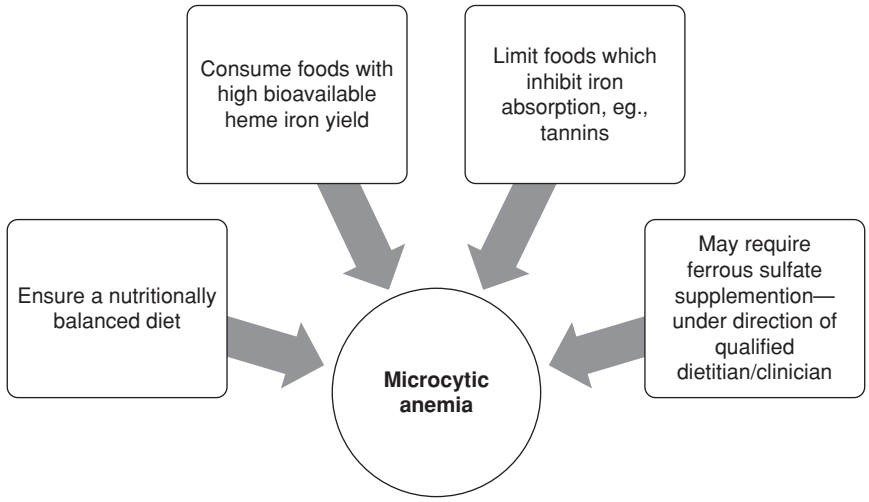

Figure 33.3 *(continued)* Recommended strategies for treatment of unexplained underperformance syndrome (UUPS) using a symptomatic approach.

these circumstances, attention to personal hygiene practices is key, e.g., frequent washing of hands. Boosting of the immune system through nutritional interventions and supplements is frequently marketed by industry as a treatment to reduce coughs and colds but there is little evidence of any efficacy. Simply, adjusting and tailoring an athlete's diet to meet nutritional requirements for their training/competition loads will support an adequate immune response and help attenuate exercise-induced immunodepression. Attempting to enhance the immune system response is not recommended unless for a clinical condition supervised by a physician as this could result in the development of autoimmune disease. In a generally healthy athlete, it may be that symptoms are more related to localized inflammation and/or undiagnosed allergic or asthmatic conditions.

Daytime Fatigue and Poor Sleep Quality in UUPS

Increased daytime fatigue and/or restless nighttime sleep are common features in athletes presenting UUPS. Even though limited studies have been conducted on the mechanisms that induce disturbed sleep–wake patterns in athletes presenting UUPS, it is likely elevations in proinflammatory, prosomnogenic cytokines (namely IL-1β, IL-6, TNFα, and IFNγ), and stress hormone responses are responsible (Banks & Dinges, 2007). For example, the onset of non-rapid eye movement (NREM) sleep coincides with dramatic increases in prosomnogenic cytokines which fall with sleep onset. Chronic increases in prosomnogenic cytokines have been observed after >24 hours of total sleep deprivation (Dinges et al., 1995), and intravenous administration of prosomnogenic, inflammatory cytokines can significantly alter normal sleep architecture (Shoham et al., 1987; Vgontzas et al., 1999). A chronic suppression of stress hormone responses, concomitant with increased prosomnogenic cytokine production (especially the "*fatigue-inducing peptide*," IL-6), is likely to contribute to the overall general and constant state of daytime sleepiness yet unrefreshing sleep reported with UUPS.

Sleep is also essential in maintaining immune defenses (Bryant et al., 2004) and chronic sleep loss can impair immune defenses predisposing an individual to a risk of infectious episodes. Furthermore, prolonged periods of disturbed sleep can reduce cognitive ability, worsen mood state and motivation, and slow motor skill acquisition, all of which will affect athletic performance.

Nutritional Strategies

1. Avoid excessive consumption of foods/drinks/supplements which contain stimulants, such as caffeine, taurine, and guarana during the day.
2. Total avoidance of the consumption of stimulant-containing foods/drinks after midday. If nocturnal sleepiness persists, remove stimulant-containing foods/drinks from the diet.
3. Be aware that certain pharmaceutical "over-the-counter" products such as cold remedies and decongestants also contain stimulants, e.g., caffeine.
4. Avoid large meals and fluid intakes before sleep time, limiting gastrointestinal discomfort and the need to urinate during the night.
5. Have the evening meal 3–4 hours prior to bedtime. A carbohydrate-rich evening meal and its associated insulin response may induce sleepiness.
6. There exists no convincing evidence that particular foods, dietary supplements, or botanical remedies improve sleep quality. Therefore supplementation to improve sleep is not recommended.

Slow Wound Healing

Another common feature of UUPS is prolonged tissues damage, e.g., perceived degree/duration of muscle soreness and delayed wound healing. Due to the physiological and mechanical stressors associated with strenuous physical exercise, it is common for both internal (e.g., muscle) and external (e.g., skin) tissues to experience damage, lesions, and/or abrasions. The presence of exercise-induced tissue damage stimulates an immune system cascade to clear damaged tissues and initiate healing processes that are part of the normal regeneration and adaptation cycle. Neutrophil immune cells, the most abundant immune cells in systemic circulation, are chiefly responsible for driving this process. In circulation, neutrophils are attracted to and migrate toward areas of damage (areas of infection and inflammation within tissues), where they combine defense strategies with adjacent immune cells in response to chemotactic signals (e.g., damaged/destroyed tissue fragments, complement activation, cytokine responses) further amplifying local innate immune responses (Peake, 2002; Smith & Pyne, 1997).

There is substantial evidence that depressed neutrophil function is strongly associated with immunodepression, slow wound healing, and increased risk of infection (systemic and local), albeit, this has been reported only in clinical populations (Alba-Loureiro et al., 2007; Ellis et al., 1988). Neutrophil function is depressed for several hours following both high-intensity and prolonged exercise (Costa et al., 2011; Robson et al., 1999). However, it is plausible that an exercise-induced depression of neutrophil function may slow tissue repair, wound healing, and, potentially, training adaptations in athletes undergoing high-level, chronic exercise training.

Recent research highlights the importance of carbohydrate and protein intake for neutrophil function following prolonged, strenuous exercise. A 23% decrease in neutrophil function was reported during recovery with placebo ingestion (no nutrition), while the consumption of a carbohydrate recovery beverage (1.2 g CHO/kg BM) or a carbohydrate–protein recovery beverage (1.2 g CHO/kg BM and 0.4 g PRO/kg BM) prevented the postexercise reduction in neutrophil function (Costa et al., 2009, 2011). It is suggested that the increase in insulin, which stimulates neutrophil function possibly through receptor activation, may maintain neutrophil function after immune-perturbing exercise (Costa et al., 2011). Moreover, the addition of protein to the postexercise carbohydrate beverage provides a favorable nutritional base to promote a positive nitrogen balance, potentially enhancing regeneration and training adaptations (Tipton & Wolfe, 2004).

Nutritional Strategies

1. Immediately after exercise, provide the body with a generous portion of carbohydrate-rich drinks and/or foods, which contain a small amount of protein to aid the recovery process (e.g., low-fat milk/yogurt-based drinks or recovery supplement drinks; lean meat-based sandwich with fruit juice or isotonic drink).

2. Drink nutrient-rich fluids in small but frequent amounts, until urine color becomes relatively clear and pale. Nutrient-rich fluids in the recovery period will help provide athletes with carbohydrates, proteins, electrolytes, and fluids, essential nutrients required for optimal recovery.

Anorexia: Stimulating Appetite But Managing Weight Gain

Chronically suppressed appetite and early satiety upon feeding are a common feature in athletes presenting UUPS. Undernutrition promotes anorexia, affecting the total quantity and quality of foods and fluids ingested resulting in malnutrition in accordance with exercise-stress load (French & Cecil, 2001). Clinical studies have identified a relationship between increased proinflammatory cytokine levels and alterations in hypothalamic and sensory regulation of appetite, albeit this has been mainly observed in cancer-related cachexia (Fearon et al., 2001). Limited research has been conducted on the association between UUPS and appetite; indeed chronic exposure to exercise-induced proinflammatory cytokines associated with high volumes of training may be a mechanism to explain the chronic reductions in appetite (Robson-Ansley et al., 2009; Scheede-Bergdahl et al., 2011). The influence of a chronic inflammatory state on appetite regulation in athletes has not been investigated but is a potential avenue for further research.

Nutritional Strategies

1. Small and frequent intake of nutrient-dense foods/drinks to aid in meeting nutritional requirements of exercising workloads but reduce total intake volume.
2. Avoid the excessive consumption of bulky, nutrient-diluted foods/drinks, e.g., whole fruits and vegetables, complex carbohydrates, and plain water, as these provide limited amounts of energy and nutrients per intake volume. Instead, focus on nutrient-dense foods/drinks, e.g., carbohydrate- and protein-dense meals, desserts, side dishes, and snacks; dried fruit; and nutritious fluids such as enriched or fortified milk shakes, fruit smoothies, and/or soups.
3. The addition of meal supplementation, e.g., powdered dietary supplements, carbohydrate supplementation, multivitamin, and mineral supplements can be beneficial in boosting total quantity and quality of nutrients consumed during periods of anorexia, if nutritional requirements are not consistently met through normal dietary habits.
4. Avoid drinking plain water with meals, which will accelerate gastric distension and induce rapid satiety (van der Lely et al., 2004). Fluid intake should be carbohydrate-dense, e.g., milk shakes, fruit smoothies, juices, soft drinks, sweet teas/coffees, milks, rice, soya and oat drinks, fortified or enriched soups.
5. Avoid social communication during feeding time, and concentrate on food/fluid intake, since slow eating will stimulate appetite suppression before meal is fully consumed.
6. Prepare and consume foods and drinks which are appealing and appetizing.
7. Dietary fat sources which present anti-inflammatory properties should be consistently included within dietary habits and may help restore appetite by their demonstrated ability in reducing inflammatory mediators. These include monounsaturated omega-9 (e.g., olive oil and products) and polyunsaturated omega-3 (e.g., oily fish such as salmon, mackerel, herring, trout, sardine, kippers, halibut, and fresh tuna), alternatively an omega-3 supplement 1–2 g/day if oily fish is not tolerable.

Microcytic Anemia

Microcytic anemia is commonly reported in the athletic population and is synonymous with iron-deficiency anemia. Microcytic anemia may occur during training overload and contributory factors include impact and intravascular hemolysis, hematuria, gastrointestinal blood loss, sweat loss, and nutritional insufficiency. Additionally, daily exposure to an acute systemic inflammatory response in athletes is associated with raised levels of the hepatic hormone, hepcidin. Hepcidin is the master regulator of iron homeostasis whose synthesis is induced by systemic inflammation. Hepcidin can inhibit dietary

iron absorption and promote the sequestering of iron by macrophages. Recent research indicates that prolonged exercise is associated with increased hepcidin levels and may be a major contributory factor to the development of microcytic anemia in athletic populations (Robson-Ansley et al., 2011).

Even though changes in hematological variables are not directly diagnostic of UUPS, levels below normal, healthy thresholds might contribute to the heightened sensation of fatigue reported in UUPS. Aside from exercise-stress load and inflammatory-related factors, microcytic anemia may be attributed to inadequate energy intake. Indeed, undernutrition is likely to result in low intake of foods containing high levels of biologically available iron and iron-assimilating nutrients, thereby promoting iron-deficiency and associated symptoms, e.g., general state of fatigue, weakness on exertion, malaise (Miret et al., 2003). Please refer to Chapter 19 for further information on iron requirements and iron status of athletes.

Nutritional Strategies: Dietary Sources Countermeasures for preventing the occurrence of iron-deficiency anemia in athletes presenting UUPS include the following:

1. Consuming a substantial diet of appropriate foods and fluids that meets the energy and nutrient requirements of the athlete.
2. Select foods with a high content of bioavailable heme iron sources, e.g., black pudding, red meats, white meats, liver/kidney, tinned fish, seafood, and eggs, as well as iron-assimilating nutrients, e.g., vitamin C: red pepper, guava, blackcurrant, cherry, kiwi, broccoli, Brussels sprouts, litchi, strawberries, peas, citrus fruits, tomatoes, melons, and fruit juices.
3. Avoid excessive intakes of iron absorption-inhibiting components such as tannins (found in tea), calcium (dairy- and flour-based food/fluid products), polyphenols, and phytates (generally found in vegetables and whole grains).

Nutritional Supplementation If iron-deficiency anemia is present:

1. Dietary iron availability may not be sufficient, even if consuming a diet rich in heme iron, to recover depleted iron stores. In this case, a period of iron supplementation, e.g., 100 mg daily for up to 4–6 weeks ferrous sulfate, may be warranted.
2. The dosage and timeframe of supplementation may be prescribed on a case-by-case basis with frequent hematological monitoring.

Summary

As athletes strive to improve their performance they will inevitably experience varying levels of fatigue and underrecovery, which will require effective management and monitoring by both the athlete and coach. An effective nutritional strategy to optimize recovery from both high-load exercise training experienced in overreaching and UUPS is fundamental to athletic success as well as protecting the long-term health of the athlete.

References

Alba-Loureiro, T.C., Munhoz, C.D., Martins, J.O., et al. (2007) Neutrophil function and metabolism in individuals with diabetes mellitus. *Brazilian Journal of Medical and Biological Research* 40, 1037–1044.

Banks, S. & Dinges, D.F. (2007) Behavioural and physiological consequences of sleep restriction. *Journal of Clinical Sleep Medicine* 3, 519–528.

Bryant, P.A., Trinder, J., & Curtis, N. (2004) Sick and tired: does sleep have a vital role in the immune system? *Nature Reviews Immunology* 4, 457–467.

Budgett, R., Newsholme, E., Lehmann, M., et al. (2000) Redefining the overtraining syndrome as the unexplained underperformance syndrome. *British Journal of Sports Medicine* 34(1), 67–68.

Burke, L.M., Kiens, B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22(1), 15–30.

Costa, R.J.S., Oliver, S.J., Laing, S.J., et al. (2009) Immune responses to immediate and delayed carbohydrate-protein feeding after prolonged strenuous exercise. *International Journal of Sports Nutrition and Exercise Metabolism* 19, 366–384.

Costa, R.J.S., Walters, R., Bilzon, J.L.J., et al. (2011) Effects of immediate postexercise carbohydrate ingestion with and without protein on neutrophil degranulation. *International Journal of Sport Nutrition and Exercise Metabolism* 21(3), 205–213.

Dinges, D.F., Douglas, S.D., Hamarman, S., et al. (1995) Sleep deprivation and human immune function. *Advances in Neuroimmunology* 5(2), 97–110.

Ellis, M., Gupta, S., Galant, S., et al. (1988) Impaired neutrophil function in patients

with AIDS or AIDS-related complex: a comprehensive evaluation. *Journal of Infectious Disease* 158, 1268–1276.

Fearon, K.C., Barber, M.D., & Moses, A.G. (2001) The cancer cachexia syndrome. *Surgical Oncology Clinics of North America* 10(1), 109–126.

French, S.J. & Cecil, J.E. (2001) Oral, gastric and intestinal influences on human feeding. *Physiology & Behavior* 74(4–5), 729–734.

Fry, R.W., Morton, A.R., & Keast, D. (1991) Overtraining in athletes. An update. *Sports Medicine* 12(1), 32–65.

Gleeson, M. (2000) Mucosal immune responses and risk of respiratory illness in elite athletes. *Exercise Immunology Review* 6, 5–42.

Lukaski, H.C. (2004) Vitamin and mineral status: effects on physical performance. *Nutrition* 20(7–8), 632–644.

Miret, S., Simpson, R. J., & McKie, A. T. (2003) Physiology and molecular biology of dietary iron absorption. *Annual Review of Nutrition* 23, 283–301.

Peake, J.M. (2002) Exercise-induced alterations in neutrophil degranulation and respiratory burst activity: possible mechanisms of action. *Exercise Immunology Reviews* 8, 49–100.

Robson, P.J., Blannin, A.K., Walsh, N.P., et al. (1999) Effects of exercise intensity, duration and recovery on *in vitro* neutrophil function in male athletes. *International Journal of Sports Medicine* 20, 128–135.

Robson, P. (2003) Elucidating the unexplained underperformance syndrome in endurance athletes : the interleukin-6 hypothesis. *Sports Medicine*, 33(10), 771–781.

Robson-Ansley, P.J., de Milander, L., Collins, M., et al. (2004) Acute interleukin-6 administration impairs athletic performance in healthy, trained male runners. *Canadian Journal of Applied Physiology* 29(4), 411–418.

Robson-Ansley, P.J., Gleeson, M., & Ansley, L. (2009) Fatigue management in the preparation of Olympic athletes. *Journal of Sports Sciences* 27(13), 1409–1420.

Robson-Ansley, P., Walshe, I., & Ward, D. (2011) The effect of carbohydrate ingestion on plasma interleukin-6, hepcidin and iron concentrations following prolonged exercise. *Cytokine* 53(2), 196–200.

Scheede-Bergdahl, C., Watt, H.L., Trutschnigg, B., et al. (2011) Is IL-6 the best pro-inflammatory biomarker of clinical outcomes of cancer cachexia? *Clinical Nutrition* 31, 85–88.

Seagle, H.M., Strain, G.W., Makris, A., et al. (2009) Position of the American Dietetic Association: weight management. *Journal of the American Dietetic Association* 109(2), 330–346.

Seifert, J., Harmon, J., & DeClercq, P. (2006) Protein added to a sports drink improves fluid retention. *International Journal of Sport Nutrition and Exercise Metabolism* 16(4), 420–429.

Shoham, S., Davenne, D., Cady, A.B., et al. (1987) Recombinant tumor necrosis factor and interleukin 1 enhance slow-wave sleep. *American Journal of Physiology* 253, R142–R149.

Smith, L.L. (2000) Cytokine hypothesis of overtraining: a physiological adaptation to excessive stress? *Medicine & Science in Sports & Exercise* 32(2), 317–331.

Smith, J. & Pyne, D.B. (1997) Exercise, training and neutrophil function. *Exercise Immunology Reviews* 3, 96–177.

Suzuki, K., Nakaji, S., Yamada, M., et al. (2002) Systemic inflammatory response to exhaustive exercise. Cytokine kinetics. *Exercise Immunology Reviews* 8, 6–48.

Tipton, K.D. & Wolfe, R.R. (2004) Protein and amino acids for athletes. *Journal of Sports Sciences* 22(1), 65–79.

van der Lely, A.J., Tschop, M., Heiman, M.L., et al. (2004) Biological, physiological, pathophysiological, and pharmacological aspects of ghrelin. *Endocrine Reviews* 25(3), 426–457.

Vgontzas, A.N., Papanicolaou, D.A., Bixler, E.O., et al. (1999) Circadian interleukin-6 secretion and quantity and depth of sleep. *Journal of Clinical Endocrinology and Metabolism* 84(8), 2603–2607.

Walsh, N.P., Gleeson, M., Pyne, D.B., et al. (2011a) Position statement. Part two: maintaining immune health. *Exercise Immunology Reviews* 17, 64–103.

Walsh, N.P., Gleeson, M., Shephard, R.J., et al. (2011b) Position statement. Part one: immune function and exercise. *Exercise Immunology Reviews* 17, 6–63.

Chapter 34

The Traveling Athlete

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Introduction

In preparation for travel to sporting events, it is important for athletes to be proactive with their tour and competition nutrition plans as inadequate nutrition can have a significant negative effect on health and athletic performance. In the weeks prior to and during sporting events, nutritional fueling and hydration strategies are of utmost importance, especially if the competition will be in a foreign country that has different customs and food cultures. This chapter is designed to help athletes prepare for traveling to sporting events. The four main topics of discussion include travel nutrition guidelines, plan B, food safety, and special issues.

Travel Nutrition Guidelines

Pretravel (1 Month Prior)

Before the athlete even step onto the plane or into their car to head to their competition venue, their travel nutrition plan comes into play. One month prior to departure, athletes should commence research and planning for their trip. Where are they going? What are the cuisines, culture, and eating habits of the country they will be visiting? Where will they be staying? What's the plan for meals? Will they be cooking for themselves, catered for, or eating out? What is the anticipated daily schedule? These are just some of the questions athletes will need to consider and research. Planning and preparation are key to successful eating and performance when traveling.

Plan the Flight Optimal fueling and hydration has become increasingly challenging for athletes who fly regularly. With all airlines, but especially with discount airlines, there are limited meal services available to passengers and airport security organizations generally do not allow fresh or liquid food items to be taken through security checkpoints. Some countries have an agency that protects all of their airports (such as Australia, where the Federal Police are responsible for security at all major airports and the United States, where airport security is the responsibility of the Transportation Security Administration (TSA)). It is unwise to make assumptions about what is allowed through security, and confiscation of items is inevitably disruptive. Knowing which items are not permitted ahead of time allows athletes to plan accordingly for their travel. Some airports have good shopping facilities after security, so again some advance information can be very helpful.

The current regulation enforced by the TSA for carry-on luggage is the 3–1–1 principle, which has gained international acceptance, and is now followed by most countries. What this means is that the volume of liquids, gels, and aerosols are limited to bottles of 3 ounces (~100 ml) or smaller, in a 1 quart-sized (1 liter, 20 cm × 20 cm) clear zip-top bag, and one bag per traveler is permitted (Figure 34.1).

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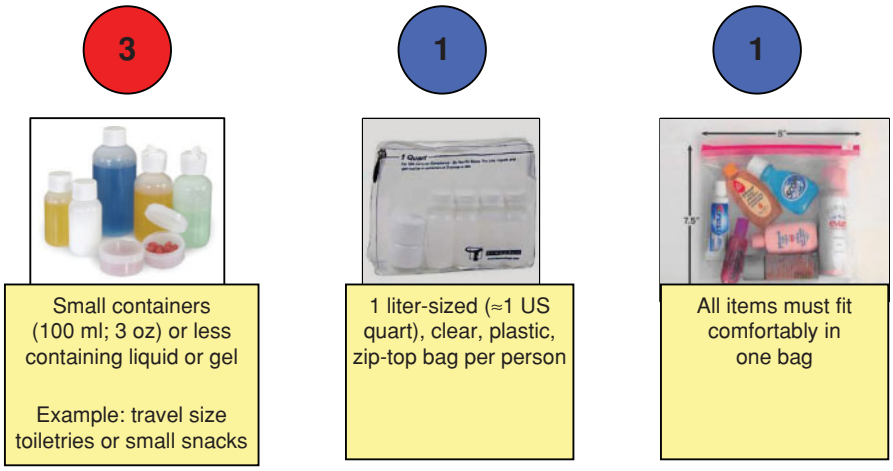

Figure 34.1 The TSA 3–1–1 principle.

Any toiletries, liquids, or gels in volumes in excess of 100 ml must be packed in checked luggage.

On arrival at many international destinations, all baggage will be inspected for food brought with passengers. All such items carried in checked baggage and/or carry-on luggage must be declared. Failure to declare food products can result in confiscation of the items and a significant fine. Inevitably there will be delays for the athlete, and possibly also for accompanying teammates, if prohibited items are identified. Specific guidelines may not be available about the admissibility of specific foods because disease and pest outbreaks occur all over the world without notice.

The general guidelines listed below will provide the athlete with assistance as to which foods are typically allowed during international travel. Athletes should be reminded to declare all food on arrival. In most cases the food will be returned to them after inspection.

- Liquids and gels brought from home or purchased before reaching the security checkpoint in containers *larger* than 100 ml (e.g., sports drinks) are allowed only in checked luggage.
- Liquids and gels brought from home or purchased before reaching the security checkpoint in a 100 ml or smaller container and in a 1 liter, clear zip-top plastic bag can be part of the carry-on luggage.
- Beverages purchased after security screening can be part of carry-on luggage.
- Medications are allowed in reasonable quantities exceeding 100 ml and are not required to be in the zip-top bag. These items must be declared for inspection at the checkpoint.
- Dry snack foods such as sports bars, crackers, muesli bars, pretzels, or rice cakes can be part of carry-on luggage.

Before athletes get to the airport they should be aware of the baggage allowance. Food items suitable for national and international travel can be packed around sporting equipment and/or placed in excess luggage.

Research the Destination If athletes will be traveling to a country that they have not been to before, it is worth researching the destination before they go. Throughout the world there is a vast array of customs and cultures. To assist in minimizing culture shock, athletes should consider the following before they depart:

- What is the typical meal pattern?
- What is the timing of meals?
- What is the cuisine?

- What are the main protein source, grain, vegetables, fruits, condiments, and style of cooking?
- What eating utensils does the country adopt?
- What are the grooming expectations and dress code?
- What language is spoken?

Prior to departing it may be worthwhile for athletes to trial the new cuisine. Make a few new dishes at home or dine out at some local restaurants serving that cuisine. The fun side of travel can be immersing oneself in the new culture.

If possible, athletes should obtain a map of their destination and locate the competition venue, their accommodation, local restaurants, shops, and supermarkets. If they know someone who has traveled there before, they should ask them some questions to find out as much practical information as they can before they depart.

Choose the Catering Style There are a number of options when it comes to your meals. In an international games competition, a dining hall is generally available to provide all of the meals and cater for individual needs (refer to Chapter 36). However, this will not be the case with every competition or travel experience. Athletes will need to determine if their meals will be catered for, if they will have the facilities for cooking for themselves, or if they will have to rely on buying all meals and eating out.

Self-catering allows greater flexibility in meal times and locations and greater control on food choices. Cooking skills, budget, and access to shops, cooking, and storage facilities will help determine if self-catering is an option. Self-catering does involve a lot of organization, planning, and workload. Athletes should consider self-catering when they have a long-term stay, a low budget, suitable facilities, and special dietary requirements. When self-catering, plan meals that are quick, easy, foolproof, appetizing, have limited number of ingredients, and can be partially prepared in advance. Minimal cooking equipment also means limited cleaning up.

Quick and easy meal ideas for self-catering include

- spaghetti bolognaise;
- pasta with a vegetable-based tomato sauce;
- stir fry chicken/pork/beef and vegetables with rice, noodles, or couscous;
- grilled fish/chicken/beef/pork with vegetables or salad;
- oven-baked risotto;
- homemade pizzas—pizza bases or pita pockets topped with lean meats, lots of vegetables, and a sprinkle of cheese;
- burritos—lean meat with salsa and salad in a wrap.

When athletes are traveling as part of a team who are self-catering the following should be considered:

- Check for likes and dislikes and determine any food allergies or intolerances.
- Assess cooking skills and motivation of athletes.
- Cooking classes should be held for the athletes who need it.
- Investigate cooking facilities and equipment at the accommodation.
- Divide workload and develop a roster with the athletes—shopping, cooking, cleaning.
- Plan to eat out every 3–4 days or have a team meal (to break it up).
- Place a grocery order in advance and have it delivered.
- If catering for all meals then consider self-catering for breakfast and lunch only.

For athletes staying in hotels, enquire if there is an option for meals to be provided by the hotel's restaurant. If not, then local restaurants should be investigated before leaving home. Information on the menu options, opening hours, and ability to cater for special dietary requirements needs to be obtained. When traveling with a large group, book the restaurant and plan the menu with the restaurant ahead of time. Buffet-style eating allows athletes a range of food choices, but it can be easy to overindulge.

Snacks are an important component of fueling for athletes. Quality snacks can be difficult to find when traveling. It is recommended that all athletes take a supply of nonperishable snacks with them or send them ahead of time to their destination. All snack foods should be checked with customs. For teams,

a communal snack room should be set up at the accommodation site.

Suitable snack foods include

- muesli, cereal, and fruit bars;
- breakfast cereal;
- crackers;
- spreads—jam, honey, peanut butter;
- trail mix—nuts, seeds, dried fruits;
- sports products—bars, gels, sports drink mix, protein powders.

Devise a Plan B Unfortunately even the best-laid plans sometimes come unstuck, so it is always worth having a back-up plan or plan B. What will athletes do if the dining hall is not open or they don't like the food? What if their accommodation does not have the equipment or facilities they expected? Before athletes leave home, they should know their plan B. This will be discussed further in the next section.

Plan the Competition Meal Plan and Supplement Regimen A month before athletes travel is the ideal time for them to sit down with their sports dietitian and design their competition meal plan. The preparation should consist of a meal plan, shopping list prior to and upon arrival, recipes, restaurant list, and supplement requirements.

Their supplement regimen should already be planned as part of their training diet well before they are due to travel. However, it may be worth discussing with the sports dietitian before they depart any supplements that may be necessary while traveling. A multivitamin may be beneficial to help them meet all of their nutrient needs, especially if they are traveling to a destination where their food intake may be limited in variety and quantity. A supplement to boost their immune system and gastrointestinal health may also be recommended.

Pretravel (1 Week Prior)

A week prior to travel should be the time athletes organize and purchase last minute items. This consists of organizing their in-flight meals, purchasing equipment and foods to take with them, and commencing their hydration and supplementation plan.

Confirm the Flight At least 1 week prior to leaving, athletes should consider contacting the airline to arrange special in-flight meals. Most airlines offer meal requests to accommodate medical, religious, or other dietary specifications. Meal options include vegetarian (lacto-ovo, vegan, or Asian-vegetarian), low-fat/low-calorie, high-fiber, low-cholesterol, low-sodium, gluten-free, low-sodium, and traveler's lighter choice meals (chilled fruit plates). At this time they can also enquire about the timing of the meals during their flight.

It is a good idea for athletes to prepare a plan or schedule for their in-flight time. They should consider when they will eat their meals, when they will sleep, stretch, etc. This can help them to ensure they eat enough but not too much and get enough sleep.

Purchase the Travel Supplies While packing their bags, athletes will need to include any food and equipment they plan to take with them. The week before departure is a good time to go shopping and stock up on all the essentials that they need. The following should be considered for purchase: favorite foods, products to cater for an unsafe or poor nutritional quality menu, dietary supplements, and sports products. If the athletes have any food sponsors they should call their international suppliers to arrange the delivery of sponsored products to their hotel.

Optimize Hydration and Commence Supplementation Due to the low humidity of the aeroplane cabin environment, there is a risk of becoming dehydrated. Athletes are unable to rely on the cabin service for sufficient fluid. The serving sizes of drinks are small and the frequency of distribution is limited. In-flight dehydration can cause excess thirst, scratchy and bloodshot eyes, dry skin, headaches, constipation, and bloody noses. To help prevent dehydration athletes should be prepared by optimizing hydration in the day leading up to the flight. They should also bring their own empty water bottle and fill it after passing through security prior to boarding. If the athletes are planning on taking any immune-boosting or multivitamin supplements, now is the time to start taking these.

During the Trip

The trip starts as soon as the athlete leaves home and not when they arrive at their destination.

Challenges can commence before and during the flight as well as upon and during their stay at their destination. Be prepared for delays at the departure airport: here at least, food and drink should be available at the airport catering outlets, though the choice may not be appealing. For longer delays, the airline is obliged to supply some food for passengers, but what is offered is often unsuitable.

During the Flight During the flight, there may be hours spent with very little activity which may lead to boredom. Athletes should make an attempt to get up and walk around at least once every 2 hours. Requesting a specific seat assignment near the aisle allows easier access to the bathroom and the ability to walk around and stretch.

Athletes should set their watch to the time zone of the destination they are traveling to and attempt to adjust their eating and sleeping schedule accordingly. This will help reduce jet lag and adjust their body clock. They should avoid over- or undereating by planning ahead and preparing some of their own snacks rather than relying on the airline food. This will allow them to control the amount of energy they consume and will decrease the temptation to snack excessively on other foods, especially if they have limited choices during a layover. High-fiber snacks, along with plenty of fluid, are encouraged for preventing constipation on long flights. It is also important for an athlete with increased energy needs to have extra snacks available to supplement meals they may receive in flight. Food available for sale at airports tends to be expensive and limited in nutritional value. Athletes should pack a supply of emergency food in case of unexpected delays.

It may also be beneficial for some athletes to sip on a sports drink as the small amount of sodium may stimulate a greater fluid intake. They should aim to drink 250–500 ml/hour of liquid during the flight. Athletes who normally consume tea, coffee, and cola drinks should limit these beverages due to increased urine production, but it is probably better not to avoid caffeine-containing beverages entirely as the diuretic effect of the caffeine in these drinks is small and there is a real risk of caffeine-withdrawal symptoms. It is usually best to avoid alcohol when traveling. Lastly, athletes should monitor urine color and frequency of urination, as these are indicators of their hydration status. Urine should be pale in color and frequency of urination should be every 2–3 hours.

When traveling by road, athletes should pack their own meals and continue with their nutrition plan. They should avoid being tempted to stop at service stations and shops to buy treats. An ice box filled with sandwiches, fresh fruit, yogurts, and drinks will prevent the need to purchase meals with potentially poor nutritional value. Athletes should try to stick to their normal meal plan and pattern and avoid excess snacking.

Orientation on Arrival Upon arrival athletes should try to establish a new routine quickly. After arriving at their destination and settling into their accommodation is a good time to orient themselves with what is available in terms of food. They should visit the dining hall, tour local restaurants, and locate their nearest supermarket to check what they stock. Reading the local food labels and asking for assistance may be required upon arrival. As most sporting venues provide poor and/or limited food choices, test the food service program at the venue and also check if it has accessible water outlets that are safe to drink. When at the venue athletes should always carry emergency snack foods and their sporting fueling and recovery needs with them. After their orientation to their new destination it is important to determine whether to adopt their plan A or B in food service.

A change of environment, e.g., heat, cold, or altitude, can also alter their nutritional needs. Introduce the necessary changes upon arrival. See Chapter 35 for further information.

Strategy for Illness or Injury Several types of illnesses can be attributed to eating contaminated foods or water during international travel. The majority of illnesses come from bacteria, viruses, parasites, and chemicals that contaminate food or water. If athletes are sick with food poisoning the following is recommended:

Step 1:

- Contact the team doctor if available, otherwise the manager or coach.

- Rest.
- Drink bottled water/ice as tolerated to attain and maintain hydration. Be careful about ice—ensure it is made from clean water and has been handled and stored without coming into contact with potential infectious agents.
- Do not handle food for others as your bacteria can be passed on to them.

Step 2:
- In cases of water loss from vomiting or diarrhea, use an electrolyte replacement drink to maintain hydration.

Step 3:
- Eat a bland diet while recovering. This means no spices, rich sauces, foods that contain lactose, e.g., milk and other dairy, or foods with a wide combination of flavors.

Step 4:
- When you are able to consume a full diet, introduce probiotics to help replace the bacteria in your gut.

If an athlete is injured or ill they will need to modify their competition diet to cater for surgery, infection, and/or treatment. Often the effect of an acute injury or illness is an increase in metabolic rate, a decrease in protein synthesis due to disuse, and a risk of weight loss due to limited intake of food. If possible, the athlete should make contact with their sports dietitian for advice.

Upon Return

When an athlete arrives home from their trip, it is wise for them to reflect on their experience and review their travel nutrition plan. What worked? What didn't? Record some recommendations for future travel to that region. It is an invaluable starting point for preparing for future trips and for sharing their knowledge with others.

If they have experienced any issues while traveling, now is the time to rectify them. They may need nutritional support to recover from an illness, overcome jetlag, reduce weight gained or gain weight lost, or overcome fatigue. The athlete should make an appointment as soon as they can with their sports dietitian for advice.

Plan B—The Backup Plan

Despite the best of intentions and all the planning, sometimes things do not quite go as expected. The facilities or equipment anticipated for cooking may be inadequate. The foods available may be unfamiliar and inaccessible, which can lead to suboptimal fueling, decreased performance, and other complications. So it is ideal for athletes to have a back-up plan or plan B ready to implement.

Travel Nutrition Kit

The travel nutrition kit provides a good back up. It is designed to help athletes with many of the food challenges experienced when traveling. The kit will provide the cooking supplies necessary to prepare meals anywhere in the world (see Tables 34.1 and 34.2).

The travel kit should include the following:

- hot pot or Birko travel cooker;
- travel power converter;
- measuring cups and spoons;
- plastic containers and zip-lock bags;
- large plastic bowl and cutlery;
- recipe cards;
- renewable supply of shelf-stable food (see below);
- tea towel, detergent, and cleaning wipes.

Food Safety

Each country has different food hygiene standards, sanitation, and water quality. In addition, the stress of traveling and competing may reduce your resistance to illness.

Several types of illnesses can be attributed to eating contaminated foods during international travel. The majority of illnesses come from bacteria, viruses, parasites, and chemicals that contaminate food or water. Common illnesses include traveler's diarrhea (which is often given various descriptive names such as Montezuma's revenge), infectious hepatitis, typhoid fever, cholera, and illness from the Giardia parasite. Symptoms of food-borne

Table 34.1 Foods for your travel kit

Nutrition kits should be used as a supplement, not as a replacement, to meals provided unless the meals are unsafe or unpalatable to the athlete. Athletes need to supplement their travel nutrition kit with the following shelf-stable foods. Prior to leaving excess packaging should be removed from the products to keep the weight down.

Carbohydrates:
- instant rice;
- instant mashed potato;
- pasta;
- couscous;
- quinoa;
- granola/dry breakfast cereals,
- instant oats;
- dried crackers, rice cakes, biscuits;
- whole-wheat tortillas;
- dehydrated vegetables, e.g., corn, peas, and beans;
- dried and pouches of fruit.

Protein: (all shelf-stable)
- chicken pouch;
- tuna pouch;
- salmon pouch;
- shelf-stable tofu;
- soy or whey protein powder;
- powdered liquid meal supplements;
- milk powder;
- nuts.

Fats:
- olive oil.

Condiments:
- jam, honey, peanut butter, mustard, vegemite;
- spice blends, herb blends, and pepper.

Table 34.2 Recipes that you can make with your travel kit

What can you make with your travel nutrition kit?

- Chili chicken and rice
  1 chicken breast pouch (100 g)
  1 teaspoon of chili powder
  1 packet of dehydrated vegetables, e.g., corn, peas
  1 packet of quick/minute rice (roasted garlic and olive oil) (250 g)
  Salt and pepper

Cook rice according to packet in hotpot, then mix chili, vegetables, and chicken breast and heat through for around 5 min.

500 kcal, 5 g fat, 72 g carbohydrates, 38 g protein

- Tuna/salmon rice or couscous
  1 packet of quick/minute rice (garden vegetables) or couscous (250 g)
  Pouch of salmon or tuna (100 g)
  Salt and pepper

Cook rice/couscous according to packet, then mix in either salmon or tuna and season with salt and pepper.

417 kcal, 3 g fat, 66 g carbohydrates, 29 g protein

- Fiesta/Mexican rice
  1 packet of quick/minute rice (roasted chicken flavor) (250 g)
  1 pouch of chicken (100 g)
  1/2 cup dehydrated vegetables, e.g., peas
  2 wheat tortillas
  1/2 pack of taco/burrito seasoning

Heat rice according to the packet instructions. Mix chicken, vegetables, and seasoning in with the rice and heat through. Place heated mix on tortillas.

622 kcal, 8 g fat, 92 g carbohydrates, 42 g protein

- Chili-corn mashed potatoes with tuna
  1 pouch of tuna (100 g)
  1 cup of instant mashed potato flakes
  1/2 cup dehydrated vegetables, e.g., corn, peas
  1 teaspoon of chili powder
  Salt and pepper

Prepare mashed potato according to packet; then add tuna, corn, and chili powder and heat through. Season with salt and pepper as desired.

384 kcal, 6 g fat, 43 g carbohydrates, 32 g protein

- Breakfast quinoa/oats
  1 packet quinoa or instant oats
  2 tablespoons of dried fruit
  1 tablespoon of walnuts
  1 teaspoon of honey

Prepare quinoa or oats according to packet and leave to slightly cool; then add fruit, honey, and walnuts. Serve with yogurt if desired.

295 kcal, 10 g fat, 452 g carbohydrates, 6 g protein

illness generally include diarrhea, vomiting, headache, malaise, fatigue, and/or dehydration. If any of these occur, try to replace lost fluids and electrolytes as soon as possible. Utilize a safe water supply and reintroduce a bland diet of dry toast, crackers, and rice. Avoid milk and foods containing fat and alcoholic beverages.

Athletes can protect themselves by developing good personal hygiene and practicing these food safety guidelines:

- *Uncooked fruits and vegetables:* Contamination of fruits and vegetables can occur during growth, harvest, transportation, preparation, and service. The fields used to grow produce are often treated with manure or sewage. Contaminated water may also come into contact with the raw fruits or vegetables during the washing process. Bacteria on the outside of fruits can be transferred to the inside when the fruit is peeled or cut. Remember to clean all produce with bottled water and thoroughly cook vegetables prior to consumption.
- *Poultry, meat, seafood, eggs, and dairy:* Foods from animals naturally contain bacteria and other contaminants that could increase chances of becoming ill. Thus, it is very important for these foods to be cooked thoroughly to destroy disease-causing organisms. Seafood should easily flake with a fork. Avoid eating raw sources. Avoid large reef fish as they can accumulate chemical toxins during their lifetime. Eggs should be properly cooked and firm to the touch.
- *Cleanliness and temperature:* Avoid eating and purchasing foods from street vendors, buffets, restaurants, and retail markets that appear unclean and if food temperature is not appropriate. Pay attention to the cleanliness of the facility. If the facility and surroundings do not look sanitary, do not take a chance. Temperature is very important because disease-causing bacteria do not grow well below about 5°C or above about 60°C. If hot foods are not hot and/or cold foods are not cold, they should not be eaten.
- *Pasteurization:* Do not consume unpasteurized dairy products (milk and cheese) or drink unpasteurized fruit juices. Unpasteurized milk and dairy products could contain harmful pathogens and are not safe to eat, drink, or to be used in making foods. The United States and Australia mandate pasteurization of dairy foods but other countries may not have these regulations. Read the food labels and ask questions. The "radura" symbol below is used internationally to mean that the food in the package has been pasteurized.

= Radura symbol

- *Water safety:* As with any international travel and competition, it is always best to rely on bottled water for drinking and brushing your teeth. Most ice machines are not treated so it is best to drink room temperature beverages and skip the ice. Consume fluids from containers with unbroken seals and avoid drinking water from showers and pools.
- *General hygiene:* Always wash your hands before eating or handling food and after using the bathroom or blowing your nose. Carry hand sanitizer on you at all times, in case there is nowhere to wash your hands appropriately.

Athletes should use these guidelines to make the best decision possible when eating or drinking during international travel. Becoming ill from contaminated food at home is uncomfortable enough, but being in a foreign country can be much more challenging. Consuming familiar and consistent food is important for optimum performance. It is important to avoid new foods until after the competition.

Special Issues

USA Team at the Beijing Olympic Games: Plans A, B, and C

Plan A: Olympic Village Plan A for all American athletes residing at the Beijing Olympic Village was to eat all meals and snacks at the Village dining halls. The international food company Aramark,

which has catered for 13 previous Olympic Games, provided the meal service in Beijing. The main dining hall was open 24 hours a day and had a seating capacity of 5200 people.

Plan B: High Performance Centre Restaurant Plan B for the American team was to eat their meals at the High Performance Centre Restaurant. Beijing Normal University (BNU) housed the United States Olympic Committee (USOC) dining hall for their athletes and their credentialed support staff. The USOC dining services staff and a US-based catering company provided all the food and equipment at BNU.

Plan C: National Governing Body (NGB) and Athlete Responsibilities Due to the concerns with food safety at the Beijing Olympic Games, the United States designed a plan C. It is important for NGBs and athletes to assume a certain level of responsibility to assist them in obtaining optimum nutrition during the Olympic Games. Helpful nutrition tips included:

- packing foods and empty water bottles on the domestic/ international travel to and from pre-Olympic training sites and China;
- working with their USOC Sport Dietitian to provide hotel menu guidelines for pre-Olympic camps;
- ensuring specific nutrition requirements are met at the different training venues by taking foods and fluids from the Olympic Village or BNU;
- utilizing their travel nutrition kit as required.

Special Issues: Competing in Japan—Women's Tennis Association (WTA)

On March 11, 2011 Japan experienced a level 9 earthquake that subsequently triggered a tsunami. This series of events caused significant damage to the Fukushima Daiichi Nuclear Power Plant, which caused radiation exposure to the local region.

Radiation contamination occurs when radioactive material is deposited on or in an object or person. When large amounts of radionuclides are discharged into the environment they affect foods by traveling via air and falling onto the surface of fruits, vegetables, and animal feed; contaminating rain, water, or snow; and by being taken up by plants, seafood, and animals. The exact health effects depend on the radionuclides been ingested and the amount.

The WTA methods adopted to ensure safety of players included the following:

- Meals to be consumed only at the hotel and tournament athlete cafe.
- Tournament food samples, e.g., leafy greens, milk, beef were sent to the isotope research institute for radioactive testing.
- Geiger counters and radiation monitoring badges were utilized.
- A portable surface dosimeter was bought and utilized with all meals.
- A list of food sources and their locations of purchasing was provided by the hotel and tournament caterers and reviewed by the WTA sport dietitian.
- Extra-nutritional supplements were provided to players in case insufficient nutrients were obtained during the week.
- Sponsor products were boxed to Japan and carried by all staff.
- Athletes were encouraged to carry additional food products to the tournament.
- Potassium iodide pills were carried by the WTA medical staff.

Special Issues—Consumption of Steroid-Contaminated Meat and Positive Urine Tests

In 2011 the Mexican Football Federation revealed a small number of national soccer players had tested "positive" for the banned drug Clenbuterol, reportedly as a result of consuming tainted beef. Subsequent tests using a more sensitive assay system revealed players from 19 of the 24 teams competing at the FIFA U-17 World Cup had adverse findings for the banned anabolic agent. Clenbuterol, which is not a steroid, but a beta-2 sympathomimetic and central nervous system (CNS) stimulant, was found in 109 of the 208 urine samples collected at the competition (http://www.fifa.com/aboutfifa/footballdevelopment/medical/news/newsid=1528706/index.html). Clenbuterol has also been found recently in the urine of 22 out of 28 non-athletic visitors to China, again presumably as a

result of eating food from animals where the drug had been administered (Guddat et al., 2011).

Farmers in Mexico, like other countries such as the United States, China, and Australia, routinely raise cows with the assistance of steroids and other pharmaceuticals to enhance their growth rate and lean-to-fat ratio. Since 1998, the European Union has prohibited the use of these drugs in animal production. While these regulations are not endorsed in the United States and Australia, these countries do have regulations controlling their use, and they adhere to strict food safety codes to ensure their meat produced is safe for the consumer. This is not the case in all countries.

Traveling athletes need to be mindful of the potential risk of consuming meat that has been produced in unregulated countries. In addition, some meats and offal, such as those derived from boar (both wild and domestic) can have naturally high concentrations of steroids (i.e., nandrolone and its derivative norandrostenedione), which may be potentially transmitted to the consumer.

Acknowledgment

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Sources for More Information

Australian Institute of Sport
http://www.ausport.gov.au/ais/nutrition
Transportation Security Administration
www.tsa.gov/

Sports Dietitian Australia
http://www.sportsdietitians.com.au/content/2495/FactSheets/

United States Olympic Committee
http://www.teamusa.org/resources/usoc-sport-performance/nutrition

Reference

Guddat, S., Fusshöller, G., Schwenke, A., et al. (2011) *Clenbuterol—Regional Food Contamination as Possible Source for Positive Findings in Doping Control?* Lecture at the 29th Cologne Workshop on Dope Analysis, February 14, 2011.

Chapter 35

Environment and Exercise

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Introduction

Athletes compete not just against each other, but also against the elements. Environmental heat, cold, altitude, and air quality can greatly impair training and competition performance. Although physiological, behavioral, and technological adaptations allow athletes to withstand the most extreme environmental challenges, the potential detriments to athletic competition are considerable. This chapter considers how environmental heat, cold, altitude, and air quality affect sports that require prolonged physical activity and, thus, prolonged environmental exposures. Emphasis is placed on realistic competitive weather conditions observed in summer and winter Olympic venues, including air temperatures between −5°C and 30°C and altitudes of up to 3000 m. Acute and adaptive physiological responses may, however, include more extreme conditions for illustrating basic principles. Air quality is discussed largely in light of laboratory evidence, complemented by limited field observations. The nutritional needs for exercise performed in widely different environments do not differ drastically from ordinary recommendations for training and competing athletes. However, unique nutritional concerns for each environment are integrated and discussed briefly alongside implications for human health. Review articles, book chapters, and books are relied upon heavily to succinctly convey classical concepts covered in this chapter. Those reviews should be consulted for a more thorough treatment of each topic and for the unique contributions of the many original research articles cited therein.

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Homeostasis in Harsh Environments

Heat and Cold

Subtle temperature changes sensed by thermal receptors in the body core and skin transmit afferent temperature information to the brain. In response to body heat gain or loss, temperature-sensitive neurons within the preoptic area of the anterior hypothalamus (PO/AH) become stimulated. Neuronal activation of thermoregulatory effector responses occurs in proportion to the displacement of body temperature from its set-point (Gonzalez & Gagge, 1996). These responses work as a negative feedback loop to control thermal balance via heat loss (sweating, skin vasodilation), prevention of heat loss (skin vasoconstriction), or heat gain (shivering). Skin blood flow is increased or decreased through sympathetic neural activation to vary convective heat transfer from the core to the skin as circumstances dictate. Skin temperature (i.e., the body shell) can vary by more than 20°C in different environments, but temperature of vital organs (body core temperature) varies by only about 4°C (Gonzalez & Gagge, 1996).

Physiological control of thermoregulation is complemented by biophysical interactions between man and environment, which can be described using a simplified heat balance equation of the form:

$$M - W \pm (R \pm C) - E = S, \text{ (Gonzalez \& Gagge, 1996)}$$

where M is metabolic rate, W is energy used to perform mechanical work, R and C represent radiation and convection, and E is evaporative heat loss. A net gain or loss of heat is reflected by a change in heat storage (S). $M - W$ (M_{net}) represents the net heat of metabolism, or the endogenous heat load. The relative contributions of dry ($R + C$) and evaporative heat exchange to total heat loss vary with the environmental conditions, or the exogenous heat load. As ambient temperature increases, the gradient for dry heat exchange diminishes and evaporative heat exchange becomes more important. When the ambient temperature equals or exceeds skin temperature, evaporative heat exchange will account for virtually all heat loss (Figure 35.1) (Sawka et al., 1996). The key to body heat removal by sweating is evaporation. When the requirement for evaporative heat loss (E_{req}) exceeds the potential of the environment (E_{max}), exercise duration must be shortened or the intensity decreased, or both to achieve thermal balance. This occurs most commonly in hot, humid weather when the air-to-skin water vapor pressure gradient narrows and skin wetness approaches 100% (Gonzalez & Gagge, 1996). Under these conditions, evaporative sweating declines and sweat drips from the skin, restricting the final major avenue of heat loss.

The most commonly used environmental index of heat strain, particularly in sport, is the wet bulb (T_w) globe (T_g) temperature (T_a) index, or WBGT (Gonzalez, 1995). The WBGT is a weighted index which recognizes the importance of evaporative cooling when air temperature is hot, such that the formula, $0.7T_w + 0.2T_g + 0.1T_a$ clearly weights T_w of primary importance (70% of index). However, a hot air temperature (T_a) is a prerequisite, since the stress of any common "air moisture" index (e.g., relative humidity) varies directly with air temperature. For example, at the same relative humidity of 50%, T_w is 17°C more at 40°C than 20°C, making the WBGT twice as large (30°C vs. 15°C). For cold weather, wind is an important physical factor and thus the wind chill index (WCI) is often used to gauge environmental cold severity, but the WCI does not account for activity level or clothing. Figure 35.2 (Gonzalez, 1995) shows how the integration of exercise intensity and clothing insulation (clo) should be used to better equate cold stress in terms of the minimum required clo at any given level of activity. Typical warm (shorts, singlet) and cold weather athletic clothing (cross country ski attire) range from about

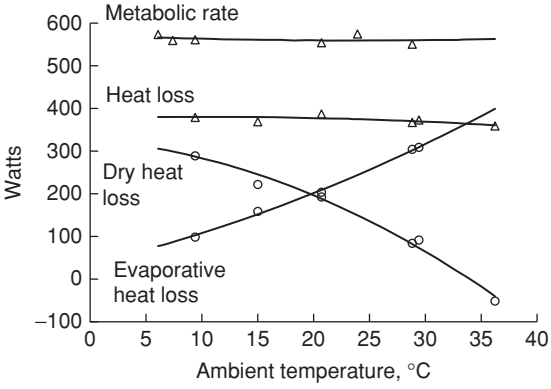

Figure 35.1 Heat-exchange data averaged over 1 hour of exercise at each ambient temperature shown. As ambient temperature rises, heat loss remains stable but the contribution from evaporative heat loss increases while dry heat loss decreases. From Sawka et al. (1996) (originally redrawn from Nielsen, 1938).

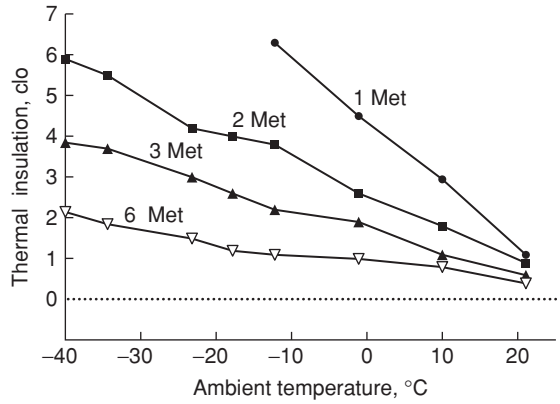

Figure 35.2 Thermal insulation in clo required at different ambient temperatures (rh = 40–50%, wind speed = 1 m/s) to maintain reasonable comfort for a person exercising at a range of exercise intensities. 1 Met = 105 Watts, or typical energy expenditure at rest. From Gonzalez (1995).

0.5 to 2.0 clo. The choice to doff or don clothing as the environment warms or cools is an example of behavioral human temperature regulation.

Altitude

The primary stress of high altitudes relates to the curvilinear decrease in barometric pressure (P_B) and partial pressure of oxygen (PO_2) with increasing elevation (Ward et al., 2000). Peripheral chemoreceptors of the carotid and aortic bodies sense acute changes in PO_2, resulting in protective autonomic reflexes such as hyperventilation, tachycardia, and arterial vasodilation that collectively defend arterial oxygen saturation (S_aO_2) (Ward et al., 2000). The magnitude of the response depends not only on the altitude, but also on the demand for oxygen supply (i.e., rest vs. exercise). In addition, air temperature decreases by ~1°C for every 150 m ascent above sea level, which tends also to reduce the absolute humidity of the air (Ward et al., 2000). For altitudes below 3100 m, the inspiration of cooler, dryer air appears to balance increases in ventilation frequency such that total respiratory water losses are similar to sea level (Hoyt & Arnold, 1996). At altitude in countries nearer the equator, the warmer climate is enough to offset the relative decline in air temperature and humidity, making competition above 1500 m both a thermal and hypoxic challenge. This too may be exacerbated by solar radiation, which, through a combined effect of reduced air density and water vapor, increases by almost 40% when ascending from sea level to 1500 m altitude (Ward et al., 2000).

Adaptation to Harsh Environments

In stark contrast to cold, where behavioral thermoregulation is key (Young, 1996), human adaptations to environmental heat and altitude are robust. Several acute and chronic physiological adaptations occur upon exposure to heat and altitude. Acclimation and acclimatization are terms often used to describe and distinguish between temporary phenotypic adaptations to simulated (i.e., chamber studies) versus genuine environmental exposures, respectively. In this chapter, the term acclimation will be used nonspecifically.

Heat

Among the most important adaptations to heat are an earlier onset and higher rate of sweating, conservation of electrolytes, and expansion of plasma volume (Sawka et al., 1996), all of which translate into a reduction in body temperature and cardiovascular strain. Sweating rates often double while electrolyte losses are halved (Sawka et al., 1996). Importantly, heat acclimation ultimately increases water needs (see Chapter 14). Athletes will therefore benefit by accurately tracking and replacing body water losses (Table 35.1). As total salt losses can still be substantial (volume × concentration), the addition of salt (or salty food choices) to the diet may be useful (Table 35.1; see Chapters 14 and 16). Various combinations of endogenous and exogenous heat stress can be used to achieve heat acclimation, but 10 consecutive days of exercise-heat exposures of 100-min duration is considered optimal to achieve full acclimation (Sawka et al., 1996). The complete withdrawal of daily heat stress results in acclimation decay over roughly twice the time period (2–3 weeks). Importantly, less structured heat acclimation paradigms, such as simple sporting activities conducted in warm environments, also result in heat acclimation (Sunderland et al., 2008). Many factors act as modifiers to classical responses (Sawka et al., 1996), but the primary stimulus for heat acclimation appears to be heat strain sufficient to produce sweating and more than 50% skin wetness (Gonzalez & Gagge, 1996).

Altitude

Acute exposure to moderate altitude results in reflex increases in ventilation and heart rate, followed by diuresis up to a loss of ~2% body mass (~1.5 l) (Hoyt & Arnold, 1996; Ward et al., 2000). Diuresis is a urinary buffering response to hypocapnia and a favorable hallmark adaptation to altitude which, when combined with hyperventilation and tachycardia, improves S_aO_2 via improved oxygen delivery to tissues and a relative increase in blood hemoglobin concentration (Hoyt & Arnold, 1996). No attempt should be made to correct this initial body water deficit, but accurate tracking and replacing of daily

Table 35.1 Nutritional considerations associated with heat, cold, or moderate altitude exposure

Environment	Nutrient	Effect	Recommendation
Heat (≤30°C)	*Fluid*	Greater fluid losses through sweating in warm environments.	Environmental heat stress and heat acclimation both increase sweat losses and fluid needs. Weigh yourself nude or semi-nude before and after workouts to estimate unreplaced water losses (1 kg = 1000 ml).
	Electrolytes (Na^+, K^+)	Increased needs due to increased sweat losses.	Additional sodium and potassium are lost in sweat. Sports drinks can help meet electrolyte needs in addition to ordinary food intake. Salting of food and/or salty food choices/snacks are advised, particularly in the early days of novel heat exposure.
Cold (≥–5°C)	*Fluid*	Body water losses can be high even during exercise in cold weather, particularly when too much clothing (sweating) or too little clothing (cold-induced urination) is worn.	Weigh yourself nude or semi-nude before and after workouts to estimate water losses (1 kg = 1000 ml).
	Carbohydrates	Cold muscle metabolism favors carbohydrate for fuel; shivering is fueled primarily by carbohydrate.	Every effort should be made to choose carbohydrate-rich foods before, during, and after workouts.
Moderate Altitude (1500–3000 m)	*Appetite*	Rapid ascent to altitudes >2000 m may cause symptoms of acute mountain sickness (AMS), one of which is reduced appetite due to nausea.	AMS is self-limiting and temporary (2–4 days). Try to maintain training, eating, and sleeping habits as best possible. Consume foods and fluids that are best tolerated.
	Fluid	Altitude-induced diuresis increases urinary water losses.	Expect a small decrease in body water/mass (1–1.5 kg) in the first few days. Do not attempt to correct by drinking "extra." Determine fluid needs from acute body mass changes incurred during workouts. Weigh yourself nude or semi-nude before and after workouts to estimate water losses.
	Carbohydrates	Increased carbohydrate utilization at altitude; potential accelerated glycogen depletion.	Every effort should be made to choose carbohydrate-rich foods before, during, and after workouts.
	Iron	Increased iron needs. Erythropoetic response at altitude, initial decrease in serum ferritin and eventual increase in hemoglobin and hematocrit.	Ensure adequate iron status prior to altitude exposure by consuming foods high in heme iron. Based on a blood test (4–6 weeks pre-altitude), an iron supplement, taken with Vitamin C, may be advised before and/or during altitude exposure.

Although exposure to air pollution increases oxidative stress, strong evidence that dietary supplementation with antioxidants can modulate the response is lacking (Romieu et al., 2008). See also Chapters 7, 14, 16, 19, and 21.

body water losses remains essential (Table 35.1). Additional structural and hematological benefits arise with more prolonged altitude exposures, some of which may require an iron supplement in sea-level residents who ascend to altitude in an iron-depleted state (Levine & Stray-Gundersen, 1997) (Table 35.1; see Chapter 19). The time course for decay of altitude acclimation is not well studied, but seems to be similar to that observed for heat acclimation (2–3 weeks) (Ward et al., 2000).

In addition to phenotypic adaptations which preserve homeostasis in hot and high-altitude

Table 35.2 Common environmental illnesses

Term	Definition
Heat Exhaustion	Mild-to-moderate illness characterized by the inability to sustain cardiac output; seen in conjunction with exercise-heat stress with elevated skin temperatures, with or without hyperthermia (>38.5°C) of the body core; symptoms include anxiety, dizziness, fainting.
Exertional Heat Stroke	Form of body core hyperthermia, typically but not always >40°C, associated with a systemic inflammatory response, leading to a syndrome of multiorgan dysfunction in which brain swelling predominates; symptoms include CNS abnormalities, delirium, convulsions.
Hypothermia	A reduction in body core temperature below 35°C; occurs when heat dissipation exceeds heat production; symptoms include overt shivering, amnesia, and difficulty speaking.
Acute Mountain Sickness	Benign, self-limiting condition most typically associated with rapid ascent to altitudes >2000 m; symptoms include headache, nausea, and fatigue.

More serious illnesses and injuries (e.g., freezing cold injuries; HAPE, high altitude pulmonary edema) are unlikely within the typical environmental limits that athletes train and compete (defined in the Introduction of this chapter). The definitions provided are commonly used.

environments, molecular adaptations that confer cellular and tissue protection against environmental illness and injury (acquired tolerance) may provide cross-tolerance to novel stressors (Horowitz, 2007). Table 35.2 lists the most common heat, cold, and altitude-related illnesses. The important implications of heat and altitude acclimation for exercise training and performance are also notable.

Exercise Performance

Heat

High skin temperatures and large sweat losses can challenge cardiovascular and thermoregulatory systems and impair performance (Cheuvront et al., 2010). During exercise, core temperature initially increases rapidly as a consequence of metabolic heat produced by skeletal muscle contraction. Thermoregulatory responses for heat dissipation respond more slowly. Eventually, these mechanisms increase heat loss sufficiently to balance metabolic heat production at a newly elevated steady-state body core temperature. The magnitude of body core temperature increase at steady-state is largely independent of climatic conditions and proportional to the metabolic rate within a "prescriptive zone" (Sawka et al., 1996). As metabolic rate increases, the prescriptive zone narrows. In addition, skin temperature increases primarily as a function of air temperature and thermoregulatory sweating increases proportionally with exercise intensity and the total thermal load (endogenous and exogenous).

The consequences of environmental heat strain have important implications for training. For example, Lafrenz et al. (2008) demonstrated that the drift in heart rate that occurs during constant intensity exercise in the heat (35°C), does not affect cardiac output or absolute oxygen uptake, but does result in a higher relative exercise intensity than in a temperate environment (22°C) because of a reduced VO_2 max. The impact on exercise prescription is therefore similar to that described below for altitude training, whereby daily training volume and/or training intensity must be reduced in the heat. Environmental heat stress also increases sweat losses and sustainable sweating rates >1.5 l/hour are possible under many circumstances, thus doubling or even tripling daily water needs (to 6–9 l/day) above ordinary recommendations (Institute of Medicine, 2005). Sweat electrolyte losses, particularly sodium, can also be significant when sweating rates are high (1–2 g/hour). However, while daily water and sodium losses can be substantial during exercise in the heat, they are perhaps only twice those observed during the same exercise in more temperate environments. Water and electrolyte recovery can therefore be achieved by following ordinary sports nutrition guidance that includes attention to fluid and food intake (Table 35.1; see Chapters 14 and 16).

The negative impact of environmental heat stress on prolonged exercise performance is well documented. Consequences include a shorter exercise time to exhaustion, an obligatory reduction in exercise intensity, or both. Empirical laboratory and field data support this proposition. For example,

Galloway and Maughan (1997) systematically compared the effects of graded heat stress on prolonged cycle ergometer exercise performance. Using an ambient temperature range of 4–31°C, they demonstrated that time to exhaustion at a fixed intensity occurred nearly 42 minutes sooner (44%) in the warmest environment, relative to the study optimum (11°C) (Figure 35.3). Although relating observations of competitive outdoor performances to environmental conditions is an imperfect science, McCann and Adams (1997) concluded that running events requiring high aerobic fitness and large E_{req} (3000–10,000 m) slow appreciably as the environment warms above a WBGT of 16–19°C. For competitive marathon running, Ely et al. (2007) found (Figure 35.4) that performances slowed by ~2% (2–3 minutes) in elite competitors as WBGT increased from 10 to 25°C. Although the size of the heat-related performance impairment varies greatly among studies, it is commonly two times larger than the typical variability in the measurement itself (Cheuvront et al., 2010), which illustrates the strong negative effect of environmental heat stress on aerobic exercise performance. In team sports such as soccer, which require prolonged running and environmental exposures, climatic heat stress impairs performance similarly (Ozgunen et al., 2010).

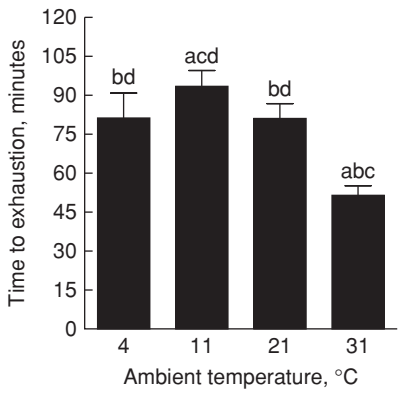

Figure 35.3 Cycling time to exhaustion under four different ambient temperatures (data are mean ± standard error). Trials 4°C (a), 11°C (b), 21°C (c), and 31°C (d) are significantly different (p <0.05) from trials (letters) placed above error bars. Adapted from Galloway and Maughan (1997) with permission.

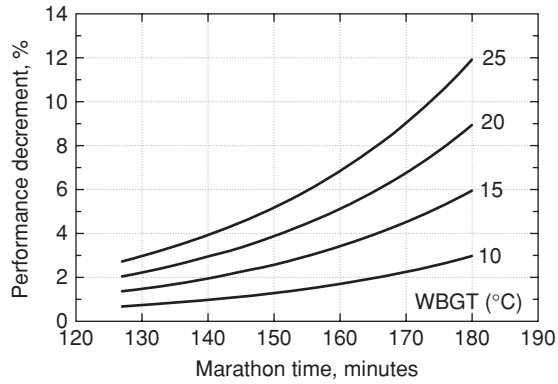

Figure 35.4 Nomogram examining the potential performance decrement (Y-axis) based on projected marathon finishing time (X-axis) with increasing WBGT. From Ely et al. (2007).

The principal physiological mechanism(s) which explain why performance is impaired by environmental heat stress are a matter of debate (Cheuvront et al., 2010). Exercise-heat stress may impair aerobic performance via one or more mechanisms related to central nervous system (critical body core temperature), skeletal muscle (metabolism), or cardiovascular system (blood flow and oxygen kinetics) function. Competitive circumstances (exercise intensity and duration) and the ratio of heat gain to loss (E_{req}/E_{max}) are among the most critical factors underpinning any physiological explanation. Many studies have shown that heat acclimation improves time to exhaustion tests in the heat (Sawka et al., 1996). Similar benefits have been observed after heat acclimation for simulated team sports activities (Sunderland et al., 2008). Lorenzo et al. (2010) demonstrated that heat acclimation improves endurance time trial performance in both hot and cool environments, analogous to the concept of improving sea-level performance through altitude acclimation. Under the conditions tested, Lorenzo et al. (2010) concluded that improvements in cardiovascular function and oxygen uptake were responsible. However, it is also important to point out that in genuine competitive warm weather endurance events, experienced runners choose a slower, constant pace from the outset, while slower runners start at a faster pace only to decelerate (Ely et al., 2008). Numerous

self-paced exercise studies comparing performance in hot versus temperate or cool environments confirm the latter finding, which supports the view that physiological cues provide sensory feedback to limit performance. However, the former observation is also consistent with learned-behavior-modifying racing strategy as a function of the environment. Performance is clearly multifaceted. Whatever the mechanism(s), heat stress forces athletes to modify their competition strategy from what they would normally do in cooler environments. Heat stress unquestionably degrades performance while acclimation improves it.

Cold

The primary challenge of exposure to a cold environment is maintenance of body temperature. During exercise in cold environments, adequate clothing and endogenous heat production are most often sufficient to overcome the temperature gradient favoring heat loss. Athletic events rarely occur in very cold environments (Castellani et al., 2006), but training necessarily occurs year-round in cold environments, and athletes are faced with the challenge of not only a cold environment, but ice, snow, wind, and reduced daylight, which may make outdoor training more difficult.

The primary consideration for training and performing in cold environments is appropriate clothing that favors heat retention and water vapor exchange. Figure 35.2 outlines the relationship between work rate and clo with environmental temperature. Exercise intensity is a key variable, as a high endogenous heat load will induce thermoregulatory sweating and cause an apparent environment-appropriate ensemble at rest to constitute overdressing during exercise. As sweat accumulation decreases the insulative properties of clothing, overdressing can result in discomfort during exercise and an increased risk of hypothermia after exercise, if wet clothing is not promptly removed (Castellani et al., 2006).

Cold disorders can be categorized by either localized nonfreezing (e.g., muscle cramps, trench foot) and freezing cold injuries (frostnip, frostbite) or whole body hypothermia (Castellani et al., 2006). Due to the increased heat production during physical activity, and because athletes are rarely exposed to the cold for an extended time, the risk of localized freezing/nonfreezing injuries is minimal during exercise. Hypothermia remains possible during whole-body cold water immersion or rainy and windy conditions due to the 70-fold greater heat transfer coefficient of water as compared to air (Castellani et al., 2006; Gonzalez & Gagge, 1996). Hypothermia is also a potential complication following exercise, or even during exercise, when an athlete slows for any reason (Maughan et al., 1985). A variety of factors are known to alter cold tolerance independent of clothing, including body size, shape, and composition (subcutaneous fat deposits), with smaller and leaner individuals requiring relatively greater heat production to maintain thermal equilibrium due to their larger surface area to mass ratios and decreased insulation (Young, 1996). The advantages of increased subcutaneous fat are apparent for all types of cold exposure, but are particularly important in events involving cold water immersion, such as open-water swimming.

Aerobic exercise performance typically improves as air temperature declines from hot to cool (Galloway & Maughan, 1997), and while this trend appears to shift in colder temperatures (4°C), the decline in performance is smaller (Figure 35.3) when compared with the challenge of exercise in the heat. Although aerobic exercise is the focus of this chapter, it is worth noting for many sports that require maximal strength, power, and explosiveness can be compromised by cold muscle temperatures. Bergh and Ekblom (1979) found that dynamic strength, power output, jumping, and sprinting performance declined by 4–6% for each centigrade decrease in muscle temperature from 39 to 30°C. A precompetition warmup to increase local heat production may offset this decline (Dixon et al., 2010). While muscle blood flow is reduced so that large muscles function as additional insulation during rest, the high blood flow requirements to those muscles during exercise may increase heat loss (Castellani et al., 1999), so clothing to cover working muscles will favor additional heat retention and maintenance of muscle temperature. Provided adequate muscle activity, heat production, and retention, strength and power performance can be sustained in the cold.

If body temperature falls before, during, or following exercise in the cold, carbohydrate oxidation is increased both for resting metabolism and through initiation of the shivering response (Young, 1990). In concert, cold exposure may lead to accelerated glycogen depletion if carbohydrate intake is inadequate. A diet high in carbohydrate (Table 35.1), as well as appropriate clothing during exercise and avoidance of prolonged post-exercise exposure where shivering is likely to occur, can help prevent glycogen depletion. Hydration is another nutritional concern of cold exposure (Table 35.1). The combination of heavy clothing and heavy work can produce sweating rates comparable with less clothing in a warmer environment, cold-induced diuresis occurs when blood is redistributed to the central circulation due to peripheral vasoconstriction, and respiratory water losses can increase due to humidification of the very dry cold air that is inhaled (Castellani et al., 2006). Adequate fluid intake is important for optimum health and performance, although the consequences of dehydration on physical performance are much smaller in cold than in temperate or hot environments (Cheuvront et al., 2010).

Altitude

Individuals experience greater physiological stress when exercising at moderate altitudes than with similar training at sea level. This is due primarily to altitude-induced decrements in S_aO_2 and VO_2 max. These physiological limitations may force individuals to reduce their daily training volume and/or training intensity, and modify their competition strategy from what they would normally do at sea level.

At rest, the partial pressure of oxygen in the blood (P_aO_2) and S_aO_2 are slightly reduced at moderate altitude despite an increase in pulmonary ventilation. During submaximal and maximal exercise at moderate altitude, however, P_aO_2 and S_aO_2 are markedly lower compared to similar exercise at sea level (Ward et al., 2000). This decrement in exercise S_aO_2 may be due to an altitude-induced reduction in the partial pressure of alveolar oxygen (P_AO_2) resulting in a decrease in pulmonary capillary diffusion time (Dempsey, 1986). Interestingly, this exercise-induced decrement in S_aO_2 appears to be more pronounced in well-trained athletes than in untrained individuals at both sea level and altitude. Lawler et al. (Lawler et al., 1988) evaluated untrained and trained males who performed incremental cycle ergometer exercise at sea level and at a simulated altitude of 3000 m. The trained group experienced a significantly greater decrement in S_aO_2 (via pulse oximetry [S_pO_2]) compared with the untrained group at both sea level (trained = 90.1%, untrained = 95.5%) and simulated altitude (trained = 77.3%, untrained = 86.3%). It has been suggested that the reduced pulmonary capillary transit time experienced at altitude, in combination with the relatively high cardiac output, pulmonary blood flow, and total hemoglobin mass of endurance athletes serves to widen the P_AO_2–P_aO_2 diffusion gradient, which results in a greater reduction in S_aO_2 in trained versus untrained individuals (Dempsey, 1986). Similar trends for reduced S_aO_2 have been observed in well-trained endurance athletes upon acute exposure to simulated elevations as low as 580 m (Gore et al., 1996).

It is well documented that VO_2 max is reduced upon exposure to moderate altitude. In a classic study, Squires and Buskirk (1982) evaluated the effect of acute exposure to hypobaric hypoxia on VO_2 max in male recreational runners (VO_2 max = 60.1 ml/kg/min). Each runner performed a maximal treadmill test in a hypobaric chamber at simulated altitudes of 363 m, 914 m, 1219 m, 1524 m, and 2286 m. VO_2 max at 362 m averaged 4.35 l/min and was significantly reduced by 5%, 7%, and 12% at 1219, 1524, and 2286, respectively (Squires & Buskirk, 1982). Several additional studies involving athletes and physically fit soldiers have demonstrated that VO_2 max declines in a curvilinear manner as altitude increases from 580 to 8848 m. Those data have been summarized in a review article by Fulco et al. (1998). As with S_aO_2, significant decrements in maximal oxygen consumption have been observed at altitudes as low as 580 m in well-trained male cyclists whose VO_2 max dropped 7% at 580 m (5.10 l/min) compared with sea level (5.48 l/min) (Gore et al., 1996).

A mathematical model has been proposed that describes the potential effect of acute altitude exposure on VO_2 max (Peronnet et al., 1991). An

individual's VO_2 max at altitude, expressed as a percentage of sea level VO_2 max (% SL VO_2 max) at a given P_B, expressed in Torr, can be approximated using the following quadratic equation:

$$\% \text{ SL } VO_{2max} = a_0 + a_1P_B + a_2(P_B^2) + a_3(P_B^3)$$

where $a_0 = -174.1448622$, $a_1 = 1.0899959$, $a_2 = -1.5119 \times 10^{-3}$, and $a_3 = 0.72674 \times 10^{-6}$. This equation was developed from data obtained between 0 m and 4000 m (760–462 Torr). For example, at an altitude of 2000 m (P_B ~600 Torr), an athlete's VO_2 max would be ~93% of sea level VO_2 max. At an altitude of 3000 m (P_B ~525 Torr), VO_2 max would be ~86% of the sea level value, whereas at 4000 m (P_B ~462 Torr), VO_2 max would be ~78% of sea level maximal oxygen uptake. This equation does not, however, take into consideration the relatively larger decrement in VO_2 max observed for more highly fit subjects (Fulco et al., 1998).

In addition to altitude-induced decrements in S_aO_2 and VO_2 max, there is also a decrease in training capacity upon exposure to altitude. Although some athletes attempt to replicate the absolute training load of their sea-level workouts, they do so at the risk of becoming ill, injured, or overtrained. Levine and Stray-Gundersen (1997) quantified the effect of altitude exposure on training intensity in competitive distance runners who were divided into three groups: (1) a "low–low" group that lived and trained at sea level (150 m); (2) a "high–low" group that lived at moderate altitude (2500 m) and trained at low altitude (1250 m); and (3) a "high–high" group that lived and trained at moderate altitude (2500 m). Table 35.3 shows the difference in training intensity between the three groups during base/overdistance training as well as interval training (1000-m intervals). Collectively, these data suggested that absolute training intensity during base and interval workouts is significantly reduced at low and moderate altitude in well-trained competitive distance runners. Due to the altitude-induced reduction in exercise intensity, the "live high + train low" (LH + TL) altitude training model was developed by Levine and Stray-Gundersen (1997). The reader is referred to the review article by Wilber (2007) for detail on LH + TL altitude training.

Empirical data as well as mathematical models suggest that aerobic performance is negatively affected upon exposure to altitude. Several scientific studies have examined the effect of altitude on either well-trained or elite endurance athletes who were evaluated on aerobic performance within the initial days of exposure to actual or simulated elevations ranging from 580 to 5200 m (Wilber, 2004). Performance measures in these studies included sport-specific time trials, work capacity tests conducted on sport-specific ergometers, and endurance time during an exhaustive incremental exercise test. All of these investigations reported decrements in aerobic performance upon acute exposure to altitude. Performance decrements ranged from 2% to

Table 35.3 Effect of altitude on absolute training intensity in competitive distance runners during base training and interval training (1000-m) workouts.

	Base Training		Interval Training	
	Running velocity (% SL 5000-m time)	VO_2 (% SL VO_2 max)	Running velocity (% SL 5000-m time)	VO_2 (% SL VO_2 max)
Low–Low ($n = 13$)	82	72	111	92
High–Low ($n = 13$)	77[a]	67	104[a]	86
High–High ($n = 13$)	76[a]	64[a]	96[a]	74[a]

Source: Adapted from Levine and Stray-Gundersen (1997) with permission.

[a]Significantly different vs. Low–Low ($p < 0.05$); Low–Low, lived and trained at sea level (150 m); High–Low, lived at moderate altitude (2500 m), and trained at low altitude (1250 m); High–High, lived and trained at moderate altitude (2500 m); SL, sea level; VO_2 max, maximal oxygen consumption.

34% depending on the altitude. Decrements in aerobic performance at altitude have been associated with altitude-induced reductions in VO_2 max secondary to reductions in S_aO_2.

A mathematical model has been proposed that quantifies the potential effect of altitude on running performance (Peronnet et al., 1991). It suggests that in running events exceeding 400 m, there is an exponential deterioration in performance due to the altitude-induced reduction in VO_2 max. For example, in Mexico City (2300 m), the decrement in average running velocity is ~2% for 800 m, 4% for 1500 m, 6% for 5000 m, and 7% for the marathon, for both men and women. Figure 35.5 shows the effect of acute altitude exposure on running velocity over various distances. Performance results from the 1968 Mexico City Summer Olympics tend to support this model. In men's track and field, world records were established in the long jump and nearly all of the sprint events. In contrast, the performances of gold medallists in longer distance events (≥1500 m) were substantially slower than the 1968 world records.

In sports where aerodynamics plays an important role, such as cycling and speedskating, performance may be enhanced when competition is conducted at altitude. It has been suggested that the optimum altitude for cycling time trials of 2–40 km length is 3200–3500 m, with times estimated to be 4–4.5% faster than sea-level performances (Olds, 2001). A model proposed by Capelli and di Prampero (1995) suggests that the optimum elevation for breaking the world record for 1-hour unaccompanied cycling is ~4000 m. At these elevations, there is a significant reduction in VO_2 max, but there is a greater relative reduction in aerodynamic resistance due to the decreased air density (Figure 35.6). The net effect is that the decrement in VO_2 max is exceeded by the enhancement in cycling aerodynamics, which results in greater cycling velocity at altitude versus sea level. Circumstantial evidence supports the same supposition for speedskating. For example, out of the 10 long-track speedskating events (500–10,000 m) at the 2002 Winter Olympics held at an elevation of 1425 m, eight world records were established. According to Capelli and di Prampero (1995), diminishing returns on performance will likely occur beyond 4000 m elevation, at which point the decrement in VO_2 max may exceed aerodynamic gains and thus lead to a reduction in performance.

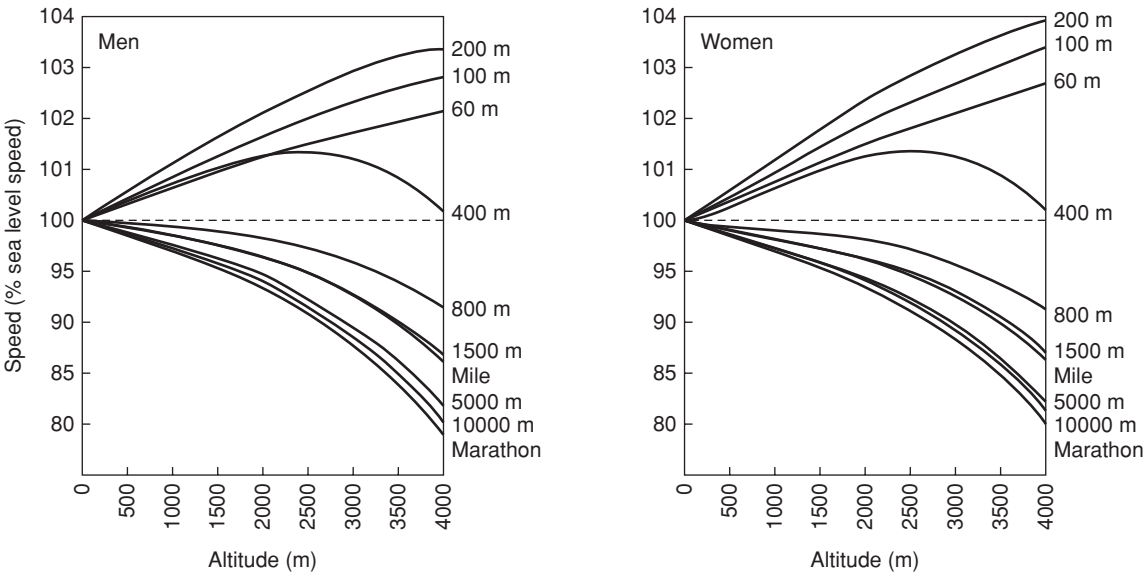

Figure 35.5 The effect of altitude (up to 4000 m) on running velocity in events ranging from 100 m to the marathon in men (left panel) and women (right panel). From Peronnet et al. (1991) with permission.

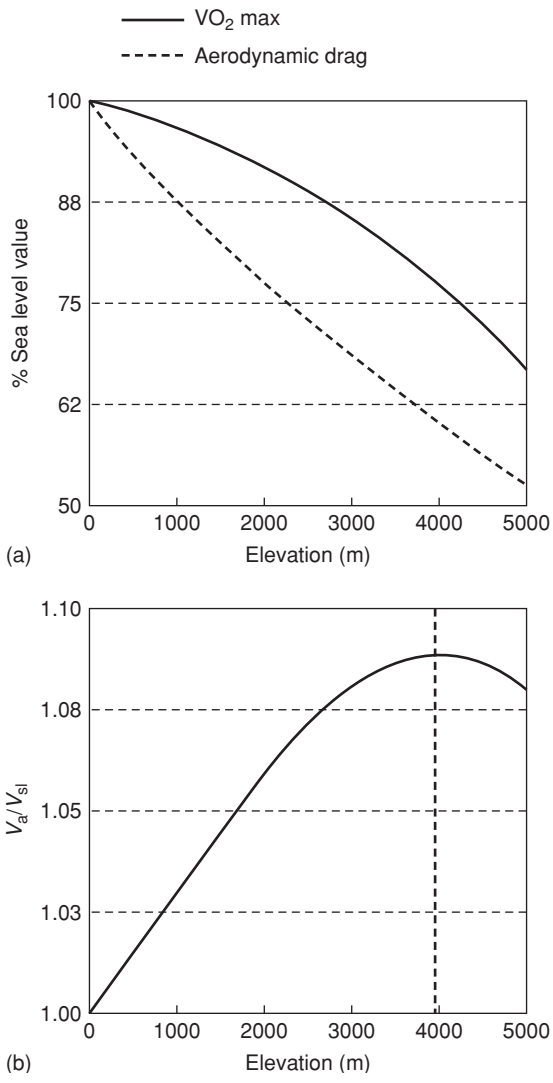

Figure 35.6 The effect of altitude on cycling performance in the 1-hour time trial. (a) Although VO_2 max (solid line) decreases as one ascends from sea level to 5000 m, the amount of aerodynamic drag (broken line) is reduced to a greater degree, allowing the cyclist to ride faster at altitude despite the drop in VO_2 max. (b) The "VO_2 max decrement versus aerodynamic benefit" is optimized at an elevation of ~4000 m, after which point the decrement in VO_2 max exceeds the beneficial effects of reduced aerodynamic drag. V_a/V_{sl} = velocity at a specific altitude relative to velocity at sea level. From Capelli and di Prampero (1995) with permission.

Adverse Air Quality and Exercise Performance

Air pollutants, either individually or synergistically, can have a negative effect on exercise performance. Air pollution sources, standards, and physiological effects are summarized in Table 35.4.

Carbon monoxide (CO) can impair exercise performance and is potentially lethal via: (1) its action as an allosteric inhibitor of oxygen; (2) a leftward shift in the hemoglobin–oxygen dissociation curve resulting from increased levels of carboxyhemoglobin (COHb); (3) inhibition of the mitochondrial enzyme, cytochrome-c oxidase; and (4) the impairment of cognitive function. With increased levels of COHb, maximal cardiac output (Q) and maximal arteriovenous O_2 difference (a-vO_2) are reduced resulting in a compensatory increase in heart rate. These reductions in Q and a-vO_2 result in a lower VO_2 max, as well as impaired work output and exercise performance (Lippi et al., 2008).

Nitrogen dioxide (NO_2) is a potent trigger of airway hyperreactivity (AHR) via generation of free radicals leading to inflammation of the epithelial cells. NO_2-induced pulmonary limitations appear to be more likely in individuals with chronic asthma and other pulmonary conditions. It is unclear whether NO_2 levels typically seen in industrialized cities will impair exercise performance in healthy, nonasthmatic individuals (Florida-James et al., 2004). However, it is possible that average urban levels of NO_2 might have a noticeable negative impact during exercise performed by well-trained athletes, whose ventilation rate (V_E) can exceed 200 l/min, thereby significantly increasing the effective dose (ED) (Florida-James et al., 2004).

Like NO_2, sulfur dioxide (SO_2) is a potent trigger of AHR via release of histamine from mast cells, leading to airway smooth muscle constriction and airflow obstruction. The combination of SO_2 and cold-dry winter air is more likely to cause pulmonary problems than inhaling SO_2 alone. Sulfur dioxide is 10 times more potent in individuals with asthma than in nonasthmatics (Florida-James et al., 2004). During submaximal exercise, some individuals experience significant

Table 35.4 Primary air pollutants and their potential negative effect on health and exercise performance

Pollutant	NAAQS	Sources	General Health	Exercise Performance
Carbon monoxide (CO)	9 ppm 8-h 35 ppm 1-h	Vehicle exhaust Cigarette smoke Open fire	Prevents O_2 from binding to hemoglobin Inhibits cytochrome-*c* oxidase Impairs cognitive function	↓ Submaximal and maximal O_2 uptake ↓ Submaximal and maximal endurance performance
Nitrogen dioxide (NO_2)	53 ppb annual 100 ppb 1-h	Vehicle exhaust Coal-burning electrical plants Coal-burning industrial sites	Potent trigger of AHR	Unknown, but may have some limiting effects when exercise V_E is high
Sulfur dioxide (SO_2)	0.03 ppm annual 0.14 ppm 24-h 75 ppb 1-h	Coal-burning power plants Petrochemical refineries Cement plants Paper mills	Potent trigger of AHR Eye irritant	Unknown, but may have some limiting effects when exercise V_E is high
Ozone (O_3)	0.075 ppm 8-h 0.012 ppm 1-h	Photochemical reaction involving bright sunlight + CO + NO_2 + O_2	Potent trigger of AHR Headache	↓ Submaximal and maximal O_2 uptake ↓ Submaximal and maximal endurance performance
Particulate matter (PM_{10})	150 ug/m^3 24-h	Construction sites Unpaved roads Industrial smoke Forest fire smoke	Potent trigger of AHR Bronchitis	Unknown, but may have some limiting effects when exercise V_E is high

AHR = airway hyper-reactivity; NAAQS = National Ambient Air Quality Standard (US Environmental Protection Agency); O_2 = oxygen; ppb = parts per billion; ppm = parts per million; ug/m^3 = micrograms per cubic meter; VE = ventilation rate; ↓ = decrease (worse performance)

decrements in pulmonary function upon exposure to low levels of SO_2, but the effect of SO_2 on endurance performance in well-trained athletes exercising at relatively high or maximal V_E, while exposed to SO_2 levels typically seen in modern industrialized cities is unclear (Florida-James et al., 2004).

Ground-level ozone (O_3) is a highly reactive oxidant produced by a photochemical reaction involving sunlight in combination with CO plus NO_2 plus O_2. The negative health effects of O_3 are similar to those of NO_2 and SO_2 in that ozone triggers AHR via production of free radicals in the epithelial membrane. Several studies have evaluated the effect of O_3 exposure on exercise performance (Carlisle & Sharp, 2001) and have consistently shown that pulmonary function was reduced and endurance performance was significantly impaired in well-trained runners and cyclists.

Particulate matter (PM) in the range of 5–10 μm can settle in the nasopharyngeal region and cause AHR and congestion. Smaller PM in the range of 3–5 μm can settle in the trachea and bronchioles leading to AHR and possibly bronchitis. Microscopic PM less than 3 μm may lodge in the alveoli, causing severe congestion and bronchitis. Individuals with asthma, chronic obstructive pulmonary disease (COPD), and cardiovascular disease are at greater risk of health complications when exposed to PM than healthy individuals (Florida-James et al., 2004). To date, the effects of PM on exercise performance have not been determined

Table 35.5 General recommendations for training and competition in challenging environments (heat, cold, altitude, air pollution)

Heat	Cold	Altitude	Air Pollution
• Allow 1–2 weeks to adapt to heat and adjust training volume or intensity as necessary, particularly in the first week. Acclimate in the warmest time of day (midday). • Alternate acclimation days with training days. Schedule training for early morning or late evening to avoid the warmest times of day and maximize exercise intensity and duration to avoid detraining. • Adjust performance goals for endurance competitions in hot environments.	• Schedule training for the warmest part of the day (midday). • Consider alternate indoor training if severe weather (snow storm, ice, high winds) is forecast. • When dressing to train or compete, consider both environment and exercise intensity. Dress in layers.	• Allow 1–2 weeks to adjust to altitude and modify training volume and/or intensity as necessary. If unable to spend 1–2 weeks acclimating, arrive as far in advance as possible, even if only for 2–3 days. • For altitude training camps (2–4 weeks), consider a "live-high, train-low" strategy for the purpose of optimizing training responses and avoiding detraining. • Adjust performance goals for competitions at altitude in long duration aerobic events that are not influenced by aerodynamics (e.g., track cycling).	• Research "real time" pollution levels via internet sites to help plan training and competition schedules. • Schedule training sessions to avoid high pollution areas (busy highways, industrial zones, construction sites) and times of day (midday, rush hour). • Consider shifting to alternate indoor training on days when air pollution is predicted to approach or surpass the "unhealthy" range.

by laboratory-based research, although a field-based retrospective analysis showed that "acceptable" levels (i.e., not "unhealthy" levels) of PM were negatively correlated to marathon performance in female recreational runners (Marr & Ely, 2010).

The concept of ED (Adams et al., 1981) can be defined mathematically as follows:

$$ED = PC \times V_E \times ET,$$

where PC is the pollutant concentration ($\mu g/mm^3$), V_E is ventilation rate (l/minute), and ET is exposure time (minutes).

Resting ED = (150 $\mu g/mm^3$ O_3) × (6 l/minute) × (480 minutes) = 4.32×10^6 μg / mm^3 O_3 / l O_2

Exercise ED = (150 $\mu g/$ mm^3 O_3) × (100 l/minute) × (30 minutes) = 4.50×10^6 μg / mm^3 O_3 / l O_2

From this example we can appreciate the profound effect of exercise and corresponding increase in V_E on significantly increasing the ED of air pollution and, simultaneously, increasing the negative impact on health and performance. Although exposure to air pollution increases oxidative stress, strong evidence that dietary supplementation with antioxidants can modulate the response is lacking (Table 35.1) (Romieu et al., 2008).

Summary

Environmental heat, cold, altitude, and air quality can significantly challenge any athlete in training or in competition. So long as sports are contested outdoors, the contents of this chapter should be given serious consideration by coaches and athletes alike. General recommendations for preparing to train and compete in these environments are provided in Table 35.5.

References

Adams, W.C., Savin, W.M., & Christo, A.E. (1981) Detection of ozone toxicity during continuous exercise via the effective dose concept. *Journal of Applied Physiology* 51, 415–422.

Bergh, U. & Ekblom, B. (1979) Influence of muscle temperature on maximal muscle strength and power output in human skeletal muscles. *Acta Physiologica Scandinavica* 107, 33–37.

Capelli, C. & di Prampero, P.E. (1995) Effects of altitude on top speeds during 1 h unaccompanied cycling. *European Journal of Applied Physiology and Occupational Physiology* 71, 469–471.

Carlisle, A.J. & Sharp, N.C. (2001) Exercise and outdoor ambient air pollution. *British Journal of Sports Medicine* 35, 214–222.

Castellani, J.W., Young, A.J., Kain, J.E., et al. (1999) Thermoregulation during cold exposure: effects of prior exercise. *Journal of Applied Physiology* 87, 247–252.

Castellani, J.W., Young, A.J., Ducharme, M.B., et al. (2006) American College of Sports Medicine position stand: prevention of cold injuries during exercise. *Medicine & Science in Sports & Exercise* 38, 2012–2029.

Cheuvront, S.N., Kenefick, R.W., Montain, S.J., et al. (2010) Mechanisms of aerobic performance impairment with heat stress and dehydration. *Journal of Applied Physiology* 109, 1989–1995.

Dempsey, J.A. (1986) J.B. Wolffe memorial lecture. Is the lung built for exercise? *Medicine & Science in Sports & Exercise* 18, 143–155.

Dixon, P.G., Kraemer, W.J., Volek, J.S., et al. (2010) The impact of cold-water immersion on power production in the vertical jump and the benefits of a dynamic exercise warm-up. *Journal of Strength and Conditioning Research* 24, 3313–3317.

Ely, M.R., Cheuvront, S.N., Roberts, W.O., et al. (2007) Impact of weather on marathon-running performance. *Medicine & Science in Sports & Exercise* 39, 487–493.

Ely, M.R., Martin, D.E., Cheuvront, S.N., et al. (2008) Effect of ambient temperature on marathon pacing is dependent on runner ability. *Medicine & Science in Sports & Exercise* 40, 1675–1680.

Florida-James, G., Donaldson, K., & Stone, V. (2004) Athens 2004: the pollution climate and athletic performance. *Journal of Sports Sciences* 22, 967–980.

Fulco, C.S., Rock, P.B., & Cymerman, A. (1998) Maximal and submaximal exercise performance at altitude. *Aviation, Space, and Environmental Medicine* 69, 793–801.

Galloway, S.D. & Maughan, R.J. (1997) Effects of ambient temperature on the capacity to perform prolonged cycle exercise in man. *Medicine & Science in Sports & Exercise* 29, 1240–1249.

Gonzalez, R.R. (1995) Biophysics of heat exchange and clothing: application to sports physiology. *Medicine, Exercise Nutrition and Health* 4, 290–305.

Gonzalez, R.R. & Gagge, P.A. (1996) Mechanisms of heat exchange: biophysics and physiology. In *Handbook of Physiology: Section 4: Environmental Physiology*, vol. 2 (eds. C.M. Blatties & M.J. Fregly), pp. 45–84. American Physiological Society.

Gore, C.J., Hahn, A.G., Scroop, G.C., et al. (1996) Increased arterial desaturation in trained cyclists during maximal exercise at 580 m altitude. *Journal of Applied Physiology* 80, 2204–2210.

Horowitz, M. (2007) Heat acclimation and cross-tolerance against novel stressors: genomic-physiological linkage. *Progress in Brain Research* 162, 373–392.

Hoyt, R.W. & Arnold, H. (1996) Environmental influences on body fluid balance during exercise: altitude. In *Body Fluid Balance* (eds. E.R. Buskirk & M.P. Susan), pp. 183–196. CRC Press Inc, Boca Raton, FL.

Institute of Medicine (2005) Water. In *Dietary Reference Intakes*, pp. 73–185. National Academies Press, Washington, DC.

Lafrenz, A.J., Wingo, J.E., Ganio, M.S., et al. (2008) Effect of ambient temperature on cardiovascular drift and maximal oxygen uptake. *Medicine & Science in Sports & Exercise* 40, 1065–1071.

Lawler, J., Powers, S.K., & Thompson, D. (1988) Linear relationship between VO_{2max} and VO_{2max} decrement during exposure to acute hypoxia. *Journal of Applied Physiology* 64, 1486–1492.

Levine, B.D. & Stray-Gundersen, J. (1997) "Living high-training low": effect of moderate-altitude acclimatization with low-altitude training on performance. *Journal of Applied Physiology* 83, 102–112.

Lippi, G., Guidi, G.C., & Maffulli, N. (2008) Air pollution and sports performance in Beijing. *International Journal of Sports* 29, 696–698.

Lorenzo, S., Halliwill, J.R., Sawka, M.N., et al. (2010) Heat acclimation improves exercise performance. *Journal of Applied Physiology* 109, 1140–1147.

Marr, L.C. & Ely, M.R. (2010) Effect of air pollution on marathon running performance. *Medicine & Science in Sports & Exercise* 42, 585–591.

Maughan, R.J., Leiper, J.B. & Thompson, J. (1985) Rectal temperature after marathon running. *British Journal of Sports Medicine* 19, 192–195.

McCann, D.J. & Adams, W.C. (1997) Wet bulb globe temperature index and performance in competitive distance runners. *Medicine & Science in Sports & Exercise* 29, 955–961.

Nielsen, M. (1938) Die Regulation der Körpertemperatur bei Muskelarbeit. *Skandinavisches Archiv Fur Physiologie* 79, 193–230.

Olds, T. (2001) Modelling human locomotion: applications to cycling. *Sports Medicine* 31, 497–509.

Ozgünen, K.T., Kurdak, S.S., Maughan, R.J., et al. (2010) Effect of hot environmental conditions on physical activity patterns and temperature response of football players. *Scandinavian Journal of Medicine and Science in Sports* 20(Suppl. 3), 140–147.

Peronnet, F., Thibault, G., & Cousineau, D.L. (1991) A theoretical analysis of the effect of altitude on running performance. *Journal of Applied Physiology* 70, 399–404.

Romieu, I., Castro-Giner, F., Kunzli, N., et al. (2008) Air pollution, oxidative stress and dietary supplementation: a review. *European Respiratory Journal* 31, 179–197.

Sawka, M.N., Wenger, C.B., & Pandolf, K.B. (1996) Thermoregulatory responses to acute exercise-heat stress and heat acclimation. In *Handbook of Physiology: Section 4: Environmental Physiology*, vol. 2 (eds. C.M. Blatties & M.J. Fregly), pp. 157–185. American Physiological Society.

Squires, R.W. & Buskirk, E.R. (1982) Aerobic capacity during acute exposure to simulated altitude, 914 to 2286 meters. *Medicine & Science In Sports & Exercise* 14, 36–40.

Sunderland, C., Morris, J.G., & Nevill, M.E. (2008) A heat acclimation protocol for team sports. *British Journal of Sports Medicine* 42, 327–333.

Ward, M.P., Milledge, J.S., & West, J.B. (2000) *High Altitude Medicine*, 3rd edn. Arnold, London.

Wilber, R.L. (2004) Physiological responses and limitations at altitude. In *Altitude Training and Athletic Performance*, pp. 22–82. Human Kinetics, Champaign, IL.

Wilber, R.L. (2007) Application of altitude/hypoxic training by elite athletes. *Medicine & Science in Sports & Exercise* 39, 1610–1624.

Young, A.J. (1990) Energy substrate utilization during exercise in extreme environments. *Exercise and Sport Sciences Reviews* 18, 65–117.

Young, A.J. (1996) Homeostatic responses to prolonged cold exposure: human cold acclimatization. In *Handbook of Physiology: Section 4: Environmental Physiology*, vol. 2 (eds. C.M. Blatties & M.J. Fregly), pp. 419–438. American Physiological Society.

Chapter 36

Food and Nutrition Considerations at Major Competitions

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Introduction

Food provision at major competition events is an important and challenging task due to the numerous cultural, religious, and sport-specific special dietary requirements and individual preferences of athletes. In addition, the food supply must be safe, nutritious, offer significant variety, be adaptable to change, and champion the local cuisine. The most significant international competition in terms of overall size and complexity is the Summer Olympic Games, which currently consists 204 nations, 11,000 competitors, 28 different sports, and 304 events over 16 days of competition. Other major competitions which also require logistical planning of food provision include, but are not limited to, the Winter Olympic Games, Paralympic Games, Commonwealth Games, Pan Am Games, FIFA World Cup, and International Amateur Athletics Federation (IAAF) world championships.

Historically, athletes have been provided with some form of food since the modern Olympic Games in 1896, where there are reports that competitors in the marathon were provided with olives, eggs, cheese, milk, and oranges before they raced (The Organising Committee for the I Olympiad, 1896). However, most meals were the responsibility of the individual athlete or team. Athletes typically used hotel dining rooms or local restaurants (Espagna, 1997), although some teams travelled with personal chefs (Lyberg, 1997; Mavrommatis, 1997). With the advent of the Olympic Village in 1932, there was expansion in catering for athletes, although food provision was basic (Table 36.1) and commonly altered at the request of individual nations (Pelly et al., 2011b).

A significant increase in complexity and volume of food provided at the Olympic Games was apparent from the 1970s, which was driven primarily by the expansion in size and number of competitors and the emergence of systematic sports nutrition research (Pelly et al., 2011b).

Currently, international catering corporations tender for food service provision at athletes' villages and competition venues. While several international catering companies have been responsible for food provision at major competition events, the ARAMARK corporation has been the primary caterer for the Summer Olympic Games of Mexico City 1968, Montreal 1972, Seoul 1988, Barcelona 1992, Atlanta 1996, Sydney 2000 (in collaboration with Spotless Services Ltd), Beijing 2008, and most recently London 2012. Provision of food at these events is highly specialized and has been implemented with varying degrees of success. Temporary facilities, casual staff, security issues, and confidentiality pose challenges for the successful catering company. Most athletes reside within an

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Table 36.1 Sample menu from the 1st August 1936 Berlin Olympic Games

Meal period	Menu items
Breakfast	Apples, bananas Porridge with milk Cornflakes, puffed wheat Grapenuts Orange marmalade, raspberry jelly Eggs prepared as desired Scrambled eggs with ham Coffee, tea, "Sanka" coffee Malt coffee, cocoa, milk Breakfast biscuits, toast
Lunch	Soup "Lison" Bouillon Veal cutlet, spinach au jus Bechamel potatoes Rice with fruit Coffee, tea
Dinner	Grapefruit Oxtail, bourguignon sauce Green peas, potatoes Salad with mayonnaise sauce Cheese Tea with lemon

Source: Adapted from the Official Report for the Organising Committee of the XI Olympiad Berlin 1936 (Organisationskomitee fur die XI. Olympiade, 1937).

athlete's village for anything from a few days up to 4 weeks. Food is provided at a number of outlets within the village, with the main dining hall playing a significant role. This temporary facility can cater for a large number of people (up to 10,000 at a time) over 24 hours of operation.

Due to the significant cost of food service at these short-term events, sponsorship by food companies is often essential. Suitable food must also be available for athletes traveling to and competing at sporting venues. Different catering companies may be responsible for the food provision at each venue, which introduces an added degree of complexity.

Nutrition support in conjunction with food provision has been a relatively recent development for major competitions. With increasing evidence for the role of sports nutrition in athletic performance (American College of Sports Medicine et al., 2000), organizing committees have supported the inclusion of nutrition expertise in catering. Nutrition support was first evident at the Los Angeles 1984 Olympic Games (Los Angeles Olympic Organising Committee, 1984). Since this time, progressive developments have included nutrition labeling of dining hall items (Barcelona 1992), a nutrition desk (Atlanta 1996), a dedicated menu website and research on dining hall apparent consumption (Sydney 2000), and appointment of the first international dietetic review committee (Beijing 2008) (Pelly et al., 2011b). The International Olympic Committee (IOC) has now mandated the inclusion of a dietitian as part of the tender process for village catering, and obtains external expert review of the village menu. However, not all competition events have progressed to the same extent as the Olympic Games. Further advances in nutrition support for all major competition events is needed to ensure suitable food is provided to competitors.

This chapter reviews the factors to consider when providing food and nutrition support at major competition events.

Food Provision for Major Competitions

Objectives of Food Provision

The primary objective when providing food at competition events is to ensure the availability of safe, appealing food that is suitable for athletes and officials of various ages and cultural backgrounds. From a sports nutrition perspective, suitable food must be provided to allow optimum performance and recovery for athletes from a wide array of sports with varying nutritional demands. This is often referred to as a "performance-based menu." Therapeutic dietary needs such as food allergies or intolerances, and any personal dietary preferences must also be considered. More recently, environmental sustainability has become an additional concern for organizing committees and caterers with a focus on the use of whole animals and reduction of packaging (London Organising Committee for the Olympic Games and Paralympic Games Ltd, 2009).

Menu Design and Service Layout

Contemporary menus are complex and contain numerous individual ingredients and recipes that must be sourced, tested, and analyzed in advance of

the event (for example, 769 individual items and 345 recipes were included in the main dining menu at the Sydney Olympic Games (Pelly et al., 2009)). Sponsorship by food and beverage companies can result in restrictions on inclusion of some items due to exclusivity rights, while access to suitable ingredients may be limited in some countries. Nonalcoholic beverages (soft drinks, water, fruit juices, and sports drinks) have been provided at the Olympic Games by the Coca-Cola company since 1984 (Pelly et al., 2011b).

To cope with the wide range of eating styles and preferences, a buffet style menu is the most common way to offer food at major events. However, there is a danger that athletes can overeat when exposed to a buffet style of eating (Smart & Bisogni, 2001). Younger athletes or those experiencing the village environment for the first time may need education about limiting their menu choices at each meal.

Most commonly, the menu is divided into:

1. All day items available over 24 hours from self-service stations (for example, breads, cereals, condiments and spreads, fruits, salad, bakery items, drinks and yoghurt, ice cream)
2. Rotational menu (usually divided into four meal periods—breakfast (5 a.m.–11 a.m.), lunch (11 p.m.–5 p.m.), dinner (5 p.m.–11 p.m.) and supper (11 p.m.–5 a.m.)

The rotational menu is traditionally offered at a number of service areas where catering staff serve athletes from a series of Bain Maries. The service areas are divided into themes based on regional cuisine as follows:

- Western/European/Mediterranean
- Pizza/pasta
- Indian/Asian
- African/Caribbean

A recent addition has been the introduction of service areas where chefs make fresh salads based on the individual's own choice of ingredients (usually a protein-based item with salad vegetables).

The challenge with menu design is to find a balance between sufficient variety to prevent boredom and cost-effectiveness. The length of the menu cycle is often dependent on the number of days the food is supplied, with a tendency to avoid following a weekly pattern. Cycles of less than one week may be appropriate for short stays (1–2 weeks), whereas stays of a month or more often require a longer cycle. The length of stay of residents depends upon the requirements of the sport, the country of origin, and the competition schedule. Some sports and nations prefer their athletes to settle into the village well in advance of commencing competition, whereas others stay only for their competition cycle. Village dining hall menu cycles for the Olympic Games have varied over the past 30 years (Table 36.2), with most recent menu cycles being 8 days duration. Shorter events such as the Delhi 2010 and Melbourne 2006 Commonwealth Games had similar cycle lengths with an 8- and 6-day cycle respectively.

There is often a focus on food that is representative of the host country. For example, at the Delhi 2010 Commonwealth Games a tandoor oven was provided with Indian chefs available to make roti and naan bread on request. At the 2004 Athens Olympic Games, Greek cuisine contributed to ~30% of menu items and was well received (Athens 2004 Organising Committee for the Olympic Games, 2005).

Food Safety

Food safety is of particular concern as an outbreak of foodborne illness may have substantial impact on an athlete's performance. While no outbreaks

Table 36.2 Length of the menu cycle at the Summer Olympic Games (1972–2012)

Olympic Games	Menu cycle
Munich 1972	10-day
Montreal 1976	5-day
Los Angeles 1984	5-day
Seoul 1988	7-day
Barcelona 1992	7-day
Atlanta 1996	7-day
Sydney 2000	10-day
Beijing 2008	8-day
London 2012	8-day

Source: Adapted from Pelly (2006).

of foodborne illness have been reported in the literature to date, gastrointestinal problems are a common complaint of athletes at major competition events. Although this may be related to other issues such as change in diet, consumption of food and fluid outside the village, and personal hygiene (see Chapter 27), caterers must follow procedures to ensure a safe food supply. This includes regular testing of both hot and cold items to ensure food is kept within critical temperature ranges to minimize microbial growth. At the Sydney 2000 Olympic Games, food samples were collected and retained in case of any microbiological outbreak (Pelly, 2006). As the size and complexity of the dining service has increased, concerns about food hygiene and safety have escalated. At the Beijing 2008 Olympic Games, the caterers incorporated an independent risk management strategy, which included an onsite Hazard Analysis and Critical Control Points (HACCP) laboratory (Wanik, 2009). Furthermore, tracking devices using GPS were implemented to monitor the distribution, transport, and delivery of food (Beijing Organising Committee for the Games of the XXIX Olympiad, 2008). Athletes are generally not permitted to take food from the dining hall due to issues with food safety, but, at some events, removal of fruit and bottled beverages has been allowed.

Contamination of food with ingredients that could lead to a positive drug test is also of concern, particularly in countries where regulation is lax. Organizers, caterers, and teams should be aware that there is a risk associated with the availability of these items within the village environment. All ingredients and food items should be carefully reviewed by experts to ensure there is no risk of inadvertent doping.

Food Provision at Venues

There has been little published information about the food supply at specific competition venues at major athletic events. Often this is catered for independently of the athletes' village and, therefore, may vary in quality. Organizers commonly use the existing venue caterers where the food provision can be focused around the needs of the spectator rather than the athlete. The availability of food usually depends on the location of the venue in relation to the main village as well as the competition schedule. Some venues may provide only limited snack items and drinks in athletes' lounges, and drinks only in the locker rooms, warm up areas and on the field of play, thus relying on the individual to supply their own food. At the Beijing 2008 Olympic Games, fruit and sports bars were reported to be available at competition venues (Beijing Organising Committee for the Games of the XXIX Olympiad, 2008).

Portable food is an option for the athlete traveling to venues. Lunch packs have been available at the Olympic Games since the 1960s (The Organising Committee of the games of the XVII Olympiad, 1960) and have acted as a pre-competition snack and portable recovery meal for athletes. In the past, these consisted of fresh food such as filled sandwiches and rolls, but concerns about food safety have resulted in a shift toward either the supply of nonperishable items or no food provision, such as at the Athens 2004 Olympic Games, where no food was allowed to be transported to venues (Athens 2004 Organising Committee for the Olympic Games, 2005). When available, packaged lunches are usually the responsibility of the organizing committee (not the principal caterers) and must be ordered 24 hours in advance. Packaged lunches at recent Olympic Games have included small bread rolls, tinned fish or meat, chocolate bars, and cookies, with a vegetarian option available on request. More recently, input from sports dietitians has provided some guidance to organizers on suitable items for recovery packs that meet the needs of the athlete. Further advances could include gluten-, nut-, and lactose-free, low- and high-energy, and vegetarian/vegan lunch packs that include rolls or crackers, tinned meat, fish or an appropriate spread, sports or muesli bars, fresh, dried and/or tinned fruit, flavored milk tetra packs, and sports drinks or water.

Consumption Patterns

There is little published data on consumption patterns at major competition events. A summary of the gross volumes of food used for village catering at the Summer Olympic Games (1896–2004) and predicted volumes for London 2012 is provided in Table 36.3. At the Sydney 2000 Olympic Games, records were kept on the uptake of each menu item

Table 36.3 Gross volumes of food used for village catering at the Summer Olympic Games (1936–2012)

	Berlin 1936[a]	Helsinki 1952[b]	Rome 1960[c]	Mexico 1968[d]	Seoul 1988[e]	Atlanta 1996[f]	Sydney 2000[g]	Athens 2004[h]	London 2012[p]
Village residents	4500	4800	6000	13,835	14,501	16,500	16,000	16,650	16,500
Nations	49	69	84	112	190	197	199	201	204
Meals served	149,611		640,232	849,447	835,344	>1,000,000	>1,160,000	>1,000,000	
Food items									
Chicken (kg)	19,729		46,039	76,780	42,561	68,040		120,000	31 tonnes
Beef (kg)	35,290	60,599[i]	61,508	118,706	42,776	120,000	110,000	143,000	>100 tonnes
Fish/shellfish (kg)	3047		15,252	31,811	30,651		50,600		>82 tonnes
Eggs (each)	232,029		318,320	403,000		576,000	153,000		19 tonnes
Fruit (kg)	37,931	33,565	241,335		180,544		105,000	140,000	330 tonnes[q]
Vegetables and potato (kg)	113,842	82,300[j]	171,181	335,777	166,237[j]			120,000	232 tonnes
Pasta/noodles/rice (kg)	8858	–	24,390	14,938		39,009	44,396		
Bread	60,827[k]	28,360	41,293				36,000		25,000 loaves
Soft drink (liters)		53,600[l]	88,993[m]			750,000[n]			
Milk (liters)	72,483	55,500	62,542	68,855		318,227	105,800[o]		75,000 liters
Water (bottles)							1,200,000	900,000	

Source: Adapted from Pelly et al. (2011b).

[a]Adapted from The Official Report of the Organising Committee of the XI Olympiad, Berlin 1936 (Organisationskomitee fur die XI. Olympiade, 1937).
[b]Adapted from The Official Report of the Organising Committee of the XV Olympiad, Helsinki 1952 (The Organising Committee for the XV Olympiad, 1955).
[c]Adapted from The Official Report of the Organising Committee of the XVII Olympiad, Rome 1960 (The Organising Committee of the Games of the XVII Olympiad, 1960).
[d]Adapted from The Official Report of the Organising Committee of the XIX Olympiad, Mexico 1968 (The Organising Committee of Games of the XIX Olympiad, 1969).
[e]Adapted from The Official Report of the Games of the XXIV Olympiad, Seoul 1988(Seoul Olympic Organising Committee, 1989).
[f]Adapted from The Official Report of the Centennial Olympiad, Atlanta 1996 (Organising Committee of the 1996 Atlanta Olympic Games, 1997).
[g]Information provided to author from SSL/ARAMARK joint venture (Pelly, 2006).
[h]Adapted from The Official Report of the XXVIII Olympiad (Athens 2004 Organising Committee for the Olympic Games, 2005).
[i]All meat.
[j]Excludes potato.
[k]Includes cake.
[l]Soft drink and fruit juice.
[m]Fanta® and Coca-Cola®.
[n]Based on 2 million cans of soft drink.
[o]37% reduced/skim milk.
[p]From Food Vision for the London 2012 Olympic Games and Paralympic Games (London Organising Committee for the Olympic Games and Paralympic Games Ltd, 2009).
[q]Fruit and vegetables.

over the course of the entire event. Over 20,000 athletes used the main dining hall daily with over 5000 choosing from this menu for breakfast, lunch, and dinner and ~900 at supper (Pelly et al., 2011b). At its peak, main dining had a daily uptake of over 36,000 meals. Breakfast, lunch, and dinner contributed around 30% and supper 5% of all meals. The average plated meal was estimated to be 20.2 MJ with a divide of 5.7 MJ (28% energy), 5.2 MJ (26% energy), 5.8 MJ (29% energy), and 3.5 MJ (17% energy) at breakfast, lunch, dinner, and supper respectively (Pelly, 2006). Fat content of the meal varied the most over the day, with the largest quantity consumed at breakfast. The average plated meal at dinner was higher in energy and varied most in macronutrient composition compared with other meal periods. While these figures do not account for plate waste and therefore are not reflective of individual consumption, they can provide a guide for future planning by caterers and sports dietitians. McDonalds Corporation™ has been a sponsor of the Olympic Games since 1996 and a feature in the athletes' village (Pelly et al., 2011b). At the Sydney 2000 Games, the uptake from McDonalds as a proportion of the total was 18% with the majority being at supper time and increasing after competition (Pelly et al., 2004).

The uptake of food steadily rises from the opening of the village to the start of the competition. However, at the Delhi 2010 Commonwealth Games where teams were initially reticent about travel to India due to safety concerns, uptake increased dramatically in the few days before competition commenced. This led to a shortage of staple food items such as bread. Caterers need to be adaptable to menu changes in such situations. This may be easier in Western countries where food items are easily sourced.

Factors Influencing the Food Intake of Athletes at Major Competitions

A number of factors can influence food intake, including sensory, cultural, and religious factors; convenience and cost; health and well-being; self-beliefs and individual preferences. For athletes, performance factors can also play a significant role. Caterers and support personnel should understand the various influences on food choice for the elite athlete.

Taste and Sensory Factors

Taste and other sensory influences (smell, sight, and texture) are key determinants of food choice, especially in males (Hess, 1997; Nestle et al., 1998). Previous research on athletes suggests no difference between preferences for high-fat animal products between male athletes and sedentary controls (Guinard et al., 1995). An alternative study found female athletes reported a lower preference for high-fat food than sedentary controls (Crystal et al., 1995). Sensory factors also have a significant influence on food choice in athletes at major competition events (Pelly et al., 2006b), with smell rated more important by higher proportion of African and Caribbean athletes (Delhi 2010 Commonwealth Games, unpublished data).

Cultural and Religious Influences

An individual's beliefs and attitudes about food are constructed from cultural values and therefore can have a strong influence on food intake (Nestle et al., 1998). In traditional societies, food choice is influenced by cost and availability of food. Eating new food is usually independent of traditional food habits (Satia et al., 2000). Fast food, confectionery, and soft drinks are easily accepted whereas foods associated with ethnic identity (e.g., rice for Chinese) are resistant to acculturation (Kittler & Sucher, 2008). Certain foods that are usually high in carbohydrate are considered staples or "core" foods of particular cultures (e.g., rice, taro, yam, wheat, potato, cassava, and plantains). Additional complementary foods are added to enhance the flavor and can assist with nutritional adequacy of the meal (Mintz & Sclettwein-Gsell, 2001). Examples are Italian noodles with tomato-based sauce, pilaf with parsley and dried fruit, nuts or corn tortillas with salsa. Secondary high-protein items (meat, legumes, and dairy) are added to core and complementary foods. Foods eaten sporadically ("peripheral" foods) are based on personal preference. Some meals and foods are unique to a particular ethnic group. For example, Koreans like to eat "kim chi" (pickled vegetables) with their meals. Some dietary regimens (e.g., vegetarian, vegan, gluten-free, preservative free) may also originate from cultural values.

Major international competition events involve the participation of athletes from a wide range of cultures, beliefs, attitudes, and needs. Since the Sydney 2000 Olympic Games, there has been an increased focus on providing food that caterer for a range of cultural needs. Anecdotal feedback from the 1996 Olympic Games in Atlanta suggested that there was a higher than anticipated demand for traditional foods from African, Asian, and Eastern European nations (Pelly, 2007; Pelly et al., 2011b). Feedback from Sydney 2000 suggested that African nations could be better catered for, while at the Delhi 2010 Commonwealth Games, athletes from the Caribbean indicated that there was a lack of culturally suitable items available on the menu (Burkhart & Pelly, 2013a). In unfamiliar environments, culturally familiar food may provide a sense of security for the athlete. In the challenging environment of the Delhi 2010 Commonwealth Games, a higher proportion of athletes rated familiar food above sensory factors or nutrient composition in influencing their food choice (Delhi 2010 Commonwealth Games, unpublished data). However, over two-thirds of all athletes reported consuming a Western style of eating regardless of their country of origin. Foods served in one locality may be known by a different name elsewhere (e.g., rockmelon vs. cantaloupe, zucchini vs. courgette) and, therefore, the naming of the food item by the caterer can influence the food selection of the athlete. There will always be some difficulty in catering for every cultural permutation, but there is now an expectation that most competing nations will have some representation on the menu to ensure equality for all competitors. A list of the various cultural eating styles is provided in Table 36.4.

Religious beliefs can also have major influence on food intake. Different cultural and ethnic groups have specific rules regarding food and these often

Table 36.4 Cultural eating styles

Region	Eating style	Main countries	Core food
Africa	Northeast African	Ethiopia, Somalia, Eritrea, Sudan	Millet, plantains
	South African		Potatoes (European influence)
	West African	Nigeria, Ghana	Starch vegetables (yams, plantains, cassava)
	East African	Kenya, Tanzania, Uganda	Cassava, corn, millet, plantains, sorghum
Asia	Indian	India, Pakistan, Sri Lanka	Rice, legumes (dhal), wheat in bread
	Southeast Asian	Philippines, Thailand, Vietnam, Indonesia, Malaysia	Rice
	Asian	China, Korea, Japan	Rice
America	North America	USA, Canada	
	Central America	Mexico, Belize, Guatemala, El Salvador, Honduras, Nicaragua, Costa Rica	Corn, rice, legumes (red and black beans)
	South America and Caribbean	Jamaica, Puerto Rico, Bahamas, Haiti, Peru, Argentina, Brazil, Chile, Bolivia	Cassava, plantains, legumes (red and black beans)
Europe	Middle Eastern	Greece, Turkey, Egypt, Iran	Wheat in leavened flat bread, pastries, loaves
	North Europe	British Isles, France	Potato, wheat bread, oats (porridge)
	South Europe	Italy, Spain, Portugal	Wheat/cornmeal use to make pasta and bread, rice
	Central Europe	Germany, Poland, Russia, former Soviet Union	Bread made from rye, cornmeal, and wheat, potato
	Scandinavian	Sweden, Denmark, Finland, Norway, Iceland	Rye bread, potato
Pacific	Australia/New Zealand	Australia, New Zealand	Wheat used in bread, cereal, potatoes, oats, pasta, rice
	Pacific Island	Polynesia, Samoa, Vanuatu, Fiji, Tonga, Hawaii	Starchy vegetables (Taro, cassava, yams)

Source: Adapted from Kittler and Sucher (2008).

Table 36.5 Religious dietary restrictions

Religion	Food avoided	Rules and comments
Buddhist	No meat, fish, shellfish	Forbidden to kill living creatures
Hindu	No meat, restricted fish and shellfish	Humoral theory—"hot" and "cold" foods
Judaism	No pork, shellfish	Kosher—only forequarters of meat; meat and dairy must be separate
Islam	No pork, alcohol	Halal— meat slaughtered according to Islamic rules
Orthodox Christianity	No meat or animal products when fasting	No olive oil on fast days, fish avoided
Roman Catholics	No meat on Fridays	Mainly observed at Lent
Seventh-Day Adventists	No meat, fish, seafood, little caffeine, no alcohol	Lacto-ovo vegetarian, no overeating, no tobacco
Mormon	No alcohol, tea, coffee	

stem from religious beliefs (Table 36.5). As an ever-increasing number of Muslim athletes compete at international events, suitable hot food choices need to be provided at each meal period and identified clearly on the menu. More recently, food stations exclusively with Halal food have been provided. As there is generally a lower demand for Kosher food, these items are often ordered from an accredited supplier and brought into the village on request.

Fasting is also practiced at various times by a number of religions, so caterers and organizers need to consider whether fasting periods fall in the competition period when planning the menu. The most significant fasting period for Muslims is Ramadan, which occurs in the ninth lunar month. This involves abstinence from all food and fluid from first light to sunset, and includes a traditional breaking of the fast after sunset ("iftar") and a meal just before dawn ("sohour"). As Ramadan coincided with the London 2012 Olympic Games, it posed significant challenges for the caterers. During Ramadan, athletes may experience increased protein breakdown and fat oxidation and alterations to hormonal and immune systems (Chaouachi et al., 2009). Athletes also report marginal change in thirst, hunger, fatigue, and concentration and slightly less sleep but no change in sleep quality (Leiper et al., 2008). However, experienced Muslim athletes appear to cope with normal training loads while fasting (Mujika et al., 2010) despite a deficit in total energy intake and fluid. Current recommendations for fasting athletes include monitoring dietary intake, hydration, recovery and fatigue, and reducing training load but not intensity during the fast if needed (Mujika et al., 2010). As most athletes will be keen to break the fast as soon as feasible, adequate high protein and carbohydrate Halal options are essential on the overnight menu, with sufficient service stations available to cope with an influx of people before sunrise and at sunset.

Sports Performance

Provision of high-protein items was the focus of the Olympic Games menu from 1932 to 1968 (Pelly et al., 2011b). The significant change toward a focus on carbohydrate-rich food was not apparent until the 1970s, which mimicked the increase in evidence for the benefit of carbohydrate on exercise performance (Pelly et al., 2011b), but the first mention of a "high starch, low fat" menu was not evident until 1996 (Organising Committee of the 1996 Atlanta Olympic Games, 1997).

While current practice at major competitions is to ensure adequate provision for all athletic needs, other factors such as cost, time, and effort may be more important to catering management (Reichler & Dalton, 1998). Chefs are also focused on the culinary and sensory factors of the recipes they produce. Yet, food and fluid intake prior to competition can

significantly influence performance (Hargreaves et al., 2004), and unplanned changes to an athlete's diet may have negative consequences. There is evidence that athletes consider the nutrient composition above sensory factors when choosing from a diverse menu, particularly prior to competition (Pelly et al., 2006b). Furthermore, a high proportion of athletes report following a diet based on the macronutrient content of the food (low fat, high protein, or high carbohydrate) (Pelly et al., 2011a). A survey of Australian athletes prior to the Sydney 2000 Olympic Games suggested a preference for low-fat, high-carbohydrate items resulting a focus on lower fat items on the menu (Pelly et al., 2009). However, this may be sport-specific and related to current popular dietary trends. More recently, athlete surveys suggest that those competing in power/sprint sports are more likely to follow a high-protein diet in comparison to weight-category athletes who follow a diet low in energy and fat (Pelly & Burkhart, 2013 [in press]).

The wide variety of items on offer may tempt the athlete to overeat or consume foods that would not normally be eaten. Although some athletes remain disciplined and closely follow their habitual diet, the unusual environment created at major competitions may influence their dietary intake. Athletes may also be influenced by other competitors' eating habits or the presence of the coach at the meal (Pelly et al., 2006b; Smart & Bisogni, 2001).

One of the more recent issues with catering for a wide range of performance needs is the ability to provide food that is suitable for athletes in weight-class sports (making weight) or those with restricted energy budgets. Caterers often focus on providing "tasty" food and, therefore, the amount of plain food cooked using simple methods such as steaming, poaching, dry baking, and broiling is limited. Inclusion of plain grilled meats and steamed vegetables would assist athletes who are concerned with the energy content of the menu item.

Age and Gender

Gender and age both influence food preferences, with males generally requiring larger serves of food due to larger body size and greater training loads. However, some differences may be related to perceptions of body image. Female athletes appear to be more particular about what they consume (e.g., low fat, fewer treats) and may be more health conscious (more fruits/vegetables, fewer snacks) than males. Adolescent girls may be more willing to try new foods (Cummings et al., 2010), eat more fruits and vegetables, and less high-fat and sugar foods, and tend to have better nutrition knowledge than adolescent boys (Nowak & Speare, 1996).

Younger athletes may have had less exposure to a variety of cuisines and less nutrition education and prefer familiar food, and therefore may be reluctant to sample different food when competing away from home. Adolescent athletes tend to be more conservative in their choices and stick to plainer food (Cummings et al., 2010). Thus, it is clear that the demographics of age and gender must be considered in menu planning for major competition events. Sufficient basic menu options without added spices or sauces should be available on the menu to accommodate younger athletes.

Special Dietary Needs

Special dietary needs may range from specific clinical conditions (e.g., celiac disease, diabetes mellitus, and cardiovascular disease) through to food allergies and intolerances (e.g., peanut and tree nut, seafood, wheat, lactose) and irritable bowel syndrome. Gastrointestinal upset due to bacterial infection or nerves can also play a role in food intake. Furthermore, personal preferences of athletes can result in very specific dietary regimens (e.g., vegetarianism, red meat avoiders, or natural food eaters). Vegetarianism may be used for improved health, environmental or humanitarian reasons, or for weight control (Chapter 31). The majority of vegetarian diets can easily be catered for on a standard menu, but vegan choices may present greater challenges in menu design. Athletes on a vegan diet must obtain protein from nonanimal food sources such as soy products, legumes, nuts, seeds, and grains. Specialized menu items must be included to provide an adequate selection for the athlete.

Identifying the number of athletes requiring a special diet is difficult, but this information is extremely useful when designing the menu. The results of a survey of 354 athletes at the Delhi 2010 Commonwealth Games found that 13% of athletes followed a dietary regimen related to a food allergy or intolerance, with most being low lactose or wheat-free, while 42% followed a diet based on personal preferences (avoiding red meat, no additives) (Pelly et al., 2011a). There appears to be an increase in requests for gluten-free items at major competition events (Wanik, 2009), but few athletes (1–2%) report following a gluten-free diet. More recently, athletes with special dietary needs have been referred to the nutrition kiosk located in the dining hall, where dietitians facilitate selection of the appropriate items from the menu.

Catering for specific food allergies is a continuing challenge that has been dealt with differently at major competitions. At the Sydney 2000 Olympic Games, high-risk allergens such as peanuts and peanut oil were removed from all recipes and not stored within the facility to reduce the risk of cross-contamination. Gluten-free thickeners were incorporated into wet dishes wherever possible to increase the choice of gluten-free items (Pelly et al., 2009). To ensure that at-risk athletes could make an informed decision, a nutrition expert was employed to identify all major allergens, which were subsequently displayed on the nutrition label above the item. However, those individuals at high risk of anaphylactic shock were provided with allergen-free food brought into the village from outside suppliers. Preparation of food in an area where contamination risk is minimal is an alternative solution. At the Delhi 2010 Games, athletes were advised not to eat outside the village due to food safety and security concerns. As allergen-free food could not be guaranteed by local suppliers, high-risk athletes relied on sports dietitians to source suitable ingredients from the ingredient stores and find a designated safe cooking zone and a suitably trained chef to cook the food. An improvement to this process would be the inclusion of a "diet" kitchen or cooking area located near to the nutrition desk with a suitably trained chef.

Nutrition Support for Athletes at Major Competitions

The degree of nutrition support for the Summer Olympic Games increased significantly after the Sydney 2000 Games as a result of the successful implementation of a number of nutrition services. These included review of the menu, development of innovative nutrition labeling, a website to house the menu, a nutrition kiosk, education of catering staff, quality assurance of menu items, and research into athletes' opinion of the menu (Pelly et al., 2009). More recently, the Winter Olympic and Commonwealth Games have introduced dietetic review of the menu, menu labeling, and nutrition kiosks. The level of nutrition support at event varies, but at a minimum level usually includes review of the menu by a dietitian and access to a nutrition expert at the event.

Menu Review and Assessment

A designated nutrition expert is required to review the proposed village menu of the Olympic Games for nutritional adequacy to ensure it meets the needs of all cultures, sports, and dietary regimes. In 2007, the IOC introduced the first international and external dietetic review of the Beijing 2008 Olympic Games menu by a group of sports nutrition experts (Professionals in nutrition and exercise science (PINES) International sports nutrition and catering working group). Subsequently, PINES has reviewed the Vancouver 2010 Winter and London 2012 Olympic Games. This quality review process is implemented after the catering tender has been awarded and the menu for the village is finalized. These steps can enhance the overall food provision for athletes at major competition events.

Nutrition Labels

Point-of-choice nutrition labeling of menu items can take many formats with varied opinions on the ideal layout and content (Almanza & Hsieh, 1995; Levy et al., 1992) and little consensus over the impact on food selection (Levy et al., 1996). The first reported use of nutrition labeling at a major

competition event occurred at the Barcelona 1992 Olympic Games (Barcelona 1992 Olympic Organising Committee, 1992). Subsequent Olympic Games and other major competition events have used nutrition labeling with varying degrees of success. Sports dietitians were involved with the content and display of the nutrition labels at the Sydney 2000 Olympic Games resulting in the most comprehensive nutrition labels to date (Figure 36.1). Athletes' feedback suggested that nutrition labels are important and used by most individuals at least some of the time (Pelly et al., 2006a, 2009), particularly by females and those in weight-category sports (Burkhart & Pelly , 2013b [in press]). African athletes appear to place more emphasis on labeling, perhaps because they are less familiar with the food on offer. Suggested improvements by athletes include more details on ingredients, ability to identify items low in fat, and easier identification of the macronutrients through color coding or pie charts.

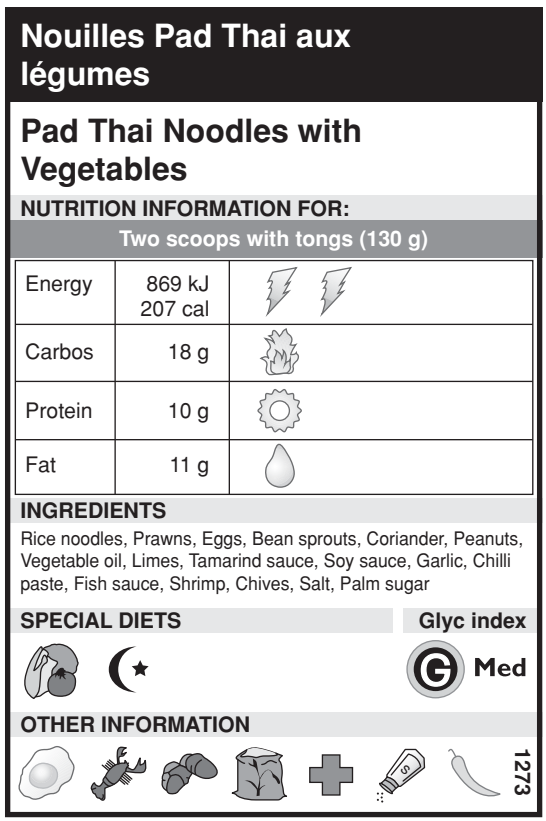

Figure 36.1 Nutrition label.

At a minimum, a nutrition label should include

- the name of the item;
- energy (kJ), protein (g), total fat, saturated fat, carbohydrate, fiber and sodium per serve and per 100 g;
- serving size in grams and household measures (e.g., 150 g = 1 large ladle) as weight alone is not useful to the consumer;
- labeling of gluten-free, vegetarian/vegan, Halal, and low energy items;
- identification of major allergens (e.g., peanuts, tree nuts, fish, seafood, eggs, soy, and dairy);
- an ingredient list.

In addition, the rating of macronutrient content and sodium as high, medium, or low can help athlete with their food choices.

A standardized nutrition label could improve nutrition support in competition environment for both the athlete and caterer. Familiarity with a standardized label format has the potential to reduce the time involved in food choice in this type of environment. Education on the format could be provided for catering staff, athletes, and other staff well in advance of the event.

Provision of a Nutrition Service

The provision of a nutrition desk within the main dining hall of the Olympic Games has been a fairly recent addition at major competition events. While nutrition experts provide education for athletes in their home country, previously there has been minimal ongoing support once in the village. The first basic nutrition information centre ("Nutrition Kiosk") was available at the 1992 Barcelona Olympic Games (2006aBarcelona 1992 Olympic Organising Committee, 1992). More extensive nutrition services were offered by sports and clinical dietitians at the Sydney 2000 Olympic Games (Figure 36.2). A nutrition desk has continued to feature at subsequent Olympic Games but with varying numbers of staff and roles.

Figure 36.2 Nutrition Kiosk at the Sydney 2000 Olympic Games.

The nutrition desk can be the central point for athletes seeking advice on the menu in relation to their particular individual needs and provides athletes and officials the opportunity to comment on the quality and range of items on the menu. The desk can be a focal point for any special dietary requests and act as the link between athletes and officials, and caterers. The function of the nutrition desk can include but is not limited to the following activities:

- Clinical and sport-specific individual consultations for athletes and officials;
- Quality assurance checks of menu items and nutrition cards (see section on Quality Assurance);
- Answer enquiries about the food provision and location or availability of specific menu items;
- Survey diners about their dining experience and provide reports to catering management;
- Manage special dietary requests in liaison with catering staff and chefs;
- Conduct group education and dining room tours for teams;
- Assist athletes with specific concerns about making weight.

There is evidence for increased use of the nutrition desk at recent events with the service rated highly by athletes (Pelly et al., 2006a, 2007, 2011a). Athletes who have less access to nutrition expertise in their home country tend to use the desk for advice on their competition eating plan, whereas athletes who have received specialized sport-specific meal plan tend to seek advice on the individual menu items (Pelly et al., 2011a).

Additional support to catering staff can be provided by nutrition experts prior to the event in the form of an education program which can improve compliance to standard recipes, highlight the

special needs of athletes, and prevent allergen contamination in kitchens.

Quality Assurance

Quality assurance (QA) is a way of maintaining quality of food over the course of the competition event. At the Sydney 2000 Olympic Games, Melbourne 2006 and Delhi 2010 Commonwealth Games, sports dietitians conducted QA with positive outcomes from the caterers.
Activities can include

- audit of nutrition labeling to ensure it is correct;
- testing of serving sizes to ensure they match nutrition labeling;
- audit on food temperature, taste, and appearance;
- audit of availability of specific items;
- survey diners, summarizing results and providing feedback to caterers;
- compile positive and negative comments received at the nutrition desk.

Future Directions

The advances in provision of appropriate food at major competition events has expanded significantly over the past decade; however, there are still a number of challenges that could be addressed at future events. While expert review of the menu is essential, the provision of suitable food and a nutrition service extends well beyond analysis of a menu on paper. Comprehensive involvement of sports nutrition experts earlier in the tender process would be beneficial. Furthermore, better communication to teams in advance of the event could assist athletes with planning a competition meal plan before entering the village. This could be implemented via the use of a dedicated website and smartphone applications. To address the increasing number of special dietary requests, there is benefit in surveying teams in advance of arrival to understand the number and type of specific menu items that may be needed. There is also a need to better cater for athletes trying to make weight or who have a restricted energy intake. A standard nutrition label format could be introduced for consistency and improved usability. Design of suitable lunch packs and supply of recovery items including formulated sports foods, should also be the included as part of the overall food provision.

Continuity in food provision at most major competitions is difficult as a result of changes in organizers, caterers, athletes, and officials between events. The development of specific guidelines for appropriate food provision and nutrition support that can be incorporated into the tender document for food service providers will ensure that athletes are provided with the ideal regimen to perform at their best.

References

Almanza, B.A. & Hsieh, H.M.Y. (1995) Consumer preferences among nutrition label formats in a restaurant. *Journal of the American Dietetic Association* 95(1), 85.

American College of Sports Medicine, American Dietetic Association, and Dietitians of Canada (2000) Joint position statement: nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 32(12), 2130–2145.

Athens 2004 Organising Committee for the Olympic Games (2005) *Official Report of the XXVIII Olympiad. Volume 1: Homecoming of the Games—Organisation and Operation*. Liberis Publications Group, Athens, Greece.

Barcelona 1992 Olympic Organising Committee (1992) *The Official Report of the Games of the XXV Olympiad. Volume III: The Organisation*. International Olympic Committee, Barcelona, Spain.

Beijing Organising Committee for the Games of the XXIX Olympiad (2008) *The Official Report of the Beijing 2008 Olympic Games*. Beijing, China.

Burkhart, S.J. & Pelly, F.E. (2013a) Athletes' opinions of food provision at the 2010 Delhi Commonwealth Games: the influence of culture and sport. *International Journal of Sport Nutrition and Exercise Metabolism* 23, 11–23.

Burkhart, S.J. & Pelly, F.E. (2013b) Athlete use and opinion of point of choice nutrition labels at a major international competition. *Appetite* (in press).

Chaouachi, A., Leiper, J.B., Souissi, N., et al. (2009) Effects of Ramadan intermittent fasting on sports performance and training: a review. *International Journal of Sports Physiology and Performance* 4(4), 419–434.

Crystal, S., Frye, C.A., & Kanarek, R.B. (1995) Taste preferences and sensory perceptions in female varsity swimmers. *Appetite* 24, 25–36.

Cummings, D.E., Pelly, F., Dang, V.H., et al. (2010) Providing meals for athletic groups. In *Clinical Sports Nutrition* (eds. L. Burke & V. Deakin). McGraw-Hill, Sydney, Australia.

Espagna, S. (1997) Gastronomy at the Olympic Games. Part 1. *Olympic Reviews* 26, 17–50.

Guinard, J.X., Seador, K., Beard, J.L., et al. (1995) Sensory acceptibility of meat and dairy products and dietary fat in male collegiate swimmers. *International Journal of Sports Nutrition* 5(4), 315–328.

Hargreaves, M., Hawley, J.A., & Jeukendrup, A. (2004) Pre-exercise carbohydrate and fat ingestion: effects on metabolism and performance. *Journal of Sports Sciences* 22(1), 31–38.

Hess, M.A. (1997) Taste: the neglected nutritional factor. *Journal of the American Dietetic Association* 97(10), S205–S207.

Kittler, P.G. & Sucher, K.P. (2008) *Food and Culture*, 5th edn. Thomson Wadsworth, Belmount, CA.

Leiper, J.B., Junge, A., Maughan, R.J., et al. (2008) Alteration of subjective feelings in football players undertaking their usual training and match schedule during the Ramadan fast. *Journal of Sports Sciences* 26 (Suppl. 3), S55–S69.

Levy, A.S., Fein, S.B., & Schucker, R.E. (1992) More effective nutrition label formats are not necessarily preferred. *Journal of the American Dietetic Association* 92(10), 1230–1235.

Levy, A.S., Fein, S.B., & Schucker, R.E. (1996) Performance characteristics of seven nutrition label formats. *Journal of Public Policy and Marketing* 15, 1–15.

London Organising Committee of the Olympic Games and Paralympic Games Ltd (2009) *Food Vision for the London 2012 Olympic Games and Paralympic Games*.

Los Angeles Olympic Organising Committee (1984) *The Official Report of the Organising Committee of the Games of the XXIIIrd Olympiad Los Angeles 1984. Volume 1: Organization and planning*. Los Angeles Olympic Organising Committee (LAOOC), Los Angeles, CA.

Lyberg, W. (1997) Swedish Imagination. Gastronomoy at the Olympic Games. Part 1. *Olympic Reviews*, 26, 50–52.

Mavrommatis, E. (1997) To each his own…menu! Gastronomy at the Olympic Games. Part 1. *Olympic Reviews*, 26, 34–35.

Mintz, S. & Sclettwein-Gsell, D. (2001) Food patterns in agrarian societies: the "core-fringe-legume hypothesis". A dialogue. *Gastronomica* 1, 41–59.

Mujika, I., Chaouachi, A., & Chamari, K. (2010) Precompetition taper and nutritional strategies: special reference to training during Ramadan intermittent fast. *British Journal of Sports Medicine* 44(7), 495–501.

Nestle, M., Wing, R., Birch, L., et al. (1998) Behavioral and social influences on food choice. *Nutrition Reviews* 56(5 Pt. 2), S50–S64.

Nowak, M. & Speare, R. (1996) Gender differences in food-related concerns, beliefs and behaviours of north Queensland adolescents'. *Journal of Paediatrics and Child Health* 32, 424–427.

Organisationskomitee fur die XI Olympiade (1937) *The Official Report of the XIth Olympiad, Berlin 1936*. Berlin Organisationskomitee fur die XI Olympiade Berlin 1936.

The Organising Committee for the I Olympiad (1896) *The Official Report of the Games of the I Olympiad, Athens 1896*. The Organising Committee for the I Olympiad, Athens, Greece.

The Organising Committee for the XV Olympiad (1955) *The Official Report of the Organising Committee for the XV Olympiad, Helsinki 1952*. The Organising Committee for the XV Olympiad, Helsinki, Finland.

The Organising Committee of the Games of the XVII Olympiad (1960) *The Official Report of the Organising Committee for the XVII Olympiad, Rome 1960*. The Organising Committee of the Games of the XVII Olympiad, Rome.

The Organising Committee of Games of the XIX Olympiad (1969) *The Official Report for the Organising Committee of the XIX Olympiad, Mexico 1968*. The Organising Committee of Games of the XIX Olympiad, Mexico.

The Organising Committee of the 1996 Atlanta Olympic Games (1997) *The Official Report of the Centennial Olympic Games, Atlanta 1996. Volume 1: Planning and Organization*. International Olympic Committee, Atlanta, GA.

Pelly, F. (2006) A comprehensive environmental nutrition intervention for athletes competing at the Sydney 2000 Olympic Games. Unpublished thesis. University of Sydney, Australia.

Pelly, F.E. & Burkhart, S. (2013) The dietary regimens of athletes: the influence of culture, religion and sport, *International Journal of Sports Nutrition and Exercise Metabolism* (in press).

Pelly, F., Denyer, G., O'Connor, H., et al. (2004) Nutrition at the Sydney 2000 Olympic Games [abstract]. *Journal of Science and Medicine in Sport* 7(4), 31.

Pelly, F., Inge, K., King, T., et al. (2006a) *Provision of a Nutrition Support Service at the Melbourne 2006 Commonwealth Games*. 2nd Conference for the Australian Association of Exercise and Sport Sciences, 2006, Sydney, University of NSW Press, p. 132.

Pelly, F., King, T., & O'Connor, H. (2006b) *Factors Influencing Food Choice of Elite Athletes at an International Competition Dining Hall*. 2nd Conference for the Australian Association of Exercise and Sport Sciences, 2006, Sydney, University of NSW Press, p. 131.

Pelly, F., O'Connor, H., Denyer, G., et al. (2007) An environmental nutrition intervention at the Sydney 2000 Olympic Games. *Medicine & Science in Sport & Exercise* 39(5), S291.

Pelly, F., O'Connor, H., Denyer, G., et al. (2009) Catering for the athletes village at the Sydney 2000 Olympic Games: the role of sports dietitians. *International Journal of Sport Nutrition and Exercise Metabolism* 19(4), 340–354.

Pelly, F., Burkhart, S., Leech, K., et al. (2011a) Provision of nutrition support to athletes competing at the Delhi 2010 Commonwealth Games. *Medicine & Science in Sport & Exercise* 43, S502.

Pelly, F., O'Connor, H.T., Denyer, G.S., et al. (2011b) Evolution of food provision to athletes at the summer Olympic Games. *Nutrition Reviews* 69(6), 321–332.

Reichler, G. & Dalton, S. (1998) Chefs' attitudes towards healthful food preparation are more positive than their food science knowledge and practices. *Journal of the American Dietetic Association* 98(2), 165.

Satia, J.A., Patterson, R.E., Taylor, V.M., et al. (2000) Use of qualitative methods to study diet, acculturation, and health in Chinese-American women. *Journal of the American Dietetic Association* 100(8), 934–940.

Seoul Olympic Organising Committee (1989) *The Official Report of the Games of the XXIVth Olympiad Seoul 1988*. Olympic Organising Committee (SOOC), Seoul, Korea.

Smart, L.R. and Bisogni, C.A. (2001) Personal food systems of male college hockey players. *Appetite* 37, 57–70.

Wanik, J. (2009) Feeding the Beijing Olympics: an interview with Jillian Wanik. Interview by Louise M. Burke. *International Journal of Sport Nutrition and Exercise Metabolism* 19(4), 433–437.

PART 5

HEALTH-RELATED AND CLINICAL SPORTS NUTRITION

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Chapter 37

Nutrition, Physical Activity, and Health

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Introduction

In order to understand the impact of nutrition and physical activity on health it is imperative to keep in mind what the overall goal is. For the athlete, the primary goal of their training/nutrition habits (at least during their competitive careers) is optimum performance. For the nonathlete, the overall goal is generally optimum health, roughly defined as minimizing risk for disease and, hopefully, supporting an active lifestyle. For a striking number of nutritional considerations, the athlete is not different from the nonathlete and it is clearly true that athletes should not ignore basic healthy eating strategies nor follow a principle that more is always better. In athletes, however, the energetic and metabolic flux associated with sport creates cellular conditions that differ from the conditions present in people who are sedentary or even moderately active. Athletes' high energy expenditure requires a high energy intake in which much of their food is fuel; put it in the engine, burn it, get more. The effects of nutrition and physical activity are mainly confined to nutrition; there is little opportunity to gain health benefits by increasing their already high levels of physical activity. However, in nonathletes (including former athletes who are no longer competing at a high level), there is ample room to achieve health benefits by increasing the duration and/or intensity of physical activity to raise the overall metabolic flux. The effects of dietary intervention have to be viewed in the context of the frequency, duration, and intensity of exercise, which can be collectively termed the exercise "dose." In that framework, exercise can be viewed as a "drug" with both acute (each time exercise is performed) and chronic (exercise training) components. Like any drug, the exercise drug has different effects on different physiological/metabolic systems and it interacts with food. The focus of this chapter is to explain what is known, and what we still do not understand, about the ways that proper nutrition and physical activity can enhance health, and prevent, and even treat chronic disease.

Energy Flux: A Key Link between Energy Intake, Expenditure, and Disease

When considering chronic disease (e.g., heart disease, vascular disease, type 2 diabetes) and the conditions that predispose one to chronic disease (obesity and aging), energetic and metabolic flux are critical components. Flux is the concept of movement through a system. Low flux is a rather stagnant system, while high flux is dynamically changing. Nutrition represents entry into the system and physical activity can be a large exit from the system. In the athlete, flux can be very high. In opposition, conditions such as obesity are characterized by low energetic flux and adding body weight can be seen as an attempt to increase flux by increasing metabolically active tissue. It is thought

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that humans evolved during a period when large changes in flux rates were common (e.g., periods of high and low food availability with changing seasons, migrations to follow animal food sources) and that these changes were responsible for the gene expression to keep one metabolically healthy.

Barring the example of deliberate restriction of energy intake, in which a low energy intake can lower risk for disease and extend life span, chronically low flux rates, i.e., sedentary behavior, are clearly related to the dramatic rise in obesity and chronic diseases, including cardiovascular diseases (CVDs), diabetes, and hypertension. Because they are so strongly intertwined, these conditions have been lumped together under the umbrella of "cardiometabolic disease" (CMD). Low energy expenditure that is not matched by an equally low intake leads to energy surplus, fat storage, and, ultimately, obesity. Although there are other important factors involved (genetics, epigenetics, environmental exposure, social factors), the links between obesity and CMD are inarguable. In addition, it is now clear that low flux itself, in the form of inadequate exercise and/or excessive sedentary behavior (e.g., sitting), causes changes to physiology that dramatically raise the risk for CMD independent of obesity.

To understand the role that physical activity and sound nutrition play in opposing the metabolic abnormalities that underlie chronic disease, it is important to look at how inactivity and poor nutrition shape the pathophysiology of the disease processes. A comprehensive review of all of the ways in which changing energy intake and expenditure affect human health and disease would require an exceedingly long book by itself. Decades of careful research suggest that many of the key physiological processes that mediate the effects of low flux rate/energy surplus on human disease are captured in several broad categories including mitochondrial number/capacity/function, production, and removal of reactive oxygen species (ROS) that can be termed "redox balance" and sensitivity of cellular metabolic processes to insulin and endothelial function. In the ensuing paragraphs, these key aspects of physiology will be defined, their role in mediating CMD will be highlighted, and research showing the effect of changing physical activity or nutrition on these parameters will be summarized and interpreted. An important and clearly related category, inflammation, will be the main subject of Chapter 38 in this volume.

Mitochondrial Function and ROS

Mitochondrial Structure and Function

Proper mitochondrial function helps to maintain the healthy state and can contribute to extreme aerobic capacity. Reciprocally, it is thought that mitochondria dysfunction is causative to a variety of chronic diseases and aging. Mitochondrial function is highly dependent on mitochondrial structure and thus it is important to understand how these two relate.

The concept of what mitochondria are has been complicated in recent years. Once thought to be distinct organelles, it is now accepted that mitochondria are a reticulum (Kirkwood et al., 1986). The reticulum may exist as subpopulations in such tissues as skeletal muscle in order to support different energetic processes (e.g., membrane transport vs. contractile activity). The concept of the reticulum is complicated even further by its constant remodeling. It has been demonstrated that the mitochondrial reticulum undergoes a series of fission and fusion events and that motility is achieved by transient fusion events along separate microtubules (Liu et al., 2009b). The coordination of these events is impressive given that with each fusion event the inner and outer membrane of two structures must be fused, essentially requiring the regulation of four membranes.

The making of new mitochondria is referred to as mitochondrial biogenesis. Given the aforementioned complexity of mitochondrial remodeling, it is not entirely clear what mitochondrial biogenesis is. Mitochondria are not made *de novo*, but rather recruit new proteins to an existing organelle with subsequent division by fission (Ryan & Hoogenraad, 2007). Therefore, mitochondrial "biogenesis" is really determined by the making of new mitochondrial proteins, the majority of which are encoded by nuclear genes with a much smaller number from mitochondrial DNA. The confusion surrounding "what" mitochondrial biogenesis is has led to a variety of markers to assess mitochondrial biogenesis,

some of which (e.g., PGC-1α mRNA) are of questionable relevance. Nonetheless, the measurement of mitochondrial biogenesis remains important since triggering the process could be therapeutic for chronic disease.

The inner membrane of mitochondria contains a series of complexes required for the electron transport chain. In this process, electrons given up by the energy-rich electron carriers, NADH and FADH2, are sequentially transferred by a series of four protein complexes (three in the case of FADH2) and two mobile electron carriers. The energetically favorable passage of these electrons through the chain is aided by three of the complexes which are also enzymes; NADH dehydrogenase (complex I), cytochrome *c* reductase (complex III), and cytochrome *c* oxidase (complex IV). The final step in the chain is for pairs of electrons to reduce molecular oxygen to water, hence the familiar designation of oxygen as the "terminal electron acceptor." The enzyme complexes also function as pumps, which move protons from the mitochondrial matrix to the inter-mitochondrial membrane space. The pumping of these protons creates an electrochemical gradient across the inner mitochondrial membrane (the energy contained in the separation of positive and negative charges is similar to the potential energy contained in a lake being held back by a dam). The potential energy generated by the pumping of protons can be exploited by allowing the protons to fall back across the membrane. ATP synthase uses the net change in free energy to phosphorylate ADP to ATP. The total energy extracted from the complete oxidation of one glucose in the TCA cycle and electron transport chain is 36–37 ATP, depending on whether the glucose was derived from the blood or from muscle glycogen.

The transfer of electrons is not an error-free process and can result in the production of ROS. More specifically, complexes I and III of the electron transport chain are the main sites of superoxide production. On the way to complex III coenzyme Q (ubiquinone) has two electrons. After one electron is passed on, an intermediate called ubisemiquinone is susceptible to interaction with oxygen to which it can donate its electron thus generating superoxide. Other ROS include hydrogen peroxide and hydroxyl radical. Further, the reactive nitrogen species nitric oxide has the potential to react with superoxide to generate peroxynitrite. Higher metabolic flux rates, and consequently increased electron transport chain activity, increases ROS generation. Finally, although mitochondria are a source of ROS, it is important to mention that they are not the sole source of ROS during contractile activity (Powers et al., 2011).

Although mitochondria produce ROS, there are abundant cellular processes to combat (or reduce) ROS. Superoxide dismutase (SOD), catalase, and glutathione peroxidase are the primary enzymes to reduce ROS (eventually) to water. The body also contains nonenzymatic antioxidants such as glutathione, vitamin C, vitamin E, and lipoic acid. In addition to the endogenous antioxidants, exogenous antioxidants can also be consumed as part of the diet in the form of micronutrients, trace elements, and other bioactive compounds. The endogenous antioxidant system operates in intracellular and extracellular compartments and may be compartmentalized even further in the intracellular environment.

The balance between ROS production and antioxidant defenses is referred to as redox balance or oxidative stress. Tipping one direction or the other on the balance leads to a more oxidative or reduced environment. Oxidative stress is believed to be associated with more than 200 diseases (Halliwell & Gutteridge, 2007; Salmon et al., 2010a; Willcox et al., 2008) and with the normal aging process (Salmon et al., 2010b). For many years, research was focused on how to prevent the more oxidative environment since this was thought to, and indeed can, lead to disease states. However, now it has become apparent that an environment that is too reduced can also be detrimental since ROS are important adaptive signals (Mattson, 2008). Maintaining a proper balance and not deviating to one extreme is consistent with the idea of hormesis.

Hormesis

A good working definition of hormesis is "a process in which exposure to a low dose of a chemical agent or environmental factor that is damaging at higher doses induces an adaptive beneficial effect on the

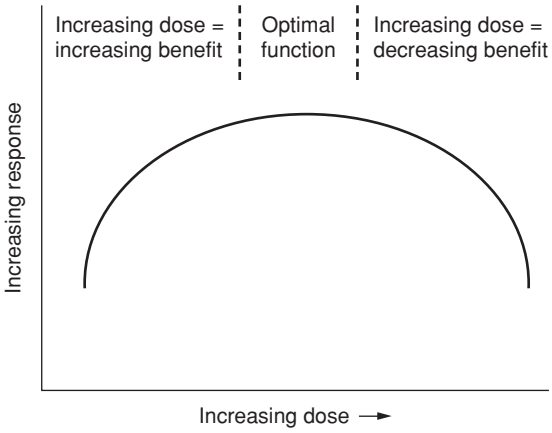

Figure 37.1 Concept of hormesis. Increasing dose results in increased benefit until a range of optimal function. From this point, increasing dose results in detrimental outcomes. Concept is useful in a variety of contexts including exercise training, nutrition, and physiological stimuli such as reactive oxygen species.

cell or organism" (Mattson, 2008). In simple terms, a little is good, but a lot is bad (Figure 37.1).

The concept of hormesis is easy to understand in terms of such examples as a virus where a little exposure induces an antibody response that protects the organism in the future. But, hormesis is less intuitive when discussing something such as exercise that is thought to be "healthy." In the case of exercise, too much can lead to a failure to further adapt inducing a state of overtraining (see Chapter 33). The hormetic response is important when considering ROS.

There is now strong evidence that ROS act as signaling molecules (Powers et al., 2011). Therefore, there is a need for ROS to induce adaptive responses in the body and the absence of ROS may lead to dysfunction. On the opposite side of the spectrum, too many ROS or a failure of antioxidant systems to deal with ROS (oxidative stress) can lead to dysfunction. Therefore, there is an appropriate range of ROS that leads to proper cellular and physiological function. Apart from external environmental factors (e.g., radiation), physical activity and nutrition play a primary role in determining oxidative stress and therefore adaptive (positive or negative) responses.

Physical Activity and Redox Balance

Contractile activity increases ROS generation (Davies et al., 1982), a finding that initially led some to question whether exercise was "damaging." However, although exercise increases ROS generation, it also increases the ability to deal with free radicals by increasing antioxidant protection (Ji, 2007). The end result is acute increases in ROS after a bout of exercise, followed by enhanced antioxidant defenses.

What has emerged in recent years is that the acute increases in ROS after a bout of exercise are powerful signals for cellular adaptation. For example, an acute bout of exercise signals for increases in SOD, a key endogenous enzymatic antioxidant (Hollander et al., 1999). Further, ROS signaling can activate the peroxisome proliferator–activated receptor gamma coactivator (PGC)-1α activated signaling pathway to potentially increase mitochondrial biogenesis (Handschin & Spiegelman, 2006; Kang et al., 2009). In short, the signals generated by ROS stimulate the making of proteins to better deal with oxidative stress and to potentially decrease ROS generation. It has been pretty convincingly demonstrated that that ROS signaling is important for many exercise training responses (Gomez-Cabrera et al., 2008; Ristow et al., 2009).

Nutrition and Oxidative Stress

Because some vitamins have antioxidant properties, supplementation with these vitamins was advanced as a means to decrease oxidative stress. Indeed, in experiments in which animals were vitamin E (Davies et al., 1982) or vitamin C (Packer et al., 1986) deficient, there were demonstrated performance decrements. However, supplementation in those not deficient does not always increase exercise performance (Powers & Jackson, 2008), and in some cases may even decrease performance (Gomez-Cabrera et al., 2008; Ristow et al., 2009). Further, many clinical studies using exogenous antioxidant supplementation in clinical populations have failed to find a benefit of supplementation (Bjelakovic et al., 2007). It is likely that exogenous antioxidant compounds that react stoichiometrically with oxidants

cannot be consumed in large enough quantities to meet the oxidant demand. Further, mammalian biology does not rely on consumption of antioxidants to maintain redox balance because excess oxidants are quenched by powerful and ubiquitous cellular enzymatic antioxidant pathways (Halliwell & Gutteridge, 2007; Powers & Hamilton, 1999). Alternatively, it is possible that provision of antioxidants through dietary supplementation may be blunting some of the signaling induced by ROS to the cell's detriment. In other words, removing ROS signaling may blunt some of the hormetic response of oxidative stress.

An alternative means of inducing ROS protection has emerged in which the endogenous antioxidant system is activated by nutritional supplementation. Nrf2 is a transcription factor that regulates the gene expression of antioxidant proteins through an enhancer sequence known as the antioxidant responsive element (ARE) (Lee et al., 2005). Nrf2 has been termed "the master regulator" of the ARE-driven cellular defense system against oxidative stress. In the presence of oxidative stress associated with injury, illness, or various biological changes, Nrf2 is activated to relocate to the nucleus. Here, Nrf2 activates the transcription of ARE-driven genes. A second means of activation does not require oxidative stress but, rather, involves the phosphorylation of Nrf2. It has been shown that Nrf2 can be activated via this pathway by treatment with phytochemicals (Nelson et al., 2006). Therefore, there exists a means to upregulate the endogenous antioxidants system by phytochemicals that do not have direct antioxidant properties. The feasibility of these treatments for disease conditions is currently being evaluated, and preliminary evidence is promising (Bogaard et al., 2009; Liu et al., 2009a; Qureshi et al., 2010).

When researching potential supplements, there is a propensity to study individual components in isolation from their biological context. However, it is more likely that biologically active compounds have their effects in the context of the other compounds around them. This concept is referred to as an emergent property and is borrowed from art and philosophy. In 1875, the psychologist G.H. Lewes offered this description of emergence: "Every resultant is either a sum or a difference of the co-operant forces; their sum, when their directions are the same - their difference, when their directions are contrary … The emergent is unlike its components insofar as these are incommensurable, and it cannot be reduced to their sum or their difference." In other words, the sum of many components in a system is often different from any in isolation and is not equal to the sum of the individual components. Therefore, the beneficial effect of a piece of fruit may not be in its vitamin C or vitamin E content, but rather how that vitamin C acts synergistically with other phytochemicals to have a positive health outcome. Some have taken this as far to suggest that, " … previously published findings tentatively suggest that fruits and vegetables may exert health-promoting effects despite their antioxidant content …" (Ristow et al., 2009).

Emerging Directions in Disease Prevention

The interactions between exercise, nutrition, oxidative stress, and disease are also dependent on the time of exposure. Prolonged oxidative stress (e.g., months, years) is associated with chronic disease and aging. However, acute increases in oxidative stress (e.g., seconds to hours) signal important adaptive responses that likely result in decreased overall oxidative stress (hormesis). Exercise and nutrition are critical components in this process because of their ability to modulate oxidative stress. Although oxidative stress is associated with many disease states and aging, it remains to be clearly delineated if the oxidative stress is a cause or a result of the disease state. There is strong evidence that physical activity over time decreases oxidative stress (Ji et al., 1998) and disease progression (Diabetes Prevention Program Research Group, 2002; Wang et al., 2002). So far, supplementation with exogenous antioxidants has shown disappointing results in diseased conditions (Bjelakovic et al., 2007). It is likely that these disappointing results are because of the mode of antioxidant administration. There is stronger evidence that foods with antioxidant properties (Abete et al., 2011) or foods that activate the endogenous antioxidant system indirectly (Nelson et al., 2006) are better means to prevent chronic disease than high doses of single antioxidants. Relatively unknown is how physical activity and

nutrition interact (deleterious, additive, synergistic) to change oxidative stress and disease progression.

Insulin Resistance/Endothelial Function

Insulin Secretion and Action

Although insulin has hundreds of functions, this review will focus on its effects in target tissues that are modulated by changes in diet and/or exercise. In broad strokes, actions of insulin are anabolic and promote nutrient/energy storage while inhibiting the catabolic processes of nutrient/energy mobilization. In muscle, insulin facilitates glucose uptake from the blood, its storage as glycogen or oxidation for energy and under certain conditions, the incorporation of amino acids into protein. In the liver, insulin stimulates storage of liver glycogen and inhibits glucose production and the export of glucose to the blood. In adipose tissue, insulin enhances fatty acid uptake and storage as triacylglycerol while inhibiting lipolysis. In the endothelium, insulin promotes vasodilation and increased blood flow presumably to increase nutrient delivery.

The net effect of circulating insulin on target tissues like muscle, liver, fat, and the endothelium will depend on the circulating concentration (a function of insulin secretion to, and clearance from, the circulation) and insulin action (also called insulin sensitivity). In addition to a low basal secretion of insulin into the blood, there is a sharp rise in the secretion of insulin after every meal, with the magnitude of secretion roughly approximating the amount and type of carbohydrate in the meal. Insulin action is broadly defined as the dose-response between circulating insulin concentrations in the blood and the magnitude of its effects on target tissue. Impaired insulin action is called insulin resistance and, as previously mentioned, is a hallmark of CMD (DeFronzo & Tripathy, 2009). Chronically, changes to basal insulin secretion and the insulin response to a meal usually track insulin action. As insulin resistance rises, circulating concentrations of insulin follow, a response known as compensatory hyperinsulinemia. Insulin resistance and hyperinsulinemia are strongly associated with greatly increased risk for CVD and hypertension (Reaven, 2011). For reasons not completely understood, the ability to compensate for insulin resistance with hyperinsulinemia is gradually lost in many people over time, leading to type 2 diabetes (see Chapter 40).

Effects of Physical Activity on Insulin Action

During exercise, there is an uptake of glucose by working muscle that is independent of any residual insulin-stimulated clearance of glucose from the blood (for an excellent review, see Holloszy, 2005). After exercise, this non-insulin-mediated glucose uptake into muscle persists for several hours and facilitates glycogen replenishment. Prior exercise also raises subsequent insulin-mediated uptake of glucose into muscle and adipose tissue, thus increasing insulin sensitivity. Exercise also accentuates the effects of circulating insulin to constrain liver glucose production and export into the blood, and has beneficial effects on lipid metabolism (e.g., more oxidation and less storage of fat) although these effects are not as reliably observed. Enhanced insulin action persists for 12–48 hours or so after exercise, depending on the exercise intensity, duration, and the nutritional "context" in terms of postexercise energy and carbohydrate intake (Cartee et al., 1989).

Habitual exercise training confers adaptations that increase insulin action in muscle, liver, and adipose tissue similarly to acute exercise but the mechanisms are likely different. Lower body fat (particularly the visceral fat stored in the abdomen around the organs), liver fat, greater mitochondrial density, and increased capillarization of muscle beds (among other adaptations) contribute to better insulin action. Much recent work has been focused on the effects of habitual exercise to enhance mitochondrial density/function (Lanza & Nair, 2009) and reduce the accumulation of lipids, particularly diacylglycerols and ceramides, stored in muscle (Dubé et al., 2011; Samuel et al., 2010).

Physical Activity and Dietary Energy Deficit

The interactions between habitual physical activity (e.g., exercise training) and dietary change have mostly been centered on exercise and lower energy

diets designed to promote weight (fat) loss. Dietary changes that induce an energy deficit (energy intake less than expenditure) to achieve fat loss potentiate the effects of exercise training (Dubé et al., 2011; Haus et al., 2011). Profound benefits to insulin action, blood sugar regulation, fat oxidation, inflammatory cytokines, and many other indices of cardiometabolic health are often observed when physical activity/dietary change results in even modest weight loss (Dubé et al., 2011; Haus et al., 2011). These adaptations seem to occur independently of the dietary strategy used to achieve the energy deficit (e.g., high carbohydrate, low carbohydrate diets). So, if maximum fat loss is desired, any dietary regimen that provides adequate energy and macronutrients to fuel a physically active lifestyle without any "extra" energy is optimal. Switching low glycaemic index (GI) for high GI foods in combination with more physical activity may confer some advantages in terms of insulin action and lipid metabolism (Haus et al., 2011) but the differences are subtle compared with the striking contrast between being sedentary or physically active. Enough energy must be consumed to promote satiety or the resultant rise in appetite will hinder long-term weight maintenance. Too much energy will minimize weight loss and likely lead to frustration that the exercise/dietary regimen is "not working." Blanket recommendations are probably counterproductive because every individual is an "*n* of 1" and counseling will require balancing the competing demands of physical activity, appetite, satiety, and weight loss/maintenance.

Interactions between Acute Exercise and Nutrition

Consuming energy after exercise erodes the acute benefits of exercise on insulin action, but whether this effect is due to energy intake *per se* or related to one or more specific macronutrients is unclear (Black et al., 2005; Newsom et al., 2010). The timing of the energy consumption before, immediately after, or several hours after exercise appears to be relatively unimportant with only subtle differences noted when timing is varied (Stephens & Braun, 2008). Consuming meals low in carbohydrate may potentiate the magnitude of the change and the length of the insulin-sensitizing effects of recent exercise in both animals (Cartee et al., 1989) and humans (Newsom et al., 2010). High sensitivity to insulin after exercise facilitates the rapid uptake of glucose and replacement of muscle glycogen in response to consumption of energy (especially carbohydrates). While an advantage to athletes for subsequent training/competition, the rapid replenishment of glycogen reverses the beneficial effects of exercise on insulin action (Cartee et al., 1989), although many other benefits are retained.

Single Nutrients

Some potentially beneficial effects on insulin resistance or the regulation of blood glucose have been reported for a variety of single foods, such as the trace mineral chromium (Nahas & Moher, 2009) and the common spices cinnamon (Nahas & Moher, 2009), curcumin (turmeric; Alappat & Awad, 2010), and the antioxidant epigallocatechin gallate (a component of tea; Hsu et al., 2011). The consistency of these results has not yet been verified in large-scale clinical trials. If there are positive effects, they are likely to be more subtle than the robust effects of acute or habitual physical activity. Potential interactions between exercise and these, or other nutrients, has not yet been systematically evaluated.

Endothelial Function

As noted above, there is tight link between insulin resistance and CVD. Lack of physical activity and inadequate diet cause changes to the vascular endothelium that parallel changes to insulin sensitivity and are some of the first signs of CVD (Mora et al., 2007).

The endothelium is a smooth, slippery surface that facilitates passage of blood through vessels. A layer of smooth muscle surrounding the endothelium responds to signals to contract and relax, causing vasoconstriction and vasodilation. The balance between signals to vasoconstrict and vasodilate is referred to as vascular tone. Insulin is partly responsible for maintenance of vascular tone by modulating the production of nitric oxide and endothelin-1

(Formoso et al., 2006). Nitric oxide is a potent vasodilator produced in the endothelium that relaxes smooth muscle to cause vasodilation. Endothelin-1 acts in opposition, causing contraction of smooth muscle and vasoconstriction. Insulin resistance lowers production of nitric oxide and increases production of endothelin-1 (Kashyap et al., 2005). *In vivo*, the two most widely accepted indicators of endothelial function are flow-mediated dilation (FMD) and the presence of circulating endothelial progenitor cells (EPCs). As a general rule, FMD (the expansion of arterial diameter in response to reperfusion after venous occlusion) is about 11% in healthy individuals but can be close to zero in people with CVD. EPCs are one type of the broad class of cells known as circulating angiogenic cells (CACs): they are derived from bone marrow and mature in the circulation to endothelial cells and have a key role in vessel maintenance and repair (Witkowski et al., 2011). Although not exhaustively studied, there appear to be beneficial effects of both exercise and diet on EPCs.

The most obvious candidate to explain the beneficial effect of acute exercise on the endothelium is the shear stress that results from higher velocity blood flow through vessels. Increases in shear stress cause rises in the levels of mRNA, protein, and activity of the vasodilator endothelial nitric oxide synthase (eNOS) and lower the availability of the vasoconstrictor endothelin-1) (Newcomer et al., 2011). Because the beneficial effects of exercise are not limited to the vessels in the working muscle (e.g., exercise by the lower limbs also causes changes in the upper extremity and vice versa) it appears that some portion of the effects is mediated systemically. In general, FMD is higher in concert with increased anterograde (forward) flow through vessels with the opposite effects associated with retrograde (backward) flow and more complex responses to oscillatory flow that might be expected during cycling (Newcomer et al., 2011). More favorable changes have been reported after moderate-to-high intensity exercise than with prolonged lower-intensity activity: this is consistent with the idea that beneficial changes to the endothelium are linked with higher rates of flow and shear stress but there are many other metabolic and cardiovascular differences as exercise intensity increases.

Adaptations associated with chronic exercise seem to be predicated on exposure to elevated shear stress. In one clever study, investigators prevented a rise in shear stress during exercise in one arm by venous occlusion (Tinken et al., 2009). While handgrip training increased forearm strength and size in both limbs, endothelial function improved only in the arm that was exposed to elevated shear stress. Although these results imply that direct effects of shear stress are a necessary stimulus to enhance vascular adaptation after exercise training, some non-exercise studies indicate that mechanisms other than shear stress are also important (Newcomer et al., 2011).

Both acute exercise and exercise training raise the concentration of circulating EPCs and improve EPC function in healthy adults (Witkowski et al., 2011). In young regularly exercising men, the density of EPCs in the circulation was higher, they had greater eNOS expression and more intercellular nitric oxide compared with sedentary men at rest. Further, 10 days of inactivity led to a decrease in circulating EPC in chronically active men (Witkowski et al., 2010).

Emerging Directions in Endothelial Function

As with insulin resistance, there is a strong association between changes in endothelial function and oxidative stress/inflammatory processes. Obesity, in particular the central adiposity that is characterized by excessive visceral fat, is tightly related to both endothelial dysfunction and insulin resistance. It is likely that the same dietary factors that attenuate insulin resistance, i.e., energy deficit, fat loss, and central fat loss will also oppose and may even reverse endothelial dysfunction. Therefore, any dietary intervention that facilitates a modest energy deficit and consistent loss of body fat is a useful nutritional strategy to maintain/enhance the endothelium and minimize the risk for CVD. There are effects of dietary nitrites and nitrates on nitric oxide synthesis and eNOS that may play independent roles or interact with exercise to modulate endothelial function (Machha & Schechter, 2011). In general, dietary nitrates (e.g., high in some plant-based foods like beets) may enhance FMD while whole nitrites (e.g., high in smoked meats) may do the opposite (Machha & Schechter, 2011).

Practical Recommendations for People Whose Goals Are to Use Nutrition and Exercise to Enhance Health/Prevent Disease Rather Than Elite Athletic Performance

The focus of this review has been on individuals whose goals are to use nutrition and exercise to enhance health and prevent disease rather than optimize athletic performance. Although the research is ongoing and the knowledge base is continually shifting, a few general themes are warranted by the research to date.

1. Individual variability attributable to differences in genetics, epigenetics, environment, sex, race, ethnicity, and so on, makes developing "general" recommendations perilous. In addition, because different goals require somewhat different strategies (e.g., you cannot just eat/exercise to maximize insulin sensitivity because muscle quantity and quality are keys to good health also), "one size fits all" blanket recommendations are rarely useful for an individual. Therefore, nutrition professionals should be used to help identify shortcomings and goals in order to tailor recommendations for improvement.
2. A well-rounded diet composed mainly of whole foods, rather than individual components, takes advantage of synergy and emergent properties of nutrients and phytochemicals. Therefore, for general health, there is no substitute for a well-rounded diet.
3. Unless there are recognized nutrient deficiencies, reliance on dietary supplements to fill gaps in the diet or provide "extra" nutrition is probably not a useful strategy. As with other dose-response and hormetic effects, supplementation of individual nutrients far in excess of recommendations can lead to detrimental outcomes.
4. To help maintain appropriate body weight and reduce risk for obesity-related disease, an emphasis on low-energy-density foods can promote satiety and potentially oppose any rise in appetite in response to habitual exercise.
5. Although they can be useful to promote performance in athletes, consumption of high GI foods and beverages that provoke a large insulin response (e.g., sport drinks, energy bars) for people using physical activity to enhance health is not necessary and may be counterproductive. If people do consume carbohydrate after exercise, the addition of protein (e.g., chocolate milk or yogurt) may promote other health benefits such as a gain in lean body mass.

Sources for More Information

World Health Organization Global Strategy on Diet, Physical Activity, and Health
http://www.who.int/dietphysicalactivity/factsheet_recommendations/en

Centers for Disease Control and Prevention: Division of Nutrition, Physical Activity, and Obesity
http://www.cdc.gov/nccdphp/dnpao/index.html

Exercise is Medicine Global Initiative
http://exerciseismedicine.org/global.htm

National Center for Complementary and Alternative Medicine
http://nccam.nih.gov

References

Abete, I., Goyenechea, E., Zulet, M.A., et al. (2011) Obesity and metabolic syndrome: potential benefit from specific nutritional components. *Nutrition, Metabolism & Cardiovascular Diseases* 21(Suppl. 2), B1–B15.

Alappat, L. & Awad, A.B. (2010) Curcumin and obesity: evidence and mechanisms. *Nutrition Reviews* 68, 729–738.

Bjelakovic, G., Nikolova, D., Gluud, L.L., et al. (2007) Mortality in randomized trials of antioxidant supplements for primary and secondary prevention: systematic review and meta-analysis. *Journal of the American Medical Association* 297, 842–857.

Black, S.E., Mitchell, E., Freedson, P.S., et al. (2005) Improved insulin action following short-term exercise training: role of energy and carbohydrate balance. *Journal of Applied Physiology* 99, 2285–2293.

Bogaard, H.J., Natarajan, R., Henderson, S.C., et al. (2009) Chronic pulmonary artery pressure elevation is insufficient to explain right heart failure. *Circulation* 120, 1951–1960.

Cartee, G.D., Young, D.A., Sleeper, M.D., et al. (1989) Prolonged increase in insulin-stimulated glucose transport in muscle after exercise. *American Journal of Physiology—Endocrinology and Metabolism* 256, E494–E499.

Davies, K.J., Quintanilha, A.T., Brooks, G.A., et al. (1982) Free radicals and tissue damage produced by exercise.

Biochemical and Biophysical Research Communications 107, 1198–1205.
DeFronzo, R.A. & Tripathy, D. (2009) Skeletal muscle insulin resistance is the primary defect in type 2 diabetes. *Diabetes Care* 32(Suppl. 2), S157–S163.
Diabetes Prevention Program Research Group (2002) Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine* 346, 393–403.
Dubé, J.J., Amati, F., Toledo, F.G., et al. (2011) Effects of weight loss and exercise on insulin resistance, and intramyocellular triacylglycerol, diacylglycerol and ceramide. *Diabetologia* 54, 1147–1156.
Formoso, G., Chen, H., Kim, J.A., et al. (2006) Dehydroepiandrosterone mimics acute actions of insulin to stimulate production of both nitric oxide and endothelin 1 via distinct phosphatidylinositol 3-kinase- and mitogen-activated protein kinase-dependent pathways in vascular endothelium. *Molecular Endocrinology* 20, 1153–1163.
Gomez-Cabrera, M.C., Domenech, E., Romagnoli, M., et al. (2008) Oral administration of vitamin C decreases muscle mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. *American Journal of Clinical Nutrition* 87, 142–149.
Halliwell, B. & Gutteridge, J. (2007) *Free Radicals in Biology and Medicine*. Oxford University Press, New York.
Handschin, C. & Spiegelman, B.M. (2006) Peroxisome proliferator-activated receptor gamma coactivator 1 coactivators, energy homeostasis, and metabolism. *Endocrine Reviews* 27, 728–735.
Haus, J.M., Solomon, T.P., Lu, L., et al. (2011) Intramyocellular lipid content and insulin sensitivity are increased following a short-term low-glycemic index diet and exercise intervention. *American Journal of Physiology—Endocrinology and Metabolism* 301, E511–E516.
Hollander, J., Fiebig, R., Gore, M., et al. (1999) Superoxide dismutase gene expression in skeletal muscle: fiber-specific adaptation to endurance training. *American Journal of Physiology* 277, R856–R862.
Holloszy, J.O. (2005) Exercise-induced increase in muscle insulin sensitivity. *Journal of Applied Physiology* 99, 338–343.
Hsu, C.H., Liao, Y.L., Lin, S.C., et al. (2011) Does supplementation with green tea extract improve insulin resistance in obese type 2 diabetics? A randomized, double-blind, and placebo-controlled clinical trial. *Alternative Medicine Review* 16, 157–163.
Ji, L.L. (2007) Antioxidant signaling in skeletal muscle: a brief review. *Experimental Gerontology* 42, 582–593.
Ji, L.L., Leeuwenburgh, C., Leichtweis, S., et al. (1998) Oxidative stress and aging. Role of exercise and its influences on antioxidant systems. *Annals of the New York Academy of Sciences* 854, 102–117.
Kang, C., O'Moore, K.M., Dickman, J.R., et al. (2009) Exercise activation of muscle peroxisome proliferator-activated receptor-gamma coactivator-1alpha signaling is redox sensitive. *Free Radicals in Biology and Medicine* 47, 1394–1400.
Kashyap, S.R., Roman, L.J., Lamont, J., et al. (2005) Insulin resistance is associated with impaired nitric oxide synthase activity in skeletal muscle of type 2 diabetic subjects. *Journal of Clinical Endocrinology and Metabolism* 90, 1100–1105.
Kirkwood, S.P., Munn, E.A., & Brooks, G.A. (1986) Mitochondrial reticulum in limb skeletal muscle. *American Journal of Physiology* 251, C395–C402.
Lanza, I.R. & Nair, K.S. (2009) Muscle mitochondrial changes with aging and exercise. *American Journal of Clinical Nutrition* 89, 467S–471S.
Lee, J.M., Li, J., Johnson, D.A., et al. (2005) Nrf2, a multi-organ protector? *FASEB Journal* 19, 1061–1066.
Liu, J., Gu, X., Robbins, D., et al. (2009a) Protandim, a fundamentally new antioxidant approach in chemoprevention using mouse two-stage skin carcinogenesis as a model. *PLoS ONE* 4, e5284.
Liu, X., Weaver, D., Shirihai, O., et al. (2009b) Mitochondrial 'kiss-and-run': interplay between mitochondrial motility and fusion-fission dynamics. *EMBO Journal* 28, 3074–3089.
Machha, A. & Schechter, A.N. (2011) Dietary nitrite and nitrate: a review of potential mechanisms of cardiovascular benefits. *European Journal of Nutrition* 50, 293–303.
Mattson, M.P. (2008) Hormesis defined. *Ageing Research Reviews* 7, 1–7.
Mora, S., Cook, N., Buring, J.E., et al. (2007) Physical activity and reduced risk of cardiovascular events: potential mediating mechanisms. *Circulation* 116, 2110–2118.
Nahas, R. & Moher, M. (2009) Complementary and alternative medicine for the treatment of type 2 diabetes. *Canadian Family Physician* 55, 591–596.
Nelson, S.K., Bose, S.K., Grunwald, G.K., et al. (2006) The induction of human superoxide dismutase and catalase *in vivo*: a fundamentally new approach to antioxidant therapy. *Free Radicals in Biology and Medicine* 40, 341–347.
Newcomer, S.C., Thijssen, D.H.J., & Green, D.J. (2011) Effects of exercise on endothelium and endothelium/smooth muscle cross-talk: role of exercise-induced hemodynamics. *Journal of Applied Physiology* 111, 311–320.
Newsom, S.A., Schenk, S., Thomas, K.M., et al. (2010) Energy deficit after exercise augments lipid mobilization but does not contribute to the exercise-induced increase in insulin sensitivity. *Journal of Applied Physiology* 108, 554–560.
Packer, L., Gohil, K., deLumen, B., et al. (1986) A comparative study on the effects of ascorbic acid deficiency and supplementation on endurance and mitochondrial oxidative capacities in various tissues of the guinea pig. *Comparative Biochemistry and Physiology* 83, 235–240.
Powers, S.K. & Hamilton, K. (1999) Antioxidants and exercise. *Clinical Journal of Sports Medicine* 18, 525–536.
Powers, S.K. & Jackson, M.J. (2008) Exercise-induced oxidative stress: cellular mechanisms and impact on muscle force production. *Physiological Reviews* 88, 1243–1276.
Powers, S.K., Talbert, E.E., & Adhihetty, P.J. (2011) Reactive oxygen and nitrogen species as intracellular signals in skeletal muscle. *Journal of Physiology* 589, 2129–2138.
Qureshi, M.M., McClure, W.C., Arevalo, N.L., et al. (2010) The dietary supplement protandim decreases plasma osteopontin and improves markers of oxidative stress in muscular dystrophy Mdx mice. *Journal of Dietary Supplements* 7, 159–178.
Reaven, G.M. (2011) Insulin resistance: the link between obesity and cardiovascular disease. *Medical Clinics of North America* 95, 875–892.
Ristow, M., Zarse, K., Oberbach, A., et al. (2009) Antioxidants prevent health-promoting effects of physical exercise in humans. *Proceedings of the National Academy of Sciences of USA* 106, 8665–8670.
Ryan, M.T. & Hoogenraad, N.J. (2007) Mitochondrial-nuclear communications. *Annual Review of Biochemistry* 76, 701–722.

Salmon, A.B., Richardson, A., & Perez, V.I. (2010) Update on the oxidative stress theory of aging: does oxidative stress play a role in aging or healthy aging? *Free Radicals in Biology and Medicine* 48, 642–655.

Samuel, V.T., Petersen, K.F., & Shulman, G.I. (2010) Lipid-induced insulin resistance: unravelling the mechanism. *Lancet* 375, 2267–2277.

Stephens, B.R. & Braun, B. (2008) Impact of nutrient intake timing on the metabolic response to exercise. *Nutrition Reviews* 66, 473–476.

Tinken, T.M., Thijssen, D.H.J., Hopkins, N.D., et al. (2009) Impact of shear rate modulation on vascular function in humans. *Hypertension* 54, 278–285.

Wang, B.W., Ramey, D.R., Schettler, J.D., et al. (2002) Postponed development of disability in elderly runners: a 13-year longitudinal study. *Archives of Internal Medicine* 162, 2285–2294.

Willcox, B.J., Willcox, D.C., & Ferrucci, L. (2008) Secrets of healthy aging and longevity from exceptional survivors around the globe: lessons from octogenarians to supercentenarians. *Journal of Gerontology and Aging in Biological and Medical Science* 63, 1181–1185.

Witkowski, S., Lockard, M.M., Jenkins, N.T., et al. (2010) Relationship between circulating progenitor cells, vascular function, and oxidative stress with long-term training and short-term detraining in older men. *Clinical Science (London)* 118, 303–311.

Witkowski, S., Jenkins, N.T., & Hagberg, J.M. (2011) Enhancing treatment for cardiovascular disease: exercise and circulating angiogenic cells. *Exercise and Sport Science Reviews* 39, 93–101.

Chapter 38

Exercise, Nutrition, and Inflammation

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Introduction

Inflammation is one of the oldest and best-recorded medical conditions. The classical symptoms of inflammation—rumor, tumor, calor, dolor, and *functio laesa* (redness, swelling, heat, pain, and loss of function, respectively)—represent a highly orchestrated series of biological events critical for host defense in response to harmful stimuli (Rock et al., 2010). While loss of these protective mechanisms would be fatal, work over the last decade has implicated chronic, low-grade inflammation in the pathophysiology of many chronic diseases, including insulin resistance, type 2 diabetes, neurodegeneration, atherosclerosis, and many cancers (Khandekah et al., 2011). Disordered regulation of inflammation and ways to address this imbalance are therefore important concepts in the context of health and prevention of noncommunicable diseases.

Individuals who engage in regular physical activity have a reduced risk of developing many of these chronic diseases (Bruunsgaard, 2005), but the molecular mechanisms responsible for the protective effects of exercise are not fully understood. Before the turn of the century, the observation was made that circulating cytokines—inflammatory signaling molecules—were elevated in response to exercise (Pedersen & Febbraio, 2008). This was interpreted as a response to muscle fiber damage, with the inflammatory cytokines believed to orchestrate tissue repair. It is now well recognized that skeletal muscle secretes some of the same immunomodulatory factors as part of a more complex role in many physiological processes such as adaptation and metabolism (Pedersen & Febbraio, 2008). Since inflammation appears to play a prominent role in many diseases and exercise has a profound effect on the incidence of these conditions, this review will examine the effect of exercise and nutritional status on inflammation.

The Acute Inflammatory Response

Before examining how exercise influences inflammation, it is necessary to briefly outline how inflammation orchestrates its effects. Acute inflammatory episodes occur in response to many biologically harmful stresses such as trauma, exposure to pathogens, and tissue damage. The response is initiated by receptors of the toll-like receptor (TLR) family, which are critical in host defense, by distinguishing between particles of infection and those produced by normal tissue turnover (Iwasaki & Medzhitov, 2004). TLR activation leads to intracellular signaling via the adaptor protein MyD88, which results in translocation of the transcription factor NF-κB to the nucleus to stimulate production of pro-inflammatory cytokines, namely tumor necrosis factor (TNF)-α, interleukin (IL)-1, IL-6, and IL-8 (Figure 38.1). These are small, secreted signaling

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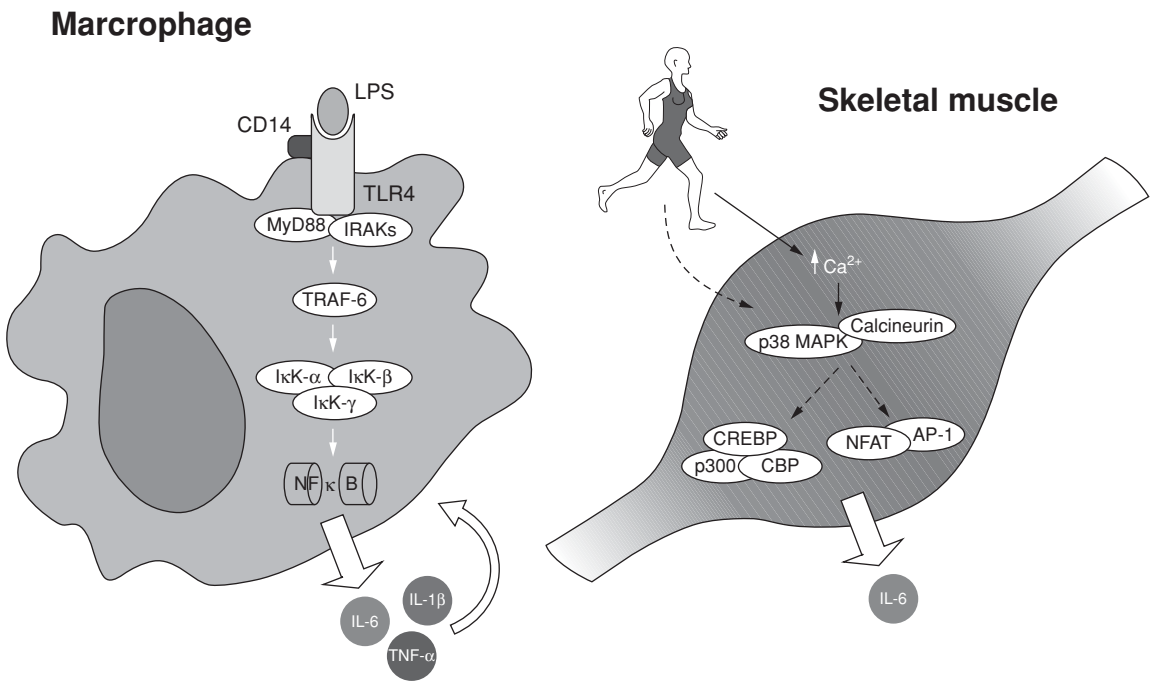

Figure 38.1 Differential signaling pathways responsible for the production of IL-6. Both the stimulus and signaling pathways responsible for IL-6 production differ between cell types. In immune cells, cytokines are produced in response to classical inflammatory signals, whereas in contracting skeletal muscle, myokine production is dependent on changes in intracellular components. Cluster of Differentiation 14, CD14; IL-1 Receptor Associated Kinase, IRAK; TNF Receptor Associated Factor, TRAF; Adenosine 3′5′ Cyclic Monophosphate Response Element Binding Protein, CREBP; E1A binding protein p300, p300; CREB Binding Protein, CBP. Reprinted with permission from Pedersen & Febbraio (2008).

molecules capable of activating the acute phase response—a release of complex immunomodulatory proteins from the liver—and orchestrating the movement and activation of immune cells. In normal physiological conditions, cytokines circulate at almost undetectable concentrations, but during times of stress or infection, cytokines increase in circulation three- to fourfold before decreasing when the stress is resolved. In healthy humans, the inflammatory response constitutes a normal physiological reaction and is not typically associated with adverse outcomes to the host. However, individuals with persistent two- to fourfold elevations in circulating inflammatory cytokines such as TNF-α and C-reactive protein (CRP) have, by definition, systemic low-grade inflammation (Bruunsgaard, 2005). The imbalance in inflammatory mediators observed in chronic low-grade inflammation causes adverse effects in peripheral tissues that are associated with the pathology of many chronic diseases.

Chronic Low-Grade Inflammation and Disease

In people with chronic low-grade inflammation, a controversy exists as to whether the persistence of elevated pro-inflammatory cytokines actively contributes to disease progression or merely reflects existing underlying pathological processes. Nevertheless, strong evidence now links chronic low-grade inflammation with the etiology of diseases of the metabolic syndrome, including insulin resistance, type 2 diabetes, cardiovascular disease, atherosclerosis, and fatty liver disease, and is strongly associated with aging and lifestyle factors such as smoking, obesity, dietary patterns, cognitive decline, and cachexia.

Importantly, chronic low-grade inflammation is an independent and consistent predictor of all-cause mortality (Bruunsgaard, 2005).

Global rates of obesity and related metabolic diseases are increasing at alarming rates. In particular, physical inactivity without a concomitant reduction in energy consumption is strongly associated with increased adiposity. Recently it was reported that 2 weeks of reduced stepping in healthy men (from ~10,000 to 1500 steps per day) was sufficient to impair glucose tolerance and lipid metabolism and to increase abdominal fat mass (Olsen et al., 2008). Research over the last decade has begun to decipher how enhanced adiposity contributes to chronic low-grade inflammation.

Adipose Tissue Biology—Dysfunction with Visceral Fat Gain

The discovery that white adipose tissue is an active endocrine organ revolutionized the way we view this organ and provided empirical evidence for a relationship between enhanced adiposity and dysregulated metabolism. White adipose tissue is now known to secrete "adipokines," which play an important role in regulating both energy homeostasis and inflammation in local and peripheral tissues. Importantly, the circulating adipokine profile is significantly different between lean and obese individuals. In obesity, the circulating concentrations of pro-inflammatory adipokines (leptin, monocyte chemo-attractant protein (MCP)-1, TNF-α, IL-6, retinol-binding protein 4 (RBP-4), and IL-18) are increased, whereas the anti-inflammatory adipokine, adiponectin, is reduced (Ouchi et al., 2011).

Current evidence suggests that white adipose tissue-derived TNF-α directly mediates the pathological processes that link adiposity with insulin resistance and other symptoms of the metabolic syndrome. In humans, circulating TNF-α is positively correlated with adiposity and is reduced with weight loss. Whole-body TNF-α deficiency, or inhibition with an anti-TNF-α antibody, renders protection to insulin resistance in dietary models of obesity in rodents (for review, see Nieto-Vazquez et al., 2008). Mechanistically, TNF-α activates several intracellular pathways such as c-Jun N-terminal kinase (JNK) and I kappa beta kinase (IκK) that link inflammation and metabolism (De Luca & Olefsky, 2008). The activation of these pathways by TNF-α ultimately impairs the ability of insulin to stimulate the translocation of glucose transporter 4 (GLUT4) to the cell membrane and thus reduces glucose uptake into cells (Hotamisligil, 2006). In addition to adipocytes, white adipose tissue also contains several other cell types and as adipocytes enlarge, certain adipokines, including MCP-1, directly recruit immune cells into the fat tissue, which can produce additional pro-inflammatory cytokines. The exact role of adipokines in the development of chronic inflammation is not completely understood, but it appears that the altered biology of the fat tissue, which occurs as a result of nutrient excess, contributes to a number of disease conditions.

Reducing Chronic Low-Grade Inflammation by Regular Exercise

Regular physical activity reduces the risk of developing many chronic, noncommunicable diseases. Physical activity is negatively correlated with chronic low-grade inflammation, although a lack of an association has also been reported (Bruunsgaard, 2005). Despite this, the molecular mechanisms accountable for the protective effects of exercise are not fully known. One way by which exercise may reduce the risk of, or be effective in the treatment of, chronic disease is by reducing chronic low-grade inflammation. Several mechanisms may contribute to a reduction in chronic low-grade inflammation by exercise, including the reduction of visceral adipose tissue mass, downregulation of TLR4 expression, and the establishment of an anti-inflammatory environment with each exercise bout.

Reduction of Visceral Fat Mass

Regular physical activity is known to promote a healthy body composition and epidemiological studies have linked vigorous physical activity with a lower waist to hip ratio indicative of reduced centrally stored fat (Ross & Bradshaw, 2009). Intriguingly, despite exercise interventions consistently reducing visceral adiposity, a concomitant

reduction in body mass index (BMI) is not always observed, suggesting an alteration in somatotype. By reducing visceral fat mass through increased energy expenditure, the pro-inflammatory profile in this tissue should be reduced with a concomitant increase in the anti-inflammatory profile. Indeed, regular exercise tends to lower basal levels of pro-inflammatory cytokines in elderly and sedentary individuals and in people with a range chronic illnesses (Bruunsgaard, 2005). Conversely, the role of exercise in the regulation of adiponectin levels remains controversial (for review, see Simpson & Singh, 2008). Adiponectin has insulin-sensitizing and anti-inflammatory properties and a negative association exists between visceral fat mass and adiponectin (Okamoto et al., 2006). This association is likely due to the local negative regulation of adiponectin by TNF-α and IL-6 (Pederson & Febbraio, 2008). Thus, elevated levels of adiponectin after training due to reduced visceral fat mass may be secondary to a reduction in local concentrations of TNF-α and IL-6. Together, these data suggest that a reduction in visceral fat mass may improve several aspects of the adipokine profile and thus improve chronic low-grade inflammation. However, the molecular mechanisms underlying reduced adipocyte size and adipokine production are not fully elucidated and remain a hot area of research.

Exercise-Induced Reduction of TLR4 Expression

Activation of TLRs in circulating immune cells results in the production of pro-inflammatory cytokines. Elevated serum and tissue-free fatty acids are commonly observed in obese people as reviewed elsewhere (Lee & Hwang, 2006) and a growing body of literature implicates fatty acid-induced TLR4 signaling with the elevated production of pro-inflammatory cytokines. Both acute and regular exercise can modulate TLR4 expression (for review, see Gleeson et al., 2006). In response to a single bout of prolonged aerobic exercise, expression of TLR4 is temporarily reduced in circulating monocytes immediately after and 2 hours postexercise (Lancaster et al., 2005). Trained elderly individuals have lower basal expression of TLR4 compared with sedentary elderly individuals (Flynn et al., 2003), and regular exercise training lowers TLR4 expression in untrained people (Stewart et al., 2005). Together, these data suggest that TLR4 expression is reduced by exercise, which may contribute to the reduction of chronic low-grade inflammation by attenuating the release of inflammatory cytokines by circulating immune cells.

Anti-inflammatory Environment Induced by Exercise

Exercise induces a robust elevation in plasma cytokines that is significantly different from the cytokine response observed during times of severe stress. In response to sepsis, TNF-α and IL1β are produced locally and stimulate the production of IL-6, IL1 receptor alpha (IL1ra), soluble TNF-α receptor (sTNFR), and IL-10 (Pedersen & Febbraio, 2008). TNF-α and IL1β are potent pro-inflammatory cytokines produced locally, and they stimulate the production of IL-6, which can, in turn, elicit both pro- and anti-inflammatory effects. Importantly, IL1ra and sTNFR have anti-inflammatory effects by antagonizing the actions of their ligands IL1β and TNF-α, respectively (Dinarello, 2000; Lantz et al., 1990). In contrast, during exercise, IL-6 is typically the first cytokine to appear in circulation and precedes the appearance of IL1ra, IL-10, and sTNFR (Pedersen & Febbraio, 2008). In addition to anti-inflammatory cytokine production, exercise also stimulates the hypothalamic–pituitary–adrenal (HPA) axis and the sympathetic nervous system to produce hormones such as adrenaline, glucocorticoids, growth hormone, and prolactin. These hormones have many critical effects during exercise, some of which may influence the cytokine response to exercise and although not discussed in this chapter, the reader is directed to several informative reviews—Barnes (1998) and Duclos et al. (2007). Together, these data suggest that exercise creates an anti-inflammatory environment characterized by the production of anti-inflammatory cytokines and inhibition of the production of pro-inflammatory cytokines.

One component of the anti-inflammatory environment invoked by exercise is the production and secretion of IL-6 by the skeletal muscle. Skeletal

muscle is known to produce several proteins, termed myokines, in response to exercise, and these appear to be functionally important in modulating the beneficial effects of exercise on systemic low-grade inflammation. How muscle-derived IL-6 and other myokines contribute to the health benefits of exercise will form the remaining section of this chapter.

Interleukin-6—The Prototypical Myokine

Of the cytokines that appear in the circulation in response to exercise, IL-6 is the most robustly increased (Pedersen & Febbraio, 2008). Plasma IL-6 increases exponentially during exercise and may increase by up to 100-fold before rapidly declining in the postexercise period (Fischer, 2006). The type of muscle contraction influences the kinetics of IL-6 appearance in the plasma. Prolonged exercise causes a peak in IL-6 at the cessation of exercise, whereas the peak in IL-6 levels occurs later in recovery following eccentric exercise. It was hypothesized that skeletal muscle damage was the stimulus for the IL-6 response based on the observation that peak IL-6 levels were associated with creatine kinase (CK) levels that are indicative of muscle damage. However, subsequent studies proved eccentric exercise was not associated with higher plasma IL-6 than exercise involving concentric "non-damaging" contractions (Hirose et al., 2004).

About 50% of the variability in the relative change of plasma IL-6 induced by exercise can be explained by exercise duration (Fischer, 2006). However, a combination of related factors such as exercise intensity and mode and both nutritional and training status also influence the IL-6 response to exercise (Pedersen & Febbraio, 2008). The IL-6 response appears sensitive to the intensity of the exercise (Ostrowski et al., 2000) with a twofold increase in plasma IL-6 occurring in response to 6 minutes of maximal ergometer rowing (Nielsen et al., 1996). In contrast, low-intensity exercise can be sustained for longer periods of time and the IL-6 peak occurs later (Pedersen & Febbraio, 2008). Although there have been few controlled comparisons between exercise disciplines, running tends to invoke the highest increase in plasma IL-6. This suggests that the size of the muscle mass recruited is an important determinant of the IL-6 response to exercise. As contracting skeletal muscle is the source of IL-6 and running utilizes large muscle groups, this observation is not surprising. Exercise involving smaller muscle groups may not be sufficient to result in an elevation of plasma IL-6 (Pedersen & Febbraio, 2008). However, recently it was observed that IL-6 release was higher across arm muscles than with the leg during 90 minutes of combined arm and leg exercise at 60% VO_2 max (Helge et al., 2011), suggesting that factors other than the size of muscle mass recruited determine the magnitude of the IL-6 response to exercise. These studies prove without doubt that exercise elevates plasma IL-6 independent of muscle damage, but the initial studies did not identify the cellular source of IL-6.

Source of Exercise-Induced IL-6

At the turn of the millennium, circulating immune cells were thought to produce the IL-6 response to exercise, as these cells were responsible for the increase in plasma IL-6 in response to sepsis. However, exercise did not increase IL-6 mRNA in blood mononuclear (immune) cells or the production of IL-6 by circulating monocytes (Pedersen & Febbraio, 2008). In a pioneering study by Steensberg et al. (2000), a net release of IL-6 from contracting skeletal muscle was demonstrated by calculating the IL-6 difference in arterial and venous blood. The prolonged contraction of a single limb (but not the contralateral resting limb) released IL-6 into circulation, which accounted for most of the exercise-induced elevation in plasma IL-6 (Steensberg et al., 2000). The expression of IL-6 mRNA in skeletal muscle biopsies is detectable after just 30 minutes of exercise compared with pre-exercise where IL-6 mRNA is almost undetectable (Keller et al., 2001). Many cell types make up a tissue bed, but evidence that myocytes *per se* express and release IL-6 in response to exercise has been confirmed in subsequent studies using immunohistochemistry and sensitive *in situ* hybridization techniques (Hiscock et al., 2004).

Although the myocytes themselves are likely to produce the majority of the IL-6 induced by exercise,

tissues such as the Achilles tendon (Langberg et al., 2002) and brain (Nybo et al., 2002) may also contribute to elevations in circulating IL-6 observed during exercise. The adipose tissue contributes significantly to circulating IL-6 at rest (Mohamed-Ali et al., 1997), but during exercise, IL-6 release from the abdominal subcutaneous tissue bed does not increase despite an elevated IL-6 mRNA expression. After exercise, the net release of IL-6 from the adipose tissue becomes quantitatively significant, suggesting a role for adipose-derived IL-6 in the recovery period (Lyngsø et al., 2002). Together, these data suggest that the adipose tissue does not contribute to the exercise-induced increase in circulating IL-6.

In summary, IL-6 is produced in large amounts in response to exercise: most of this comes from the skeletal muscle cells with small contributions from other tissues. The net release of IL-6 from an exercising limb becomes significantly different after 2 hours, suggesting that the IL-6 response to exercise is related less to inflammation and regeneration (recovery/healing) and more to changes in substrate availability (Steensberg et al., 2000).

Influence of Muscle Glycogen and Carbohydrate Ingestion

Glucose is the preferred fuel utilized by exercising muscles during prolonged exercise and a number of studies have confirmed that carbohydrate (CHO) ingestion during exercise attenuates exercise-induced elevations in plasma IL-6 (Pedersen & Febbraio, 2008). Despite CHO supplementation attenuating the exercise-induced increase of IL-6 in plasma, the expression of IL-6 mRNA within contracting skeletal muscle is unaffected, suggesting that IL-6 release from contracting skeletal muscle is regulated by substrate availability. Given that prolonged exercise results in depletion of muscle glycogen, IL-6 gene expression and IL-6 release may be modulated by glycogen depletion. In support of this, intramuscular IL-6 mRNA expression and protein release are enhanced when intramuscular glycogen stores are depleted either before or during exercise (Pedersen & Febbraio, 2008). Based on these studies, it was hypothesized that IL-6 released from contracting skeletal muscle may act as an endocrine signal to the liver and adipose tissues to mobilize substrates for uptake into the skeletal muscle to prolong exercise capacity and/or to fuel cellular repair during recovery from exercise.

Training Status and IL-6 Sensitivity

A negative association between levels of physical activity and basal plasma IL-6 has been reported in several epidemiological studies. However, in healthy people, regular exercise typically does not alter basal IL-6 levels. It is more likely that exercise training and level of conditioning play an important role in determining the IL-6 response to exercise. In elderly subjects performing knee extension exercise at 50% maximal power, the net IL-6 release across the working leg was similar to that seen in young participants despite performing less than half the absolute power (Pedersen et al., 2004). When healthy men performed 10 weeks of knee extensor exercise, the expression of the IL-6 mRNA during an acute exercise bout was reduced from 76-fold before training to eightfold after training. This reduction occurred despite their absolute workload increasing by 44% and a similar increase in plasma IL-6 during exercise bouts before and after training (Fischer et al., 2004).

In contrast to a reduction in exercise-induced mRNA expression of IL-6, both an acute exercise bout and exercise training appear to upregulate the expression of the IL-6 receptor (IL-6R) (Pedersen & Febbraio, 2008). Unlike IL-6 expression and release from the skeletal muscle, which is sensitive to glycogen status, the expression of the IL-6R is not enhanced when intramuscular glycogen levels are reduced (Keller et al., 2005). Interestingly, CHO consumption during training attenuates the increase in basal IL-6R expression in skeletal muscle. Participants performed 10 weeks of single leg, knee extension exercise. One leg was trained while consuming a CHO drink while the other leg was training on alternate days while consuming a placebo drink. While 10 weeks of training reduced the IL-6 response to exercise by ~56%, CHO ingestion did not attenuate this effect. Training increased IL-6R protein expression in skeletal muscle, but this increase was attenuated in the leg trained while consuming CHO (Akerstrom et al., 2009b). The

biological consequence of this adaptation remains unknown as performance, substrate utilization, and glycogen content did not differ between the leg trained with CHO or placebo ingestion (Akerstrom et al., 2009a). These data suggest that an adaptation of the skeletal muscle to reduced IL-6 with training is an upregulation of the IL-6R. This adaptation, which maintains the sensitivity of the skeletal muscle to IL-6, is attenuated when CHO is consumed.

Signaling Requirements for Muscle-Derived IL-6

The signaling pathways responsible for the contraction-induced production of IL-6 in skeletal muscle differ from those when IL-6 is produced in response to a wide range of stimuli, including inflammatory cytokines, reactive oxygen species, and lipopolysaccharide (LPS). The transcription of IL-6 in the skeletal muscle is rapidly induced at the onset of exercise. The specific pathways mediating muscle IL-6 transcription have not been well characterized, but factors induced by exercise, such as nitric oxide, stimulate IL-6 expression, independent of classic NF-κB inflammatory signaling (Pedersen & Febbraio, 2008). Muscle contraction is a potent stimulus for Ca^{2+} release from the sarcoplasmic reticulum and repeated muscle contraction results in elevated intracellular concentrations of Ca^{2+}. Fluctuations in intracellular Ca^{2+} can activate a number of transcription regulators, and a role for the calcium-sensitive phosphatase, calcineurin, in IL-6 transcription has been postulated by Holmes et al. (2004a). Interestingly, an exacerbation of IL-6 mRNA expression has been shown in muscles with low glycogen concentrations, an effect perhaps mediated by the stress-activated kinase p38 Mitogen-Activated Protein Kinase (MAPK) (Chan et al., 2004). Several important transcription factors are activated by p38 including activating transcription factor (ATF)-2 and Elk1. Intriguingly, ATF-2 is a subunit of the AP-1 heterodimer (jun:ATF) and Elk1 is a member of the Ets superfamily of transcription factors, which have binding sites within the IL-6 promoter region (Pedersen & Febbraio, 2008). In summary, mechanisms controlling intramuscular IL-6 gene transcription in response to contraction are different from those under pro-inflammatory circumstances and are likely to involve a cross talk between the Ca^{2+}/Nuclear Factor of Activated T-cells (NFAT) and MAPK pathways (Figure 38.1).

Role of IL-6 in Mediating the Anti-inflammatory Environment of Exercise

Exercise results in the elevated circulation of anti-inflammatory cytokines preceded by the myokine IL-6. It has been hypothesized that the cytokine profile observed during exercise can be modulated by IL-6 (Pedersen, 2011). Seldom are changes in TNF-α and IL1β observed with non-damaging exercise, most likely due to the negative regulation of TNF-α and IL1β production by IL-6. IL-6 inhibits LPS-stimulated TNF-α production in cultured human monocytes and elevated levels of TNF-α have been observed in IL-6-deficient mice, suggesting an involvement of IL-6 in TNF-α production (Pedersen & Febbraio, 2008). In addition, the infusion of rhIL-6 into humans increases plasma levels of the anti-inflammatory markers IL1ra, IL-10, cortisol, and sTNFR (Pedersen, 2011). Together, these data suggest that the infusion of IL-6 could recapitulate the anti-inflammatory cytokine cascade observed during exercise. Importantly, IL-10 can also elicit anti-inflammatory effects by inhibiting the production of IL1β and TNF-α in LPS-stimulated human monocytes (Pedersen & Febbraio, 2008). To evaluate whether exercise *per se* could induce an actual anti-inflammatory effect, healthy volunteers were injected with a low dose of *Escherichia coli* endotoxin either following exercise or at rest with or without rhIL-6 infusion. The administration of endotoxin induced an elevation in plasma TNF-α at rest, which was blunted with prior exercise and rhIL-6 infusion (Starkie et al., 2003). Collectively, these data suggest that an acute bout of exercise produces an anti-inflammatory cytokine cascade that elicits an anti-inflammatory effect *in vivo*. Despite this, it remains to be proven if the anti-inflammatory environment produced by acute exercise protects from diseases associated with chronic low-grade inflammation. In addition to the effects on the inflammatory environment, a large body of research has focused on the direct effects of IL-6 on metabolism in health and disease.

Role of IL-6 in Metabolism

For almost half a century, researchers in the exercise metabolism field had hypothesized that skeletal muscle possessed a "humoral" factor secreted in response to exercise, to mediate metabolic effects in peripheral tissues such as liver and adipose tissue (Goldstein, 1961). Muscle-derived IL-6 is a plausible candidate for this "humoral" exercise factor (Pedersen & Febbraio, 2008). In an editorial published in *The Journal of Physiology*, Gleeson (2000) hypothesized that muscle-derived IL-6 acts in an endocrine manner, signaling the liver to mobilize glucose to maintain euglycemia in response to depletion of muscle glycogen and an increasing reliance on blood-borne substrates. A number of studies have indeed implicated muscle-derived IL-6 in energy homeostasis during exercise.

IL-6 and Carbohydrate Metabolism

Although IL-6 has been implicated in liver glucose production during exercise, this appeared dependent on a contraction-induced cofactor (Febbraio et al., 2004). The release of IL-6 is positively related to exercise intensity and muscle glucose uptake (Helge et al., 2003), suggesting that IL-6 may enhance glucose uptake into working muscle. Indeed, when IL-6 was infused during low-intensity exercise, peripheral glucose uptake was higher than when low-intensity exercise was performed without IL-6 infusion. These effects occurred without differences in the hormonal milieu, suggesting that IL-6 can influence muscle glucose metabolism during exercise (Febbraio et al., 2004). In resting healthy humans, infusion of IL-6 at physiological levels either increases (Carey et al., 2006) or has no effect on whole-body glucose uptake when measured during a euglycemic-hyperinsulinemic clamp (Pedersen & Febbraio, 2008). Adenosine monophosphate–activated protein kinase (AMPK) is the key cellular energy sensor and regulator of glucose and lipid metabolism and appears important in mediating the metabolic effects of IL-6 (Ruderman et al., 2006). Myotubes infected with an AMPK-dominant negative adenovirus are unable to induce glucose uptake in response to IL-6 (Carey et al., 2006) and mice deficient in IL-6 have altered AMPK activity in response to exercise (Kelly et al., 2004). Together, these data highlight how muscle-derived IL-6 may enhance whole-body glucose uptake, and that this is likely to be mediated by the actions of AMPK.

IL-6 and Fatty Acid Metabolism

More consistently, IL-6 appears to be important in fatty acid metabolism where it can stimulate lipolysis and fatty acid oxidation (Pedersen & Febbraio, 2008). The infusion of rhIL-6 into humans stimulates the turnover of fatty acids (Petersen et al., 2005) and the release of free fatty acids (Wolsk et al., 2010), which become available for oxidation (Van Hall et al., 2003). Furthermore, the pharmacological inhibition of lipolysis resulted in elevated levels of IL-6 in healthy men at rest and during exercise (Holmes et al., 2004b). More recently, IL-6 was reported to enhance whole-body fatty acid metabolism and increase arterial palmitate concentration via the stimulation of lipolysis in the skeletal muscle but not in the adipose tissue (Wolsk et al., 2010). Together, these studies suggest IL-6 can act as a lipolytic factor and enhance fatty acid oxidation in peripheral tissues. However, the contribution of IL-6 to the lipolytic effects observed during exercise remains debatable. Infusion of IL-6 into exercising men did not alter fatty acid flux or metabolism, suggesting that additional factors mediate the exercise-induced alterations in fat metabolism (Hiscock et al., 2005).

It has not been experimentally tested, but exercise-derived IL-6 may act to reduce visceral fat mass. By virtue of a lipolytic factor in the peripheral tissues, enhanced IL-6 during exercise may promote lipolysis in the visceral fat tissue to provide fatty acids for oxidation to fuel muscle contraction and/or subsequent recovery. By reducing visceral fat mass, this may reduce the inflammation in the visceral fat tissue and protect from chronic low-grade inflammation.

Role of IL-6 in Low-Grade Inflammatory Diseases: Focus on Insulin Resistance

Obesity is characterized by systemic low-grade inflammation and elevated levels of IL-6 are

correlated strongly with adiposity. Furthermore, IL-6 is not elevated in lean insulin-resistant people or associated with insulin resistance when obesity is accounted for (Pedersen & Febbraio, 2008). These observations led to the hypothesis that IL-6 was responsible for obesity-related insulin resistance and early studies reported IL-6 to induce whole-body insulin resistance in mice (Kim et al., 2004). It would seem illogical from a physiological point of view, and considering the current literature, that contracting skeletal muscle would secrete a protein that impairs insulin signaling at a time when insulin action is enhanced. Several studies have now been performed in humans that confound these earlier findings. The infusion of recombinant human IL-6 into healthy humans does not impair glucose uptake and may enhance insulin sensitivity in patients with type 2 diabetes (Petersen et al., 2005). Together, these data suggest that IL-6 may have beneficial effects on insulin sensitivity and highlight the potential limitations of using rodent models to investigate human metabolism. Further evidence for a beneficial role of IL-6 in metabolism comes from clinical trials of patients with inflammatory diseases treated with an anti-IL-6 receptor antibody. Recently, the U.S. Food and Drug Administration (FDA) has approved the submission for ACTEMRA® (tocilizumab), an anti-IL-6R antibody, for the treatment of rheumatoid arthritis and multicentric Castleman's disease (Febbraio et al., 2011). Patients treated with ACTEMRA responded with an elevation in non-fasting total cholesterol and triglycerides, suggesting that blockade of IL-6 signaling may promote insulin resistance and an atherogenic lipid profile (Febbraio et al., 2011). In line with these observations, IL-6-deficient mice develop mature-onset obesity and glucose intolerance (Wallenius et al., 2002). It must be noted that the obese IL-6-deficient phenotype has not been observed in another independently generated strain (Di Gregorio et al., 2004). Recently, the global deletion of IL-6 or a liver-specific deletion of the IL-6 receptor was reported to result in reduced glucose tolerance and steatosis and inflammation in the liver (Matthews et al., 2011; Wunderlich et al., 2011). Together, these data suggest that IL-6 has a beneficial role in lipid homeostasis and protects against diet-induced inflammation in the liver.

As IL-6 promotes both lipolysis and fat oxidation in humans, whereas TNF-α induces lipolysis without inducing fat oxidation, one theory is that elevated TNF-α is the "culprit" behind obesity-induced insulin resistance, whereas elevated IL-6 observed in states of chronic low-grade inflammation represents local production of TNF-α and may act as a defense mechanism to blunt excessive TNF-α signaling in the adipose tissue (Pedersen & Febbraio, 2008). Therefore, exercise-derived IL-6 may operate in an important feedback loop between the skeletal muscle and adipose tissue to provide protection from the deleterious effects of adipose-derived TNF-α observed during physical inactivity and/or gains in visceral adiposity.

Other Myokines

IL-6 is the most studied myokine to date, but several others such as IL-8, IL-15, brain-derived neurotrophic factor (BDNF), and leukemia inhibitory factor (LIF) have also been discovered and potentially play a role in the response to exercise (Pedersen, 2011).

Interleukin-8

IL-8 is a chemotactic cytokine that appears in the circulation only after exhaustive exercise, such as running, with smaller changes observed after intense cycling. IL-8 mRNA and protein are significantly elevated in muscle biopsies and within skeletal muscle fibers during and after exercise, respectively, suggesting that skeletal muscle *per se* produces IL-8. An inflammatory role for IL-8 has been proposed where eccentric exercise has been performed, whereas little or no difference in plasma IL-8 is observed after concentric exercise despite a small net release of IL-8 being measured. A more likely role for IL-8 is to stimulate angiogenesis. The high local, but small systemic changes observed in IL-8 in response to exercise suggests that IL-8 functions locally to stimulate angiogenesis, possibly via C-X-C motif Receptor 2

(CXCR2) receptor signaling (Pedersen & Febbraio, 2008). Whether IL-8 contributes to an anti-inflammatory role of exercise is yet to be established experimentally.

Interleukin-15

IL-15 is a cytokine with anabolic effects on skeletal muscle (Quinn et al., 2002). In response to prolonged aerobic exercise, no changes in skeletal muscle IL-15 mRNA expression or plasma levels have been observed (Pedersen & Febbraio, 2008). The IL-15 response to resistance exercise has provided inconsistent responses. An acute bout of resistance exercise has been reported to elevate plasma IL-15, whereas skeletal muscle IL-15 mRNA expression has been observed to increase, or not change in response to the same (Pedersen & Febbraio, 2008).

More consistent evidence suggests that IL-15 may have an important role in lipid metabolism, where it is involved in muscle–adipose tissue cross talk. When IL-15 is administered to rats for a week, white adipose tissue mass was reduced by 33% (Carbo et al., 2001). Furthermore, the oversecretion of IL-15 from skeletal muscle decreased visceral but not subcutaneous fat mass in male mice, suggesting that IL-15 may be involved in the regulation of visceral fat stores. Neither the changes in immune parameters were measured nor the initial state of the mice associated with any pathogenic state, but the reduction in visceral fat mass suggests that a reduction in this fat store could reduce the local inflammation in this tissue and contribute to the anti-inflammatory effect of exercise. In summary, although IL-15 may reduce white adipose tissue in mice, a definitive effect of exercise-induced production of IL-15 is yet to be proven. Furthermore, a reduction in chronic low-grade inflammation as a result of reduced visceral adiposity by exercise-induced IL-15 is yet to be experimentally tested.

The Search for Additional Myokines

The list of myokines is expanding and recently follistatin-like 1 and fibroblast growth factor 21 have been identified as proteins secreted from the skeletal muscle (Walsh, 2009). Myokine discovery has previously been restricted by conventional molecular methodologies, which limit the investigative scope to only a few proteins. Powerful large-scale proteomic studies using mass spectrometry are now being conducted to characterize the skeletal muscle secretome. Recently ~600 potential myokine candidates were identified during myoblast differentiation (Henningsen et al., 2010), indicating that much is still unknown about the endocrine function of the skeletal muscle. Further studies will be required to unravel the role of these gene products and to identify target tissues and how (if?) these factors are influenced by exercise and whether they contribute to the anti-inflammatory effects of exercise.

Conclusions

Inflammatory mechanisms are now known to contribute directly to the development and progression of many chronic diseases as well as the aging process. Symptomatic of these diseases is the persistence of a state of chronic low-grade inflammation. The exact biological mechanism for the development of this low-grade inflammation is not known, but several pathways that link inflammatory signaling to metabolism have been identified and appear sensitive to both nutrient excess and physical inactivity.

Physical activity is an energy-demanding process and adaptations to regular exercise enhance glucose homeostasis, energy metabolism, and weight control. In response to contraction, the skeletal muscle produces and releases myokines that have immune and metabolic effects at a local and systemic level.

Exercise induces an anti-inflammatory hormonal milieu that contributes to the beneficial effects of exercise and chronic disease. As part of this hormonal milieu, myokines are becoming established as important mediators of an anti-inflammatory effect of exercise. Research is ongoing to elucidate further myokine candidates and to establish the role of these novel myokines in metabolism, inflammation, and disease prevention.

References

Akerstrom, T.C., Fischer, C.P., Plomgaard, P., et al. (2009a) Glucose ingestion during endurance training does not alter adaptation. *Journal of Applied Physiology* 106, 1771–1779.

Akerstrom, T.C., Krogh-Madsen, R., Petersen, A.M., et al. (2009b) Glucose ingestion during endurance training in men attenuates expression of myokine receptor. *Experimental Physiology* 94, 1124–1131.

Barnes, P.J. (1998) Anti-inflammatory actions of glucocorticoids: molecular mechanisms. *Clinical Science (London)* 94, 557–572.

Bruunsgaard, H. (2005) Physical activity and modulation of systemic low-level inflammation. *Journal of Leukocyte Biology* 78, 819–835.

Carbo, N., Lopez-Soriano, J., Costelli, P., et al. (2001) Interleukin-15 mediates reciprocal regulation of adipose and muscle mass: a potential role in body weight control. *Biochimica et Biophysica Acta* 1526, 17–24.

Carey, A.L., Steinberg, G.R., Macaulay, S.L., et al. (2006) Interleukin-6 increases insulin-stimulated glucose disposal in humans and glucose uptake and fatty acid oxidation in vitro via AMP-activated protein kinase. *Diabetes* 55, 2688–2697.

De Luca, C. & Olefsky, J.M. (2008) Inflammation and insulin resistance. *Federation of European Biochemical Societies—Letters* 582, 97–105.

Di Gregorio, G.B., Hensley, L., Lu, T., et al. (2004) Lipid and carbohydrate metabolism in mice with a targeted mutation in the IL-6 gene: absence of development of age-related obesity. *American Journal of Physiology—Endocrinology and Metabolism* 287, E182–E187.

Dinarello, C.A. (2000) The role of the interleukin-1-receptor antagonist in blocking inflammation mediated by interleukin-1. *New England Journal of Medicine* 343, 732–734.

Duclos, M., Guinot, M., & Le Bouc, Y. (2007) Cortisol and GH: odd and controversial ideas. *Applied Physiology, Nutrition, and Metabolism* 32, 895–903.

Febbraio, M.A., Hiscock, N., Sacchetti, M., et al. (2004) Interleukin-6 is a novel factor mediating glucose homeostasis during skeletal muscle contraction. *Diabetes* 53, 1643–1648.

Febbraio, M.A., Rose-John, S., & Pedersen, B. K. (2011) Is interleukin-6 receptor blockade the Holy Grail for inflammatory diseases? *Clinical Pharmacology and Therapeutics* 87, 396–398.

Fischer, C.P. (2006) Interleukin-6 in acute exercise and training: what is the biological relevance? *Exercise Immunology Review* 12, 6–33.

Fischer, C.P., Plomgaard, P., Hansen, A.K., et al. (2004) Endurance training reduces the contraction-induced interleukin-6 mRNA expression in human skeletal muscle. *American Journal of Physiology—Endocrinology and Metabolism* 287, E1189–E1194.

Flynn, M.G., Mcfarlin, B.K., Phillips, M.D., et al. (2003) Toll-like receptor 4 and CD14 mRNA expression are lower in resistive exercise-trained elderly women. *Journal of Applied Physiology* 95, 1833–1842.

Gleeson, M. (2000) Interleukins and exercise. *Journal of Physiology* 529(1), 1.

Gleeson, M., Mcfarlin, B., & Flynn, M. (2006) Exercise and toll-like receptors. *Exercise Immunology Review* 12, 34–53.

Goldstein, M.S. (1961) Humoral nature of the hypoglycemic factor of muscular work. *Diabetes* 10, 232–234.

Helge, J.W., Stallknecht, B., Pedersen, B.K., et al. (2003) The effect of graded exercise on IL-6 release and glucose uptake in human skeletal muscle. *Journal of Physiology* 546, 299–305.

Helge, J.W., Klein, D.K., Andersen, T.M., et al. (2011) Interleukin-6 release is higher across arm than leg muscles during whole-body exercise. *Experimental Physiology* 96, 590–598.

Henningsen, J., Rigbolt, K.T., Blagoev, B., et al. (2010) Dynamics of the skeletal muscle secretome during myoblast differentiation. *Molecular and Cellular Proteomics* 9, 2482–2496.

Hirose, L., Nosaka, K., Newton, M., et al. (2004) Changes in inflammatory mediators following eccentric exercise of the elbow flexors. *Exercise Immunology Review* 10, 75–90.

Hiscock, N., Chan, M.H., Bisucci, T., et al. (2004) Skeletal myocytes are a source of interleukin-6 mRNA expression and protein release during contraction: evidence of fiber type specificity. *Federation of American Societies for Experimental Biology* 18, 992–994.

Hiscock, N., Fischer, C.P., Sacchetti, M., et al. (2005) Recombinant human interleukin-6 infusion during low-intensity exercise does not enhance whole body lipolysis or fat oxidation in humans. *American Journal of Physiology—Endocrinology and Metabolism* 289, E2–E7.

Holmes, A.G., Watt, M.J., Carey, A.L., et al. (2004a) Ionomycin, but not physiologic doses of epinephrine, stimulates skeletal muscle interleukin-6 mRNA expression and protein release. *Metabolism* 53, 1492–1495.

Holmes, A.G., Watt, M.J., & Febbraio, M.A. (2004b) Suppressing lipolysis increases interleukin-6 at rest and during prolonged moderate-intensity exercise in humans. *Journal of Applied Physiology* 97, 689–696.

Hotamisligil, G.S. (2006) Inflammation and metabolic disorders. *Nature* 444, 860–867.

Iwasaki, A. & Medzhitov, R. (2004) Toll-like receptor control of the adaptive immune responses. *Nature Immunology* 5, 987–995.

Keller, C., Steensberg, A., Pilegaard, H., et al. (2001) Transcriptional activation of the IL-6 gene in human contracting skeletal muscle: influence of muscle glycogen content. *Federation of American Societies for Experimental Biology* 15, 2748–2750.

Keller, C., Steensberg, A., Hansen, A.K., et al. (2005) Effect of exercise, training, and glycogen availability on IL-6 receptor expression in human skeletal muscle. *Journal of Applied Physiology* 99, 2075–2079.

Kelly, M., Keller, C., Avilucea, P.R., et al. (2004) AMPK activity is diminished in tissues of IL-6 knockout mice: the effect of exercise. *Biochemical and Biophysical Research Communications* 320, 449–454.

Khandekah, M.J., Cohen, P., & Spiegelman, B.M. (2011) Molecular mechanisms of cancer development in obesity. *Nature Reviews Cancer* 11, 886–895.

Kim, H.J., Higashimori, T., Park, S.Y., et al. (2004) Differential effects of interleukin-6 and -10 on skeletal muscle and liver insulin action in vivo. *Diabetes* 53, 1060–1067.

Lancaster, G.I., Khan, Q., Drysdale, P., et al. (2005) The physiological regulation of toll-like receptor expression and function in humans. *Journal of Physiology* 563, 945–955.

Langberg, H., Olesen, J.L., Gemmer, C., et al. (2002) Substantial elevation of interleukin-6 concentration in peritendinous tissue, in contrast to muscle, following prolonged exercise in humans. *Journal of Physiology* 542, 985–990.

Lantz, M., Gullberg, U., Nilsson, E., et al. (1990) Characterization in vitro of a human tumor necrosis factor-binding

protein. A soluble form of a tumor necrosis factor receptor. *Journal of Clinical Investigation* 86, 1396–1402.

Lee, J.Y. & Hwang, D.H. (2006) The modulation of inflammatory gene expression by lipids: mediation through toll-like receptors. *Molecules and Cells* 21, 174–185.

Lyngsø, D., Simonsen, L., & Bulow, J. (2002) Interleukin-6 production in human subcutaneous abdominal adipose tissue: the effect of exercise. *Journal of Physiology* 543, 373–378.

Matthews, V.B., Allen, T.L., Risis, S., et al. (2011) Interleukin-6-deficient mice develop hepatic inflammation and systemic insulin resistance. *Diabetologia* 53, 2431–2441.

Mohamed-Ali, V., Goodrick, S., Rawesh, A., et al. (1997) Subcutaneous adipose tissue releases interleukin-6, but not tumor necrosis factor-alpha, in vivo. *Journal of Clinical Endocrinology and Metabolism* 82, 4196–4200.

Nielsen, H.B., Secher, N.H., Christensen, N.J., et al. (1996) Lymphocytes and NK cell activity during repeated bouts of maximal exercise. *American Journal of Physiology* 271, R222–R227.

Nieto-Vazquez, I., Fernandez-Veledo, S., Kramer, D. K., et al. (2008) Insulin resistance associated to obesity: the link TNF-alpha. *Archives of Physiology and Biochemistry* 114, 183–194.

Nybo, L., Nielsen, B., Pedersen, B.K., et al. (2002) Interleukin-6 release from the human brain during prolonged exercise. *Journal of Physiology* 542, 991–995.

Okamoto, Y., Kihara, S., Funahashi, T., et al. (2006) Adiponectin: a key adipocytokine in metabolic syndrome. *Clinical Science (London)* 110, 267–278.

Olsen, R.H., Krogh-Madsen, R., Thomsen, C., et al. (2008) Metabolic responses to reduced daily steps in healthy nonexercising men. *Journal of the American Medical Association* 299, 1261–1263.

Ostrowski, K., Schjerling, P., & Pedersen, B.K. (2000) Physical activity and plasma interleukin-6 in humans—effect of intensity of exercise. *European Journal of Applied Physiology* 83, 512–515.

Ouchi, N., Parker, J.L., Lugus, J.J., et al. (2011) Adipokines in inflammation and metabolic disease. *Nature Reviews Immunology* 11, 85–97.

Pedersen, B.K. (2011) Muscles and their myokines. *Journal of Experimental Biology* 214, 337–346.

Pedersen, B.K. & Febbraio, M.A. (2008) Muscle as an endocrine organ: focus on muscle-derived interleukin-6. *Physiological Reviews* 88, 1379–1406.

Pedersen, M., Steensberg, A., Keller, C., et al. (2004) Does the aging skeletal muscle maintain its endocrine function? *Exercise Immunology Review* 10, 42–55.

Petersen, E.W., Carey, A.L., Sacchetti, M., et al. (2005) Acute IL-6 treatment increases fatty acid turnover in elderly humans in vivo and in tissue culture in vitro. *American Journal of Physiology—Endocrinology and Metabolism* 288, E155–E162.

Quinn, L.S., Anderson, B.G., Drivdahl, R.H., et al. (2002) Overexpression of interleukin-15 induces skeletal muscle hypertrophy in vitro: implications for treatment of muscle wasting disorders. *Experimental Cell Research* 280, 55–63.

Rock, K.L., Latz, E., Ontiveros, F., et al. (2010) The sterile inflammatory response. *Annual Review Immunology* 28, 321–342.

Ross, R. & Bradshaw, A.J. (2009) The future of obesity reduction: beyond weight loss. *Nature Reviews Endocrinology* 5, 319–325.

Ruderman, N.B., Keller, C., Richard, A.M., et al. (2006) Interleukin-6 regulation of AMP-activated protein kinase. Potential role in the systemic response to exercise and prevention of the metabolic syndrome. *Diabetes* 55(Suppl. 2), S48–S54.

Simpson, K.A. & Singh, M.A. (2008) Effects of exercise on adiponectin: a systematic review. *Obesity (Silver Spring)* 16, 241–256.

Starkie, R., Ostrowski, S.R., Jauffred, S., et al. (2003) Exercise and IL-6 infusion inhibit endotoxin-induced TNF-alpha production in humans. *Federation of American Societies for Experimental Biology* 17, 884–886.

Steensberg, A., Van Hall, G., Osada, T., et al. (2000) Production of interleukin-6 in contracting human skeletal muscles can account for the exercise-induced increase in plasma interleukin-6. *Journal of Physiology* 529(1), 237–242.

Stewart, L.K., Flynn, M.G., Campbell, W.W., et al. (2005) Influence of exercise training and age on CD14+ cell-surface expression of toll-like receptor 2 and 4. *Brain, Behaviour, and Immunity* 19, 389–397.

Van Hall, G., Steensberg, A., Sacchetti, M., et al. (2003) Interleukin-6 stimulates lipolysis and fat oxidation in humans. *Journal of Clinical Endocrinology and Metabolism* 88, 3005–3010.

Wallenius, V., Wallenius, K., Ahren, B., et al. (2002) Interleukin-6-deficient mice develop mature-onset obesity. *Nature Medicine* 8, 75–79.

Walsh, K. (2009) Adipokines, myokines and cardiovascular disease. *Circulation Journal* 73, 13–18.

Wolsk, E., Mygind, H., Grondahl, T. S., et al. (2010) IL-6 selectively stimulates fat metabolism in human skeletal muscle. *American Journal of Physiology—Endocrinology and Metabolism* 299, E832–E840.

Wunderlich, F.T., Strohle, P., Konner, A.C., et al. (2011) Interleukin-6 signaling in liver-parenchymal cells suppresses hepatic inflammation and improves systemic insulin action. *Cell Metabolism* 12, 237–249.

Chapter 39

Exercise, Nutrition, and Immune Function

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Introduction

Prolonged and intensive exercise has transient but significant and wide-ranging effects on the immune system (Gleeson, 2007; Nieman, 1997). The exercise-induced immune perturbations and associated physiologic stress are associated with an elevated risk of upper respiratory tract infections (URTIs), especially during the 1- to 2-week period following competitive endurance races (Nieman, 2000, 2007, 2009).

Immunonutrition support for athletes is an active area of research endeavour, and this chapter will summarize the efficacy of various nutritional products in countering exercise-induced immune dysfunction, oxidative stress, and inflammation (Gleeson et al., 2004; Nieman, 2008; Walsh et al., 2011). In contrast, near-daily moderate physical activity is associated with a reduced URTI risk, and favorable acute immune changes, and falls outside the context of this chapter (Nieman et al., 2011).

The value of using immunonutrition support for athletes has been questioned because blocking the transient oxidative stress, inflammation, and elevations in stress hormones following heavy exertion potentially interferes with important signaling mechanisms for training adaptations (Gomez-Cabrera, et al., 2008; Ristow et al., 2009). Another viewpoint is that even the most effective immunonutrition support systems only partially block exercise-induced physiologic stress indicators, analogous to the beneficial use of ice packs to reduce swelling following mild injuries (Sureda et al., 2008; Yfanti et al., 2010). In the end, the value of immunonutrition support (still to be determined) for athletes during periods of heavy exertion and competitive races will be evaluated by whether or not the athlete has improved recovery, lowered URTI, reduced muscle damage and soreness, and enhanced overall athletic performance.

Exercise-Induced Immune Dysfunction and Infection Risk

Each acute bout of heavy exertion leads to alterations in immunity and host pathogen defense, and elevations in stress hormones, pro- and anti-inflammatory cytokines, and reactive oxygen species (ROS) (Gleeson, 2007; Nieman, 1997). Natural killer cell activity, various measures of T and B cell function, upper airway neutrophil function, salivary IgA concentration, granulocyte oxidative burst activity, skin delayed-type hypersensitivity response, and major histocompatibility complex (MHC) II expression in macrophages are suppressed for several hours to days during recovery from prolonged, intense endurance exercise. These immune changes occur in several compartments of the immune system and body including the skin, upper respiratory tract mucosal tissue, lung, blood, muscle, and peritoneal cavity. Many mechanisms appear to be involved including exercise-induced changes in stress hormone and sympathetic nervous system stimulation, body temperature changes, increases in blood flow, dehydration, muscle damage, oxidative stress, and

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the use of nonsteroidal anti-inflammatory drugs including ibuprofen (Nieman, 2007, 2009).

Intensive and sustained exercise can create an imbalance between ROS and antioxidant defenses, increasing oxidative stress. F2-isoprostanes are one of the best oxidative stress biomarkers (McAnulty et al., 2011). Inflammation also rises, and typical measures include C-reactive protein (CRP) and a variety of cytokines and chemokines including interleukin (IL)-6, IL-10, IL-8, IL-1ra, granulocyte colony-stimulating factor (G-CSF), monocyte chemotactic protein 1 (MCP-1), macrophage inflammatory protein-1 beta (MIP-1β), macrophage migration inhibitory factor 1 (MIF-1), and tumor necrosis factor alpha (TNF-α) (Nieman, 1997, 2009). The longer and more intense the exercise bout, the greater and more prolonged the inflammation response, with the highest levels measured in athletes with the greatest muscle damage after ultramarathons (Nieman, 2007, 2009; Nieman et al., 2012). Figure 39.1 summarizes plasma IL-6 levels following a variety of exercise workloads.

Despite the large but transient acute changes in oxidative stress, inflammation, and immune function, chronic, resting immunity of athletes varies little from that of nonathletes (Nieman et al., 1995). Natural killer cell activity may be elevated in some types of athletes while neutrophil function is slightly suppressed. The adaptive immune system is largely unaffected by athletic endeavour. In general, the acute immune changes during and after each exercise bout appear to better explain URTI risk in athletes than the small alterations in chronic, resting immunity.

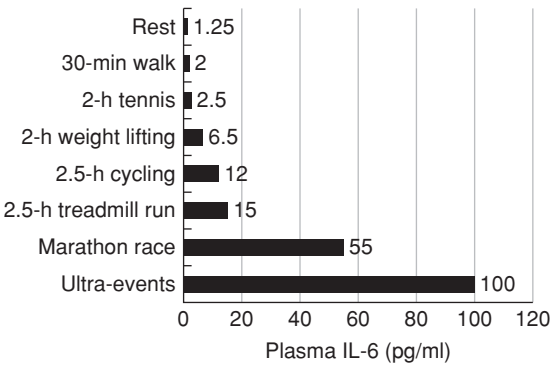


Figure 39.1 Plasma IL-6 response to varying exercise workloads. Data taken from a wide range of studies from Nieman's lab.

During the "open window" of impaired immunity (which may last between 3 and 72 hours, depending on the immune measure), pathogen resistance is lowered, increasing the risk of subclinical and clinical infection (Gleeson, 2007; Murphy et al., 2009; Nieman, 2000). Epidemiological studies indicate that athletes engaging in marathon and ultramarathon race events and/or very heavy training are at increased risk of URTI (Nieman, 2000; Nieman et al., 1990; Peters et al., 1993). Nearly 13% of marathoners reported URTI during the week following the Los Angeles Marathon race compared with 2.2% of control marathon runners (odds ratio, 5.9) (Nieman et al., 1990). In total, 40% of the runners reported at least one URTI episode during the 2-month winter period prior to the marathon race. Controlling for various confounders, it was determined that runners training more than 96 km/week doubled their odds for sickness compared with those training less than 32 km/week.

Similar results have been reported by other investigators in both humans and rodents (Davis et al., 2004; Murphy et al., 2009; Scherr et al., 2011). One in four athletes reported URTI during the 2-week period following the 160-km Western States Endurance Run (WSER), and this was linked to low post-race salivary IgA output levels but not the variation in change of inflammatory factors such as plasma cytokines (Nieman, 2009). A 1-year retrospective study of 852 German athletes showed that URTI risk was highest in endurance athletes who also reported significant stress and sleep deprivation (Konig et al., 2000). Thus, URTI risk may be exceptionally high when an athlete goes through repeated cycles of unusually heavy exertion, has been exposed to novel pathogens, and has experienced other stressors to the immune system including lack of sleep, severe mental stress, malnutrition, or weight loss.

Immunonutrition Support for Athletes

Various nutritional agents have been tested for their capacity to attenuate immune changes, oxidative stress, and inflammation following intensive

Table 39.1 Summary of rationale and findings for selected immunonutrition supplements

Immunonutrition supplement	Rationale	Recommendation based on current evidence
Carbohydrate	Maintains blood glucose during exercise, lowers release of cortisol and epinephrine, and thus counters negative immune changes postexercise	Recommended; up to 60 g/h of heavy exertion helps dampen increases in leukocyte subset counts, and cytokines, but does not counter changes in T and natural killer (NK) cell function
Fruit and vegetable extracts rich in polyphenols and flavonoids (e.g., green tea extract, black currant and blueberry extract)	Act as ibuprofen substitutes by attenuating exercise-induced inflammation; also decrease oxidative stress	Recommended, but most research focused on countermeasure effects for oxidative stress and inflammation; more research needed on immune function
Quercetin (aglycone and glucoside forms such as isoquercetin)	*In vitro* studies show strong anti-inflammatory, antioxidative, and antipathogenic effects. Animal data indicate increase in mitochondrial biogenesis and endurance performance, reduction in mortality following pathogen exposure	Recommended when mixed with other flavonoids and nutrients; human studies show reduction in illness rates during heavy training and a small, 3% influence on performance
Bovine colostrums	Mix of immune, growth, and hormonal factors improve immune function and the neuroendocrine system, and lower illness risk	Mixed results, and more data needed
Probiotics	Improve intestinal microbial flora, and thereby enhance gut and systemic immune function	Mixed results, and more data needed
β-Glucan	Receptors found on intestinal wall immune cells interact with β-glucan as it passes through, improving systemic innate immunity and thereby reducing infection rates	Mixed results: Oat β-glucan was not effective in one human study; mushroom β-glucan may be more effective, but more data needed
Vitamin E	Quenches exercise-induced reactive oxygen species (ROS) and augments immunity	Not recommended; may be pro-oxidative and pro-inflammatory with heavy exertion
Vitamin C	Quenches ROS and augments immunity	Not recommended; not consistently different from placebo
Multiple vitamins and minerals	Work together to quench ROS and reduce inflammation	Not recommended; not different from placebo; balanced diet is sufficient
Glutamine	Important immune cell energy substrate that is lowered with prolonged exercise	Not recommended; body stores exceed exercise-lowering effects
N-3 PUFAs (fish oil)	Exerts anti-inflammatory and immune-regulatory effects postexercise	Not recommended; no different from placebo
Herbal supplements (e.g., Ginseng, *Echinacea*)	Contain bioactive molecules that augment immunity and counter infection	Not recommended; human studies do not show consistent support within an athletic context

exercise (Gleeson et al., 2004; Nieman, 2008, 2010; Walsh et al., 2011). The list of efficacious immunonutrition support practices and products for athletes is small, but increasing attention is being directed to this field of scientific endeavour (see Table 39.1) (Walsh et al., 2011). Results for most nutritional supplements tested as countermeasures to exercise-induced inflammation, oxidative stress, and immune dysfunction following heavy exertion have been disappointing, but some, such as carbohydrate and fruit/vegetable extracts, have impressive results.

Carbohydrate

A series of studies dating back to the mid-1990s showed that ingestion of carbohydrate supplements before and/or during prolonged, intensive exercise (e.g., about 30–60 g carbohydrate/hour) attenuated increases in blood neutrophil and monocyte counts, stress hormones, and anti-inflammatory cytokines such as IL-6, IL-10, and IL-1ra (Figure 39.2) (Chen et al., 2008; Davison & Gleeson, 2005; Gleeson, 2007; Nieman, 1998; Nieman et al., 2006). At the same time, however, little effect of carbohydrate ingestion was measured for exercise-induced decrements in salivary IgA output, and T cell and natural killer cell function. Thus, carbohydrate ingestion during heavy exercise emerged as an effective but partial countermeasure to immune dysfunction. Little is known regarding whether or not URTI rates are decreased when athletes remain carbohydrate-fed during endurance races. One study of marathon runners showed that illness rates following a competitive marathon tended to be lower in athletes ingesting carbohydrate compared with placebo beverages during the race (Nieman et al., 2001).

Carbohydrate may exert these effects through multiple mechanisms including an elevation in blood glucose and tissue glucose uptake leading to a diminished stress hormone output, decreased

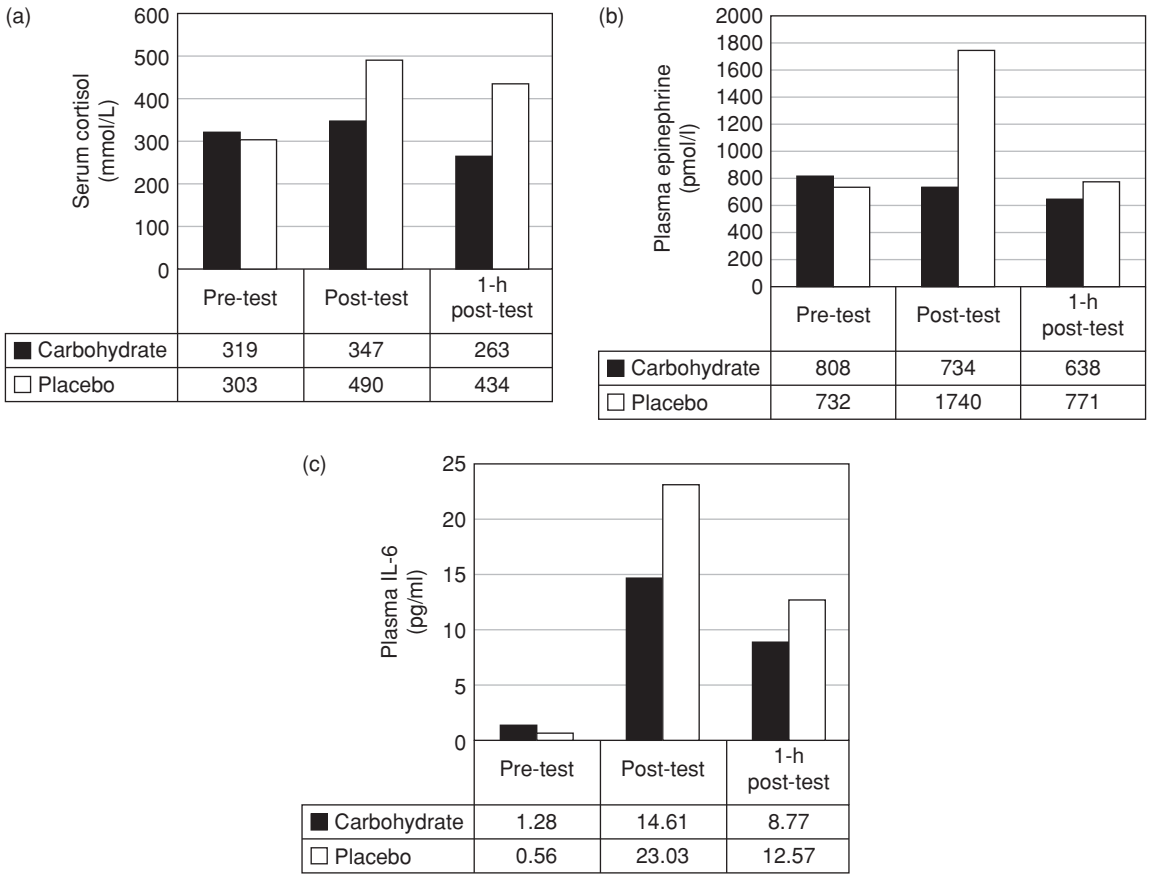


Figure 39.2 Comparison of (a) cortisol, (b) epinephrine, and (c) IL-6 responses to 2 hour exercise in cyclists consuming carbohydrate or placebo beverages (1 liter/h, 6% carbohydrate). Interaction effects, all $p < 0.05$. Data obtained from Nieman et al. (2006).

cytokine mRNA expression, reduced pro-inflammatory signals, and attenuated IL-6 release from the working muscle tissue (Gleeson, 2007; Nieman, 2008). A reduction in blood glucose levels during intense and prolonged exertion when athletes drink plain water increases hypothalamic–pituitary–adrenal activation, leading to a release of adrenocorticotropic hormone and cortisol, growth hormone, and epinephrine. Stress hormones have an intimate link with genes that control cytokine production, and multiple cell types of the immune system.

Antioxidants

Multiple studies have focused on large dose antioxidant supplements, and no consistent countermeasure benefit has been measured (Davison & Gleeson, 2005, 2006; Gleeson et al., 2004; Nieman et al., 2002, 2004). Heavy exertion causes oxidative stress, lipid peroxidation, and protein oxidation (McAnulty et al., 2011). Exercise-induced oxidative stress and immune dysfunction may be linked, but data support is largely lacking (Nieman, 2009). The proposed benefits of antioxidant supplementation in attenuating oxidative stress and immune dysfunction during exercise remain unsubstantiated (Nieman, 2008, 2009).

For example, most well-designed studies do not support that vitamin C supplementation modulates immune responses following heavy exertion (Davison & Gleeson, 2005, 2006; Nieman et al., 2002). Studies of South African ultramarathon runners demonstrated that vitamin C (but not E or β-carotene) supplementation (about 600 mg/day for 3 weeks) was related to fewer reports of URTI symptoms, but this has not been a consistent finding (Peters et al., 1993).

Most studies also indicate that vitamin E supplementation is not an effective strategy for countering the inflammatory, oxidative stress, and immune response to intensive and prolonged exercise. Two months of vitamin E supplementation at a dose of 800 IU/day α-tocopherol did not counter increases in plasma cytokines, perturbations in other measures of immunity, or oxidative stress in triathletes competing in the Kona Triathlon World Championship race event (Nieman et al., 2004). Triathletes in the vitamin E compared with the placebo group actually experienced greater lipid peroxidation and increases in plasma levels of several cytokines following the race event (see Figure 39.3).

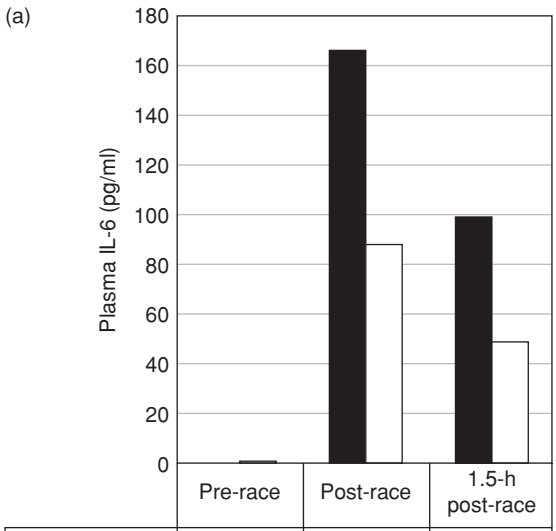
In general, antioxidant supplementation for athletes during heavy exertion cannot be recommended based on current evidence. The majority of investigations have failed to show that ingestion of antioxidants such as vitamins E and C has meaningful effects on exercise-induced inflammation, muscle damage, increases in plasma cytokines, and immune perturbations. Large dose vitamin E supplementation can actually exacerbate oxidative stress and inflammation during prolonged exercise in the heat.

Glutamine

Glutamine supplements are not recommended because the best studies show no benefits when compared with placebo, perhaps due to abundant storage pools within the body that cannot be sufficiently depleted by exercise (Gleeson, 2007, 2008; Krzywkowski et al., 2001).

Glutamine is an important fuel for lymphocytes and monocytes as supported by *in vitro* experiments showing that stepwise depletion of glutamine has a direct effect in lowering proliferation rates of T and B lymphocytes. Glutamine is an important component of currently available enteral immune-modulating formulas for patients who are critically ill or have experienced trauma or surgery.

Reduced plasma glutamine levels have been observed in response to intense and prolonged exertion, and exercise immunologists have tested the value of glutamine ingestion as a countermeasure to exercise-induced immune dysfunction (Krzywkowski et al., 2001). The majority of studies, however, do not support that exercise-induced reductions in plasma glutamine levels cause impaired immunity and diminished host protection against viruses in athletes. For example, in a crossover, placebo-controlled study of eight males,



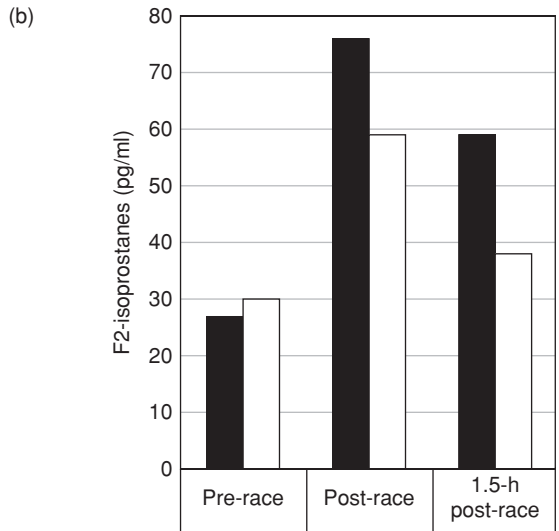


Figure 39.3 (a) Plasma IL-6 and (b) F2-isoprostane responses in elite triathletes ingesting vitamin E or placebo (800 IU/day for 2 months) prior to the Kona Ironman World Championship (interaction effects, all $p < 0.06$). Data obtained from Nieman et al. (2004).

glutamine supplementation abolished the postexercise decrease in plasma glutamine concentration but still had no influence relative to placebo on exercise-induced decreases in T and natural killer cell function (Rohde et al., 1998).

One problem with the glutamine hypothesis is that plasma concentrations following exercise do not decrease below threshold levels that are detrimental to lymphocyte function as demonstrated by *in vitro* experiments. In other words, even marathon-type exertion does not deplete the body of glutamine to a degree sufficient to diminish lymphocyte function.

β-Glucan

The growing realization that extra vitamins, minerals, and glutamine do not provide countermeasure benefits for healthy and well-fed athletes during heavy training has shifted the focus to other types of nutritional components. β-glucans are polysaccharides found in the bran of oat and barley cereal grains, the cell wall of baker's yeast, certain types of fungi, and many kinds of mushrooms. Rodent studies indicate that oat β-glucan supplements offset the increased risk of infection associated with exercise stress through augmentation of macrophage and neutrophil function, but these results were not upheld in a study of human cyclists (Murphy et al., 2009; Nieman et al., 2008). Recent studies with athletes suggest that mushroom β-glucan may exert beneficial effects (Bergendiova et al., 2011).

A wide variety of β-glucans exist that vary in macromolecular structure, solubility, molecular weight, and biological activity. Most β-glucans from yeast and fungi consist of d-glucose linked in the β-(1→3) position with β-(1→6) linkage with glucose side branches. Oat and barley β-glucans have a mixed β-(1→3) and β-(1→4) linkage. Humans lack small intestine enzymes to separate the glucose molecules of β-glucans, and they pass to the large intestine undigested. Oat and barley β-glucan is a fermentable, viscous fiber that decreases low-density lipoprotein

(LDL) cholesterol by partially blocking enterohepatic recirculation of cholesterol and bile acids. Consumption of oat and barley products with at least 3 g of β-glucan soluble fiber per day is effective in lowering blood total and LDL cholesterol.

Receptors of β-glucans have been identified on a wide variety of cell types including macrophages, dendritic cells, natural killer cells, neutrophils, some types of T cells, epithelial cells, vascular endothelial cells, and fibroblasts (Murphy et al., 2009, 2010). Thus, despite the lack of enzymatic breakdown in the small intestine, the widespread distribution of β-glucan receptors throughout the body suggests some degree or a unique method of absorption. Studies with rodents indicate that the bioavailability of β-glucans is about 4–5%, and that soluble glucans can translocate from the gastrointestinal (GI) tract to the systemic circulation. Although the exact pathway is still undetermined, current evidence suggests that β-glucans interact with a variety of GI cells including mucosal dendritic and epithelial cells. GI mucosal dendritic cells may sample or interact with soluble β-glucans via projections across the epithelium and then migrate via afferent lymphatics to the mesenteric lymph nodes where immune modulation is initiated. GI macrophages may also engulf β-glucans, shuttle them to reticuloendothelial tissues and the bone marrow, and then degrade and secrete small β-glucan fragments that bind to receptors on bone marrow granulocytes. A subpopulation of intestinal epithelial cells and gut-associated lymphoid tissue (GALT) cells appear to be capable of actively binding and internalizing β-glucans, which then leads to small but significant increase in blood β-glucan levels.

Growing evidence from studies conducted with rodents, fish, poultry, and swine indicates that β-glucan ingestion stimulates innate immune defenses and antitumor responses, and increases resistance to a wide variety of infections (Murphy et al., 2009, 2010). β-Glucans may activate macrophages and neutrophils directly, stimulating their phagocytic, cytotoxic, and antimicrobial activities through several types of cellular receptors including Dectin-1, lactosylceramide, toll-like receptors 2 and 6, scavenger receptors, and the type-3 complement receptor (CR3).

Rodent studies indicate that oat β-glucan supplements offset the increased risk of infection associated with exercise stress through augmentation of macrophage and neutrophil function (Davis et al., 2004; Murphy et al., 2009, 2010). In one study with mice, ingestion of oat β-glucan for 10 days before intranasal inoculation of herpes simplex virus-1 (HSV-1) countered the increase in morbidity and mortality, and the decrease in macrophage antiviral resistance, following exhaustive 140-minute exercise bouts over 3 consecutive days (Davis et al., 2004). In a follow-up study, these investigators showed that 10 days of oat β-glucan by mice blocked the increased susceptibility to morbidity and mortality following HSV-1 inoculation and three consecutive days of running to volitional fatigue on rodent treadmills (Murphy et al., 2009). Depletion of lung macrophages using clodronate negated the beneficial effects of β-glucan, indicating that these immune cells are at least partially involved.

A similar study with human athletes, however, failed to confirm these results (Nieman et al., 2008). Trained male cyclists were randomized to β-glucan or placebo groups and under double-blind procedures received oat β-glucan (5.6 g/day) or placebo for 2 weeks prior to and during a 3-day period in which they cycled for 3 h/day at high intensity. URTI symptoms were monitored for 2 weeks before and 2 weeks after the 3-day period of intensified exercise. Blood samples were taken before and after 14 days of β-glucan supplementation (chronic immunity) and immediately after the last bout of exercise and 14-hour of recovery (acute immunity), and were assayed for a wide variety of immune function measures including natural killer and T cells, granulocytes, and plasma cytokine levels. None of these immune measures differed between β-glucan and placebo groups, and URTI incidence did not differ during the 31 days of monitoring. These data indicate that oat β-glucan supplementation does not alter chronic immune function or acute exercise-induced immune perturbations during a period of intensified exercise training.

A recent 3-month supplementation study with 50 athletes indicated that oyster mushroom β-glucan reduced URTI incidence and had a favorable effect on phagocytosis relative to placebo (Bergendiova

et al., 2011). Although mechanisms were not explored in this study, the β-(1,3/1,6)-d-glucan from the *Pleurotus ostreatus* (oyster mushroom) appears to travel through the digestive system and stimulate GALT immune cells with surface-specific receptors Dectin-1 and CR3 (and is more effective than β-(1,3/1,4)-d-glucan).

Other Advanced Supplements

In vitro/cell culture, animal, and epidemiological research indicate that advanced supplements such as probiotics, bovine colostrum, flavonoids and polyphenols such as quercetin, resveratrol, curcumin, and epigallicatechin-3-gallate (EGCG), N-3 polyunsaturated fatty acids (N-3 PUFAs or fish oil), herbal supplements, and unique plant extracts (e.g., green tea extract, blackcurrant extract, tomato extract with lycopene, anthocyanin-rich extract from bilberry, polyphenol-rich pomegranate fruit extract), have potential benefits for athletes (Bakker et al., 2010; Lyall et al., 2009; Nieman, 2010; Nieman et al., 2009b, 2010a).

Human research testing efficacy in athletes has just begun for many types of advanced nutritional supplements. Limited data from athletes are nonsupportive or mixed for use of N-3 PUFAs (Nieman et al., 2009b), probiotics (West et al., 2009), bovine colostrums (Carol et al., 2011; Davison & Diment, 2010; Shing et al., 2009), ginseng (Senchina et al., 2009), or *Echinacea* (Senchina et al., 2009) (see Table 39.1 for a summary).

N-3 PUFAs, for example, decrease the production of inflammatory eicosanoids, cytokines, and ROS, and have immunomodulatory activities. Some investigators promote that the majority of athletes need 1–2 g marine n-3 PUFAs per day to counter excessive oxygen radical formation, inflammation, and trauma from high-intensity exercise and the high n-6 PUFA levels of the Western diet. However, a double-blinded, placebo controlled study with trained cyclists demonstrated that 6 weeks supplementation with 2.4 g/day eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) did not alter the measures of inflammation and immunity before and after 3 days of prolonged and intensive exercise (Nieman et al., 2009b).

Bovine colostrum includes immune, growth, and antimicrobial factors, and supplementation theoretically may promote exercise performance and help maintain immune function during intense exercise (Shing et al., 2009). Studies are mixed, however, and the optimum dosing paradigm is still being explored. Carol et al. (2011) studied subjects consuming 25 g/day freeze-dried bovine colostrum for 10 days, but found no influence relative to skimmed milk powder placebo on exercise-induced immune changes.

An evolving hypothesis is that the immune system is so diverse that a mixture of these advanced supplements within a carbohydrate beverage will probably perform better than one supplement by itself (Bakker et al., 2010; Nieman et al., 2009a). In other words, the "pharma" approach of using large doses of a single nutritional component is not as effective as a "cocktail" strategy for nutritional supplements.

A secondary hypothesis is that the primary immune target of nutrient supplements should be the nonspecific, innate arm of the immune system to enhance immunosurveillance against a wide variety of pathogens in athletes. If the nutritional supplement improves the natural killer cell, macrophage, and granulocyte function before and/or after heavy exertion, then the risk of infection is more effectively countered than when the target is the slower moving adaptive immune components (Nieman, 2008; Nieman et al., 2007, 2009a).

Flavonoids

Phytochemicals are chemicals produced by plants and include tannins, lignins, and flavonoids. The largest and best-studied polyphenols are the flavonoids, with more than 6000 identified and classified into at least 6 subgroups: flavonols, flavones, flavanones, flavanols (and their oligomers, proanthocyanidins), anthocyanidins, and isoflavonoids. Flavonoids are widely distributed in plants and function as plant pigments, signaling molecules, and defenders against infection and injury.

Many flavonoids possess strong anti-inflammatory, antiviral, antioxidant, antiobesity, and anticarcinogenic properties when studied *in vitro* using large doses of the purified form. Inflammation and oxidative stress are key mechanisms in the pathogenesis of

certain disease states, supporting the proposed strategy of increased flavonoid intake for the prevention of cancer, diabetes mellitus, and cardiovascular disease. However, results from randomized, double-blinded studies in humans with large doses of purified flavonoids such as quercetin have been disappointing (Nieman, 2010). Flavonoids vary widely in bioavailability, and most are poorly absorbed, undergo active efflux, and are extensively conjugated and metabolically transformed, all of which can affect their bioactive capacities (Harwood et al., 2007). Despite low bioavailability of the parent flavonoid, some of the *in vivo* metabolites may accumulate in tissues and produce bioactive influences, but conclusive human data are lacking. For example, animal data indicate that quercetin metabolites accumulate in the vascular tissue where they act as complementary antioxidants, with plasma albumin facilitating the translocation of quercetin metabolites to the vascular target.

There is a growing realization that bioactive influences of individual flavonoids are potentiated when mixed with other flavonoids (e.g., the flavonol quercetin with the flavanol EGCG) or included in a cocktail or extract of other polyphenols and nutrients (Lila, 2007). Two or more flavonoids ingested together may increase bioavailability and decrease elimination via competitive inhibition of glucuronide and sulfate conjugation in both the intestine and liver, and by inhibiting efflux transporters such as P-glycoprotein, breast cancer resistance protein (BCRP), and multidrug resistance protein 2 (MRP2).

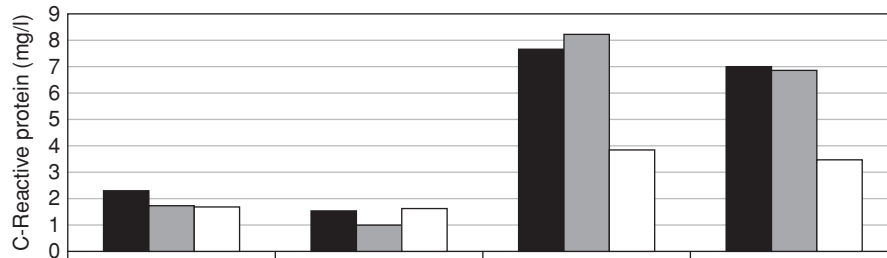
The health-protective effects of plant foods are not produced by a single component but rather complex mixtures of interacting molecules. The polyphenols and natural components provide a multifaceted defensive strategy for both plants and humans. Thus the "pharma" approach of using large doses of a single bioactive molecule is seldom successful in the application of nutrition to human health and performance. Additionally, a metabolomics or nutrigenomics approach is needed to improve the capacity of investigators to capture the complex and subtle influences of flavonoid supplements or flavonoid-rich extracts, foods, and beverages on whole-body metabolism and physiology (Bakker et al., 2010).

The physiologic effects of dietary polyphenols such as quercetin, EGCG, curcumin, lycopene, resveratrol, luteolin, and tiliroside are of great current interest to exercise immunologists due to their antioxidative, anti-inflammatory, antipathogenic, cardioprotective, anticarcinogenic, and mitochondrial stimulatory activities (Nieman, 2008, 2010).

Flavonoids such as quercetin, EGCG, and isoflavones, or flavonoid-rich plant extracts are being tested by an increasing number of investigative teams as performance aids and countermeasures to exercise-induced inflammation, delayed onset of muscle soreness (DOMS), oxidative stress, immune dysfunction, and URTI (Nieman et al., 2010a). Most studies have focused on the ability of flavonoid-rich tea, fruit, and vegetable extracts to counter oxidative stress, and the majority indicates an effective response. The second most common outcome measure is related to inflammation and DOMS, and again, most studies support protective effects when flavonoid mixtures or plant extracts are ingested before demanding bouts of exercise. Results are mixed for performance outcomes, and few studies have included immune and URTI measures.

For any particular flavonoid or plant extract studied within an exercise context, few papers are available, and research designs vary widely in regard to the supplementation dose and regimen, the mode of exercise stress, and outcome measures. The flavonoid supplementation period varies from 15 minutes to 60 days prior to an exercise challenge, with most studies clustered between 7 and 21 days. Nonetheless, the data in general support that flavonoid-rich plant extracts and unique flavonoid–nutrient mixtures (e.g., quercetin with green tea extract and fish oil, or isoflavones with lycopene) help counter exercise-induced oxidative stress and inflammation/DOMS.

More exercise-related research has been conducted with quercetin than any other flavonoid (Konrad et al., 2011; Nieman, 2010; Nieman et al., 2007, 2009, 2010a, 2010b). In one of the earliest studies with exercise-stressed cyclists, supplementation with pure quercetin (1000 mg/day) over a 5-week period reduced illness rates but did not counter postexercise inflammation, oxidative stress, or immune dysfunction (Nieman et al., 2007). In a follow-up study using a similar design, quercetin supplementation combined with green tea extract, isoquercetin,



	Pre-suppl. (8 am)	Post-suppl. (8 am)	Post-3 day exercise (6 pm)	14-h recovery (8 am)
■ Placebo (N = 12)	2.25	1.49	7.64	6.97
▒ Quercetin (N = 13)	1.7	0.96	8.2	6.82
□ Quercetin-mixture (N = 14)	1.64	1.61	3.83	3.46

Figure 39.4 C-reactive protein before and after 2 weeks supplementation with a mixture of quercetin, isoquercetin, EGCG-enriched green tea extract, fish oil, vitamin C, and niacin, and then immediately after and 14 hours after 3 days of intensified exercise (3 h/day, 57% Wattsmax). Data obtained from Nieman et al. (2009a).

and fish oil did cause a sizeable reduction in exercise-induced inflammation and oxidative stress, with chronic augmentation of innate immune function (Nieman et al., 2009a) (see Figure 39.4). Quercetin's role as a performance aid has been tested by several research teams with mixed results, but a recent meta-analysis indicates a small but significant 3% performance enhancement (Kessler et al., 2011; Nieman et al., 2010b). Animal studies support a role for quercetin as an exercise mimetic for mitochondrial biogenesis, and one study with untrained human subjects indicated a modest enhancement in skeletal muscle mitochondrial density and endurance performance, but far below what was reported in mice (Nieman et al., 2010b).

Summary and Future Research

Athletes must train hard for competition and are interested in strategies to keep their immune systems robust and illness rates low despite the physiologic stress they experience. The ultimate goal is to provide athletes with a sports drink or supplement bar containing carbohydrate and a cocktail of advanced supplements that will lower infection risk, exert significant and measurable influences on their innate immune systems, and attenuate exercise-induced oxidative stress and inflammation. The athlete can combine this strategy with other approaches that help maintain immunity and health.

Carbohydrate beverage supplementation (~60 g sugar/h) during prolonged and intensive exercise has a strong effect in lowering plasma levels of cortisol, epinephrine, and inflammatory immune responses including blood neutrophil and monocyte counts and cytokines. Flavonoid-rich extracts when consumed just before or chronically for days or weeks before heavy exertion partially counter postexercise inflammation and oxidative stress. Research is needed to better define optimal dosing regimens and whether unique flavonoid mixtures that include several of the most bioactive flavonoids across different subgroups amplify these influences, while also bolstering immunity and operating as exercise mimetics for mitochondrial biogenesis.

Antioxidant, N-3 PUFAs, and glutamine supplements do not counter exercise-induced immune dysfunction, and vitamin E may actually compound the oxidative stress and inflammation experienced by the endurance athlete. More research is needed for mushroom β-glucan, probiotics, and bovine colostrum. Additional research will broaden our understanding of the effects of these advanced supplements and others in providing immune benefits to athletes during physiologic stress.

References

Bakker, G.C., van Erk, M.J., Pellis, L., et al. (2010) An antiinflammatory dietary mix modulates inflammation and oxidative and metabolic stress in overweight men: a nutrigenomics approach. *American Journal of Clinical Nutrition* 91, 1044–1059.

Bergendiova, K., Tibenska, E., & Majtan, J. (2011) Pleuran (β-glucan from Pleurotus ostreatus) supplementation, cellular immune response and respiratory tract infections in athletes. *European Journal of Applied Physiology* 111, 2033–2040.

Carol, A., Witkamp, R.F., Wichers, H.J., et al. (2011) Bovine colostrum supplementation's lack of effect on immune variables during short-term intense exercise in well-trained athletes. *International Journal of Sports Nutrition and Exercise Metabolism* 21, 135–145.

Chen, Y.J., Wong, S.H., Wong, C.K., et al. (2008) The effect of a pre-exercise carbohydrate meal on immune responses to an endurance performance run. *British Journal of Nutrition* 100, 1260–1268.

Davis, J.M., Murphy, E.A., Brown, A.S., et al. (2004) Effects of oat beta-glucan on innate immunity and infection after exercise stress. *Medicine & Science in Sports & Exercise* 36, 1321–1327.

Davison, G., & Diment, B.C. (2010) Bovine colostrum supplementation attenuates the decrease of salivary lysozyme and enhances the recovery of neutrophil function after prolonged exercise. *British Journal of Nutrition* 103, 1425–1432.

Davison, G., & Gleeson. M. (2005) Influence of acute vitamin C and/or carbohydrate ingestion on hormonal, cytokine, and immune responses to prolonged exercise. *International Journal of Sports Nutrition and Exercise Metabolism* 15, 465–479.

Davison, G., & Gleeson, M. (2006) The effect of 2 weeks vitamin C supplementation on immunoendocrine responses to 2.5 h cycling exercise in man. *European Journal of Applied Physiology* 97, 454–461.

Gleeson, M. (2007) Immune function in sport and exercise. *Journal of Applied Physiology* 103, 693–699.

Gleeson, M. (2008) Dosing and efficacy of glutamine supplementation in human exercise and sport training. *Journal of Nutrition* 138, 2045S–2049S.

Gleeson, M., Nieman, D.C., & Pedersen, B.K. (2004) Exercise, nutrition and immune function. *Journal of Sports Science* 22, 115–125.

Gomez-Cabrera, M.C., Domenech, E., Romagnoli, M., et al. (2008) Oral administration of vitamin C decreases muscle mitochondrial biogenesis and hampers training-induced adaptations in endurance performance. *American Journal of Clinical Nutrition* 87, 142–149.

Harwood, M., Danielewska-Nikiel, B., Borzelleca, J.F., et al. (2007) A critical review of the data related to the safety of quercetin and lack of evidence of *in vivo* toxicity, including lack of genotoxic/carcinogenic properties. *Food and Chemical Toxicology* 45, 2179–2205.

Konig, D., Grathwohl, D., Weinstock, C., et al. (2000) Upper respiratory tract infection in athletes: influence of lifestyle, type of sport, training effort, and immunostimulant intake. *Exercise Immunology Reviews* 6, 102–120.

Konrad, M., Nieman, D.C., Henson, D.A., et al. (2011) The acute effect of ingesting a quercetin-based supplement on exercise-induced inflammation and immune changes in runners. *International Journal of Sports Nutrition and Exercise Metabolism* 21, 338–346.

Kressler, J., Millard-Stafford, M., & Warren, G.L. (2011) Quercetin and endurance exercise capacity: a systematic review and meta-analysis. *Medicine & Science in Sports & Exercise* 43, 2396–2404.

Krzywkowski, K., Petersen, E.W., Ostrowski, K., et al. (2001) Effect of glutamine supplementation on exercise-induced changes in lymphocyte function. *American Journal of Physiology—Cell Physiology* 281, C1259–C1265.

Lila, M.A. (2007) From beans to berries and beyond: teamwork between plant chemicals for protection of optimal human health. *Annals of the New York Academy of Sciences* 1114, 372–380.

Lyall, K.A., Hurst, S.M., Cooney, J., et al. (2009) Short-term blackcurrant extract consumption modulates exercise-induced oxidative stress and lipopolysaccharide-stimulated inflammatory responses. *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology* 297, R70–R81.

McAnulty, S.R., Nieman, D.C., McAnulty, L.S., et al. (2011) Effect of mixed flavonoids, n-3 fatty acids, and vitamin C on oxidative stress and antioxidant capacity before and after intense cycling. *International Journal of Sports Nutrition and Exercise Metabolism* 21, 328–337.

Murphy, E.A., Davis, J.M., Carmichael, M.D., et al. (2009) Benefits of oat beta-glucan and sucrose feedings on infection and macrophage antiviral resistance following exercise stress. *American Journal of Physiology—Regulatory, Integrative and Comparative Physiology* 297, R1188–R1194.

Murphy, E.A., Davis, J.M., & Carmichael, M.D. (2010) Immune modulating effects of β-glucan. *Current Opinion in Clinical Nutrition & Metabolic Care* 13, 656–661.

Nieman, D.C., Johansen, L.M., Lee, J.W., et al. (1990) Infectious episodes in runners before and after the Los Angeles Marathon. *Journal of Sports Medicine and Physical Fitness* 30, 316–328.

Nieman, D.C. (1997) Immune response to heavy exertion. *Journal of Applied Physiology* 82, 1385–1394.

Nieman DC. (1998) Influence of carbohydrate on the immune response to intensive, prolonged exercise. *Exercise Immunology Review* 4, 64–76.

Nieman, D.C. (2000) Is infection risk linked to exercise workload? *Medicine & Science in Sports & Exercise* 32(Suppl. 7), S406–S411.

Nieman, D.C. (2007) Marathon training and immune function. *Sports Medicine* 37, 412–415.

Nieman, D.C. (2008) Immunonutrition support for athletes. *Nutrition Reviews* 66, 310–320.

Nieman, D.C. (2009) Immune function responses to ultramarathon race competition. *Medicine Sportiva* 13, 189–196.

Nieman, D.C. (2010) Quercetin's bioactive effects in human athletes. *Current Topics in Nutraceutical Research* 8, 33–44.

Nieman, D.C., Buckley, K.S., Henson, D.A., et al. (1995) Immune function in marathon runners versus sedentary controls. *Medicine & Science in Sports & Exercise* 27, 986–992.

Nieman, D.C., Henson, D.A., Smith, L.L., et al. (2001) Cytokine changes after a marathon race. *Journal of Applied Physiology* 91, 109–114.

Nieman, D.C., Henson, D.A., McAnulty, S.R., et al. (2002) Influence of vitamin C supplementation on oxidative and immune changes following an ultramarathon. *Journal of Applied Physiology* 92, 1970–1977.

Nieman, D.C., Henson, D.A., McAnulty, S.R., et al. (2004) Vitamin E and immunity after the Kona Triathlon World Championship. *Medicine & Science in Sports & Exercise* 36, 1328–1335.

Nieman, D.C., Henson, D.A., Gojanovich, G., et al. (2006) Influence of carbohydrate

on immune function following 2 h cycling. *Research in Sports Medicine* 14, 225–237.

Nieman, D.C., Henson, D.A., Gross, S.J., (2007) Quercetin reduces illness but not immune perturbations after intensive exercise. *Medicine & Science in Sports & Exercise* 39, 1561–1569.

Nieman, D.C., Henson, D.A., McMahon, M., et al. (2008) Beta-glucan, immune function, and upper respiratory tract infections in athletes. *Medicine & Science in Sports & Exercise* 40, 1463–1471.

Nieman, D.C., Henson, D.A., Maxwell, K.R., (2009a) Effects of quercetin and EGCG on mitochondrial biogenesis and immunity. *Medicine & Science in Sports & Exercise* 41, 1467–1475.

Nieman, D.C., Henson D.A., McAnulty S.R., et al. (2009b) N-3 polyunsaturated fatty acids do not alter immune and inflammation measures in endurance athletes. *International Journal of Sports Nutrition and Exercise Metabolism* 19, 536–546.

Nieman, D.C., Stear, S.J., Castell, L.M., et al. (2010a) A–Z of nutritional supplements: dietary supplements, sports nutrition foods and ergogenic aids for health and performance: part 15, flavonoids. *British Journal of Sports Medicine* 44, 1202–1205.

Nieman, D.C., Williams, A.S., Shanely, R.A., et al. (2010b) Quercetin's influence on exercise performance and muscle mitochondrial biogenesis. *Medicine & Science in Sports & Exercise* 42, 338–345.

Nieman, D.C., Henson, D.A., Austin, M.D., et al. (2011) Upper respiratory tract infection is reduced in physically fit and active adults. *British Journal of Sports Medicine* 45, 987–992.

Nieman, D.C., Konrad, M., Henson, D.A., et al. (2012) Variance in the acute inflammatory response to prolonged cycling is linked to exercise intensity. *Journal of Interferon & Cytokine Research* 32, 12–17.

Peters, E.M., Goetzsche, J.M., Grobbelaar, B., et al. (1993) Vitamin C supplementation reduces the incidence of postrace symptoms of upper-respiratory-tract infection in ultramarathon runners. *American Journal of Clinical Nutrition* 57, 170–174.

Ristow, M., Zarse, K., Oberbach, A., et al. (2009) Antioxidants prevent health-promoting effects of physical exercise in humans. *Proceedings of the National Academy of Sciences of USA* 106, 8665–8670.

Rohde, T., MacLean, D.A., & Pedersen, B.K. (1998) Effect of glutamine supplementation on changes in the immune system induced by repeated exercise. *Medicine & Science in Sports & Exercise* 30, 856–862.

Scherr, J., Nieman, D.C., Schuster, T., et al. (2011) Non-alcoholic beer reduces inflammation and the incidence of upper respiratory tract infections following the Munich Marathon: a randomized, controlled, double-blinded trial. *Medicine & Science in Sports & Exercise* 44, 18–26.

Senchina, D.S., Shah, N.B., Doty, D.M., et al. (2009) Herbal supplements and athlete immune function—what's proven, disproven, and unproven? *Exercise Immunology Reviews* 15, 66–106.

Shing, C.M., Hunter, D.C., & Stevenson, L.M. (2009) Bovine colostrums supplementation and exercise performance: potential mechanisms. *Sports Medicine* 39, 1033–1054.

Sureda, A., Tauler, P., Aguiló, A., et al. (2008) Influence of an antioxidant vitamin-enriched drink on pre- and post-exercise lymphocyte antioxidant system. *Annals of Nutrition and Metabolism* 52, 233–240.

Walsh, N.P., Gleeson, M., Pyne, D.B., et al. (2011) Position statement. Part 2: maintaining immune health. *Exercise Immunology Reviews* 17, 64–103.

West, N.P., Pyne, D.B., Peake, J.M., & Cripps, A.W. (2009) Probiotics, immunity and exercise: a review. *Exercise Immunology Reviews* 15, 107–126.

Yfanti, C., Akerström, T., Nielsen, S., et al. (2010) Antioxidant supplementation does not alter endurance training adaptation. *Medicine & Science in Sports & Exercise* 42, 1388–1395.

Chapter 40

The Diabetic Athlete

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Introduction

For many years, athletes with "diabetes" have struggled to find enough information and appropriate levels of care and input to allow elite performance to be maintained. At the 2000 Olympics, two individuals with type 1 diabetes, Gary Hall Jr and Sir Steve Redgrave, won gold medals in swimming and rowing, respectively. These achievements have contributed to inspiring both athletes and practitioners to pursue regimens surrounding treatment, nutrition, and training to further establish 'normal' performance in the athletic environment (Gallen et al., 2003). Changes in insulin regimens, the understanding of diet, most notably carbohydrate counting, and the use of the multidisciplinary team including medicine, physiology, and nutrition have proved key.

Diabetes

Diabetes is a group of chronic metabolic disorders characterized by hyperglycemia resulting from a relative deficiency in insulin, either through reduced insulin secretion, reduced insulin action or both. Diabetes mellitus is a major cause of morbidity and mortality worldwide, with the effects of chronic hyperglycemia altering tissue metabolism and causing long-term end-organ damage and severe health complications. These complications include retinopathy, neuropathy, peripheral vascular disease, atherosclerotic cardiovascular and cerebrovascular diseases, fractures, frailty, depression, and cognitive decline (Goff et al., 2007). About 346 million people worldwide have diabetes (WHO, 2011), and the overall diabetes cost to the US economy was $178 billion in 2007 (Sieverdes et al., 2010). Worryingly, the worldwide prevalence of diabetes appears to be increasing, so diabetes and its complications are set to be an increasing burden on healthcare budgets.

The diagnosis of diabetes has been standardized by the World Health Organization (WHO) and is based on typical symptoms as well as blood glucose levels in defined parameters (WHO, 2010). There are several types of diabetes but almost all fall into two broad categories; type 1 (T1DM) and type 2 (T2DM) diabetes. T1DM is due to an absolute deficiency of insulin production secondary to autoimmune destruction of the pancreas and typically presents acutely in childhood. T2DM develops due to insulin resistance and eventual failure of the pancreatic beta cells to provide an adequate compensatory insulin secretory response. It is associated with inactivity, obesity, and family history (DeFronzo, 2004) and it typically presents later in life. However, with the rise in obesity in younger age groups, children and young adults are increasingly being diagnosed with T2DM (Deckelbaum & Williams, 2001).

Exercise and nutrition are recognized as being important aspects of diabetic management. The physiological response to exercise is different in T1DM and T2DM and while exercise in individuals

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with T1DM will improve general health, it can be a challenge for the recreational or elite athlete and requires careful medical management. Exercise in T2DM is the "cornerstone" of management and can improve glycemic control, minimizing the need for medication and diabetic complications.

Normal Glucoregulation During Exercise

Glucose homeostasis is finely balanced in health with tissues in the body, particularly the brain, requiring a steady supply of glucose to function. Increased glucose requirements in exercise are met by muscle glycogenolysis and glucose uptake. This is matched by an increase in blood glucose derived from hepatic glycogen stores and gastrointestinal absorption if carbohydrate is consumed. In prolonged exercise, there is increasing contribution from gluconeogenesis and utilization of free fatty acids (FFAs). Insulin, catecholamines, and glucagon play key roles and their circulating levels change depending on the metabolic demand and environment. In simple terms, insulin concentrations fall during exercise allowing for an increasing influence of glucagon and catecholamines to promote glucose and FFA production and mobilization from storage. Conversely in the postprandial or postexercise state insulin rises, promoting glucose storage in the form of hepatic and skeletal muscle glycogen.

At rest in the fasted state, the insulin–glucagon ratio falls, promoting glucose availability. Specifically, plasma insulin concentrations fall and glucagon levels rise, facilitating increased glycogenolysis and hepatic gluconeogenesis in order to increase the circulating plasma concentration of glucose.

In the postprandial state, blood glucose concentration increases and the insulin–glucagon ratio increases, promoting glucose storage by triggering inhibition of gluconeogenesis and glycogenolysis while concurrently stimulating glucose uptake, mainly into peripheral skeletal muscle cells, thereby reducing blood glucose levels (Peirce, 1999). Insulin stimulates peripheral glucose disposal through facilitated diffusion via the translocation of specific transporter proteins (glucose transporter 4, GLUT4) from the cell cytosol to the cell surface membrane (Zisman et al., 2000) while insulin-mediated storage of glucose requires a fully functioning insulin receptor substrate (IRS) phosphorylation pathway to allow efficient conversion to muscle glycogen (Frosig & Richter, 2009).

During the early stages of moderate exercise (50–65% VO_2 max), skeletal muscle uses glucose derived from muscle glycogen stores to fuel activity, but as exercise continues blood glucose and FFAs are used increasingly. At low intensity, FFA oxidation predominates while carbohydrate oxidation (CHO) predominates at exercise intensities above about 80% VO_2 max (Maughan et al., 1997). The levels of blood glucose and FFA are maintained by a reduction of circulating insulin (Robertson et al., 1976) and an increase in "counterregulatory hormones" (adrenaline, cortisol, glucagon, and growth hormone). The resulting decrease in the insulin–glucagon ratio increases hepatic gluconeogenesis and glycogenolysis while mobilizing FFA from the adipose tissue. The reduction in the insulin–glucagon ratio is vital to maintain exercise and prevent hypoglycemia. Despite low circulating insulin levels, the rate of glucose uptake is elevated in skeletal muscle during exercise as GLUT4 is recruited to the membrane surface in contracting muscle through calcium channel activation, independent of insulin (Chibalin et al., 2000). In prolonged exercise, hepatic glycogen levels fall and blood glucose is relied on. In endurance athletes, FFA oxidation predominates over CHO oxidation at relatively higher levels of VO_2 than in untrained individuals (Maughan et al., 1997), so hepatic glycogen levels are maintained for longer and fatigue is delayed. However, glucose levels will continue to fall as hepatic glycogen is depleted.

At the end of exercise, the body in essence enters a fasted state where glycogen stores in the muscle and the liver are low. Hepatic gluconeogenesis is promoted by a rise in glucagon decreasing relatively the insulin–glucagon ratio. After exercise, circulating catecholamine levels particularly adrenaline fall, disinhibiting insulin levels therefore causing a rise in the insulin–glucagon ratio. This environment of hyperinsulinemia, with increased insulin sensitivity after exercise, and hyperglycemia are ideal conditions for replenishment of muscle glycogen levels allowing the body to recover from this bout of exercise and prepare itself for the next.

High-intensity exercise (above VO_2 max) is characterized by lactate accumulation (Marliss & Vranic, 2002) and high levels of catecholamines, which can be in the order of 20 times higher than usual. Low insulin causes a decrease in the insulin–glucagon ratio which increases glucose production. Glucose production is greater than glucose uptake so circulating levels rise. After explosive exercise, the insulin–glucagon ratio increases as insulin levels rise in response to hyperglycemia and the removal of circulating catecholamines. During this period of hyperglycemia and hyperinsulinemia, glycogen stores are replenished preparing the athlete for further exercise.

Type 1 Diabetes

The general health benefits of exercise are well described and are in the large part no different in individuals with T1DM. However, exercise in those with T1DM provides a number of challenges in achieving the balance between tight control, regimens that suit performance, and regimens that can adjust to the unpredictability of the sporting environment.

Unfortunately, there is no evidence that athletic exercise improves glycemic control (Moy et al., 1993) and indeed intense exercise may even provide greater excursions in blood control, important to any athletes with T1DM. Reassuringly, there is evidence that athletes with T1DM who have good glycemic control have the same cardiopulmonary capacities and the ability to improve with training as those individuals without diabetes (Baldi et al., 2010). It is therefore vital that medical teams assisting these athletes formulate regimens that control glycemia well for short-term athletic gains as well as long-term avoidance of diabetic complications.

Aerobic Exercise

Athletes with T1DM have an absolute deficiency of insulin production and therefore glycemic control is achieved by exogenously administered insulin thereby affecting insulin–glucagon ratios. During exercise, insulin concentration levels may not fall meaning the insulin–glucagon ratio remains relatively high increasing the risk of hypoglycemia. The increased circulating plasma insulin inhibits hepatic glucose production (glycogenolysis and gluconeogenesis) and mobilization of FFA while inhibiting glucagon secretion and catecholamine responses enhancing peripheral skeletal muscle glucose uptake. Indeed, if insulin is administered intramuscularly into an exercising limb rather than subcutaneously, or if exercise is occurring in hot and humid conditions, its effect is again enhanced.

Furthermore, exercise increases insulin sensitivity, increasing the risk of postexercise hypoglycemia, which usually occurs within 4 hours after exercise although it can be delayed. This is due to both increased insulin sensitivity of GLUT4 translocation and depleted glycogen after a bout of exercise. Delayed-onset hypoglycemia (DOH) which can occur up to 24 hours after exercise often causes hypoglycemia at night (Tattersall, 1995) and is more likely if two or more episodes of exercise have occurred on the same day. To complicate matters further, an episode of hypoglycemia is a major risk factor for further hypoglycemic episodes soon after, with antecedent hypoglycemia blunting the counterregulatory response: this means that further exercise soon afterward increases the susceptibility to hypoglycemia (Galassetti et al., 2003). The intensity of exercise may mask symptoms of hypoglycemia as well, such that during vigorous activity this feedback may be impaired. Orthostatic hypotension, impaired thermoregulation, and neuropathy can be confused with the hypoglycemic symptoms.

Anaerobic Exercise

High-intensity exercise, just as in health, causes a marked increase in circulating catecholamines and glucagon, which promote hyperglycemia on a background of stable insulin levels. The overall result is postexercise hyperglycemia. Athletes with T1DM are unable to increase insulin levels endogenously, so postexercise glycogen replenishment is less effective than in healthy individuals.

In summary, exogenous insulin is crucial in the management of T1DM but demands planning and rigor. At rest, too much can cause hypoglycemia and too little can cause hyperglycemia, resulting in

complications. Thus athletes with T1DM must balance nutrition and insulin regimens that promote "tight" control and yet do not cause hypoglycemia during or after aerobic exercise and yet control postexercise hyperglycemia, especially that seen in high-intensity exercise.

Management of Type 1 Athletes

Nutrition and Insulin Regimens

The nutritional requirements of athletes with T1DM are in the large part no different from those of any other athlete with a large energy expenditure, and demand high CHO intake to match. Dietary strategies must therefore encompass matching insulin regimens to the nutritional demands and a normal varied diet with a balance of macronutrients. Exercise brings with it the risk of hypoglycemia and so additional CHO may need to be consumed and/or insulin dosage adjusted before, during, and after exercise. A tailored nutritional and insulin regimen for each individual is needed. Patient education significantly improves glycemic control but, unfortunately, a degree of trial and error is necessary in all individuals with T1DM who take up any new activities.

"Counting the carbohydrate" content of meals has become increasingly popular as a means to adjust insulin doses dependent on dietary CHO consumed and activity carried out. Validated, structured patient education programs such as Dose Adjustment for Normal Eating (DAFNE) are commonplace (DAFNE, 2002) and provide a method of obtaining good glycemic control. Individuals with T1DM are taught to estimate the amount of CHO consumed in each meal. Every 10 g of CHO requires an additional unit of insulin to be administered. In addition, a number of online and smartphone apps can facilitate this process (http://caloriecount.about.com; see Figure 40.1).

Our understanding of appropriate insulin regimens has improved in recent decades and with this has come a significant improvement in the range of commercially available insulin and insulin-associated technology allowing greater flexibility. With the variety of insulins available, all of which act for different durations, an individualized regimen can be tailored but an athlete must self monitor blood glucose levels regularly to develop an understanding of what affects their glucose level.

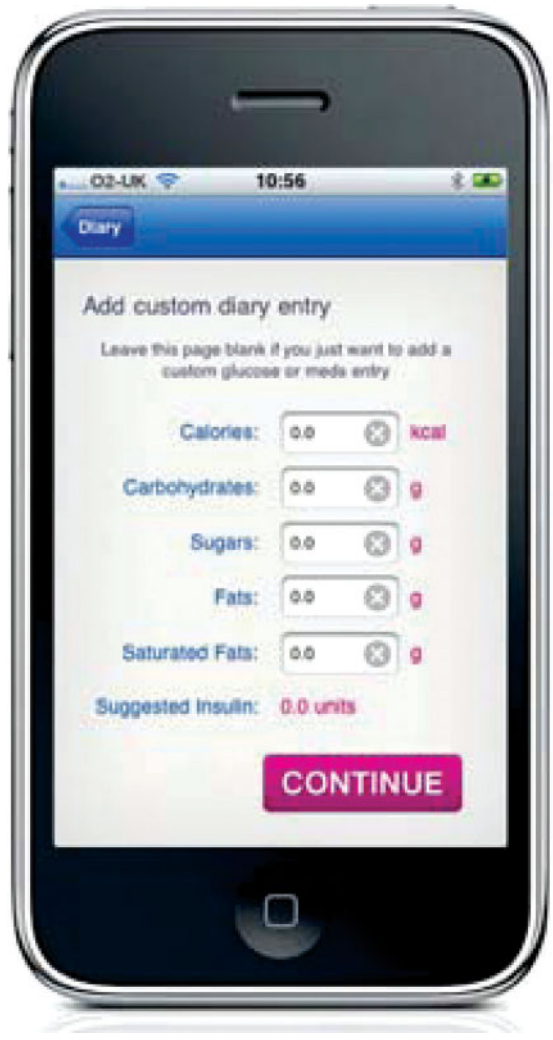

Figure 40.1 Example of a smartphone carbohydrate counting application.

The use of continuous subcutaneous insulin infusion (CSII), otherwise known as "insulin pumps," has been a significant advance in the ability to control insulin delivery and therefore control blood glucose tightly (Frohnauer et al., 2000). Insulin pumps are devices that attach externally to the athlete.

They deliver a background "basal" amount of insulin and provide bolus amounts with each meal. Insulin pumps use fast-acting insulins and deliver them in precise amounts. This ability to change the dose quickly is ideal for athletes who may need to exercise in a less structured way, for example, those needing to perform multiple times in a day. The athlete can suspend, or reduce, the basal insulin prior to exercise to prevent hypoglycemia. Insulin pumps do have limitations, especially for those involved in contact sports and until recently waterproof pumps were not available. Indeed, the tighter glycemic control during exercise has been associated with greater postexercise hyperglycemia (Lumb & Gallen, 2010), which could affect overall glycemic control and increase the risk of ketosis in the short term and diabetic complications long term.

Exercise

Many factors contribute to the amount of insulin required by an athlete. It is vital that enough CHO is available before, during, and after activity to maintain glycogen levels. Traditionally 60% of the energy in an athlete's diet should be derived from CHO (Burke et al., 2004). However, a more practical method is to advise the amount of CHO dependent on activity and relative to body mass (Burke & Deakin, 2003). Broadly, very light exercise usually requires 5 g of CHO/kg of body weight (BW)/day. Moderately intense exercise needs between 5–7 g of CHO/kg of BM/day if performed for 1–2 hours each day. Intense activity may require up to 12 g of CHO/kg of BW/day if performed up to 4–5 hours per day. Therefore, the amount of insulin administered needs to be adjusted by athletes with T1DM depending on the type, intensity, and time of the day exercise is performed and how much carbohydrate has been consumed (Gallen et al., 2011). A varied diet that supports performance in athletes also requires fluid, sufficient protein ingestion (1.2–1.7 g/kg/day) dependent on whether endurance or strength training is performed, and sufficient fat (less than 30–40 g/day) (Burke et al., 2004).

The common practice of CHO loading in athletes has not been investigated fully in T1DM. Excessive CHO load in the days prior to an event may adversely affect glycemic control and T1DM are generally advised not to CHO load (Gallen et al., 2011). Some older studies have suggested the glycogen levels and therefore performance suffer on higher CHO regimens in T1DM.

Any physical activity can potentially reduce the requirement for insulin but minimal adjustments are needed if exercise lasts only for 20–30 minutes at an intensity of less than 70% VO_2 max. Ideally, a meal high in CHO of low glycemic index should be eaten 1–3 hours before exercise (Horton, 1988). If moderate exercise is carried out adjustments to insulin should be considered, but if exercise is unplanned and adjustments to insulin dose cannot be made, 20–30 g of CHO should be consumed just before and every half an hour during exercise.

If early morning physical activity is planned, the intermediate or long-acting insulin should be reduced by 20–50% the evening before and the morning pre-exercise blood glucose should be checked. If it rises above 7–8 mmol/l it may compromise long-term control, although levels below 10–12 mmol/l will allow safe exercise. Conversely levels below 6 mmol/l may increase the risk of hypoglycemia even if the exercise intensity is likely to be moderate (50–70% VO_2 max). The morning regular-acting insulin dose should also be reduced by 30–50% before breakfast or even omitted if exercise is taken before food. Insulin should otherwise be taken 30–60 minutes before the meal as usual.

It is accepted that exercise should be avoided if a pre-exercise blood glucose reading is above 17 mmol/l or above 14 mmol/l with ketones found on urinalysis. If blood glucose is between 5.5 and 6.0 mmol/l and exercise is expected to be vigorous, then extra CHO should be ingested (Peirce, 1999). Table 40.1 outlines this and looks further at the metabolic control of athletes with T1DM generally.

Elite athletes, depending on their sport, tend to exercise for longer and at higher intensity. The night before a planned early morning session, the intermediate- to long-acting insulin should be reduced by 50–70%. A reduction of 70–90% of insulin is also more likely to be required in the morning prior to exercise too. Insulin may be omitted completely for especially intense exercise approaching VO_2 max as long as the athlete is monitored closely. Insulin

Table 40.1 Metabolic control in type 1 diabetes

Monitor	Activity
Establish good control before exercise	If unstable avoid exercise until control established
Check nature of exercise, ambient conditions, changes in weight, hydration status	Adjust for cold/heat/winds/weight loss/intense game/activity
Blood glucose >14 mmol/l (250 mg/dl) if accompanied by ketosis ($1–2^{++}$) urinalysis	Avoid exercise
Blood glucose >17 mmol/l (300 mg/dl)	Avoid exercise
Blood Glucose <5.5–6.0 mmol/l (100 mg/dl)	Ingest extra carbohydrate and avoid vigorous or prolonged exercise unless established protocol
Monitor glucose before and after exercise	Establish routine and response to exercise Identify changes required prior to activity Identify symptoms of hypoglycemia
Establish timing and quantity of food	Allow for delayed gastric emptying for glucose absorption and discomfort Provide readily available and absorbable carbohydrate
Adjustment in inactivity/illness	May alter/reduce insulin sensitivity. May need higher insulin dosages relative to CHO
Adjust insulin doses according to intensity of exercise	For moderate intensity exercise allow 50% reduction in insulin dosages For high/prolonged intensity reduce dose by 70–90%

Source: Reproduced from ADA (2008), with permission from the American Diabetes Association.

pumps are an option in this group and they ideally need to be adjusted 30–60 minutes prior to exercise to allow the glucose level to stabilize (Frohnauer et al., 2000), so CHO (20–30 g) may be consumed prior to exercise in this scenario to boost blood glucose levels.

During Exercise

Ingestion of 20–30 g of CHO may typically be needed every half an hour during endurance exercise. Explosive, anaerobic, athletes will usually need no additional CHO during events. More CHO may be needed if the endurance exercise is intense and prolonged, if the athlete has a higher body mass, or if the pre-exercise bolus of insulin could not be reduced prior to exercise. Horton reported that athletes with T1DM may need to consume 70–80 g of CHO per hour of exercise (Horton, 1988). This is in keeping with Francescato, who found that CHO requirements in T1DM undertaking moderate exercise were as high as 1 g of CHO/kg/h exercised (Francescato et al., 2004).

Athletes with T1DM ought to derive their CHO, prior to exercise, from high-CHO foods such as bananas, pasta, and bread (wholemeal). Many athletes struggle to consume food during events because either the sport or activity does not lend itself to doing so or athletes may find they suffer from gastrointestinal discomfort which impairs performance. During exercise, sports drinks or gels, which are CHO-rich solutions and suspensions, respectively, provide a convenient well-tolerated alternative. In addition it has become clear that minimal insulin is required during exercise with equal performance such as observed in those using insulin pumps set at 50% normal requirement or switched off (Admon et al., 2005).

As discussed, hypoglycemia is a risk for the athlete with T1DM. At its worst it can cause collapse and coma. Athletes should be encouraged to learn the early symptoms of hypoglycemia in light of the fact that normal symptoms during prolonged exertion may make identification of hypoglycemia difficult. Athletes with T1DM should either reduce the intensity of exercise or stop and should

have immediate access to rapid-acting CHO to boost circulating glucose levels to treat hypoglycemia quickly. Dextrose (glucose) tablets or sports gels are quick acting and can be easily carried by the athlete during exercise or kept close by, e.g., pitchside. Ideally a glucose meter should be available to check blood glucose but the priority is administration of fast-acting CHO. After hypoglycemia, athletes with T1DM should consume food containing longer acting CHO (low glycemic index) to maintain blood glucose, e.g., a sandwich at the earliest point after exercise has been stopped (Table 40.2).

After Exercise

The postexercise or recovery period is particularly important for the athlete with T1DM in preparing for future exercise. The goal of this period is to replenish glycogen stores as well as avoiding hypoglycemia. It has been recommended that CHO should be consumed at a rate of 1.2–1.5 g of CHO per kilogram per hour for the initial hours after endurance exercise (Burke et al., 2004). About 500–600 g of CHO may be needed to replenish the glycogen stores in an endurance athlete (Ivy, 1991) meaning a considerable amount of food must be consumed. Burke showed that high glycemic index (i.e., fast-acting) CHO replenished glycogen stores quicker than low glycemic index CHO (Burke et al., 1993). However, using high glycemic index CHO exclusively may make DOH more likely so a combination of high and low glycemic index CHO is recommended.

When insulin reduction has occurred prior to exercise, hyperglycemia is more likely after exercise. However, insulin doses should still be reduced by 25–50% to try to reduce the likelihood of nocturnal hypoglycemia. Reductions in insulin maybe needed for 24–36 hours after intense exercise (Table 40.2).

Recently some strategies have developed to reduce hypoglycemia during and after exercise, which are particularly relevant in elite athletes. Caffeine

Table 40.2 Blood glucose challenges in exercise in type 1 diabetes

Table of performance issues	Strategies
Muscle glycogen depletion	• Reduce postexercise hyperglycemia with bolus short-acting insulin • CHO loading strategies using insulin to boost uptake
Postexercise hyperglycemia	• Use insulin after exercise to suppress counterregulatory hormones
Unexpected exercise • Sudden/unexpected exercise (too much insulin or insufficient CHO) • Delayed exercise (delayed schedule)	• CHO ingestion 30–50 g gel prior to 30–40 minutes prior to exercise/regular CHO 4–6% solution during exercises • Reduce intensity exercise/add caffeine • Use short-acting insulin bolus (1–2 units every 1–2 hours + 30–50 g CHO/protein). Glucose levels may start rising if insulin bolus wears off
Hypoglycemia during exercise	• CHO snacks before exercise • Glucose ingestion during exercise • Reduce insulin doses before exercise including long-acting dose the night before • Prevent hypoglycemia in days before so normal counterregulatory response
Hyperglycemia during exercise	• Increase aerobic training base to improve insulin sensitivity • Use of low-dose insulin pump • Slight increase in long-acting insulin before exercise • Very small amount of short-acting insulin prior to exercise • Consider insulin sensitizer • Review CHO intake. Consider reduced high glycemic index and replace with very low glycemic index/long-acting
Heavy training fatigue	• Recurrent heavy training challenges CHO stores • Challenges hormonal milieu

intake has been suggested before exercise to reduce hypoglycemia during and after exercise. Caffeine, if taken at 5 mg/kg, appears to reduce CHO requirements (Gallen et al., 2010) but can overstimulate the athlete, affecting recovery strategies including sleep (see Chapter 25). Ten-second sprints performed before or after exercise increase circulating catecholamines and appear to reduce postexercise hypoglycemia (Bussau et al., 2006, 2007). This novel strategy could be useful to supplement plans to avoid hypoglycemia when unplanned exercise occurs. Athletes performing high-intensity exercise tend to have higher postexercise hyperglycemia than athletes performing endurance activities. In health, hyperinsulinemia would accompany hyperglycemia after exercise, producing an ideal environment to replenish glycogen. The T1DM athlete cannot increase the endogenous circulating insulin, so strategies have developed whereby a small amount of insulin is administered after exercise to try to replicate the physiology seen in health (Marliss & Vranic, 2002). This method may improve glycogen replenishment after exercise optimizing recovery for the elite T1DM athlete. This additional insulin would have to be administered with additional CHO eaten by the athlete. This strategy would require close blood glucose monitoring and may increase the risk of nocturnal hypoglycemia so would appear best used only in the closely managed elite athlete.

Performance Issues in T1DM

Traditionally it has been felt that individuals with diabetes cannot succeed at the highest level of athletic endeavour, but there are examples to prove otherwise. However, despite the best laid plans, there are still some performance issues. The potential role of supplements in correcting these is outlined in Table 40.3.

As with any athlete, if performance is impaired a full medical workup should entail checks for undercurrent illness and cardiorespiratory fitness and or dysfunction. In athletes with T1DM, there are a number of specific issues that are worth considering. These are outlined in the Table 40.4 and in general center around the difficulties in balancing starting blood sugars, insulin regimens, and nutritional input prior, during, or after activity. In addition, specific sports produce unique challenges, with some examples outlined in Table 40.5.

Table 40.3 Consideration of use of supplements in athletes with diabetes

Supplements in exercise	Positives	Negatives
Caffeine	May help delay or prevent hypoglycemia Helps mobilize FFA either before, during, or after exercise	Altered counterregulatory response may produce unknown dosage response
Protein	Can be used as part of normal anabolic training process. Can be used in conjunction with small doses of short acting insulin as anabolic. Most athletes consume 1.2–17 g/kg/day	Mindful of abuse and potential for nephropathy. Not recommended >20% of energy intake (ADA) or 1 g/kg/day (UKD) are conservative
Medium/long chain CHO such as maltodextrins	Long chain/complex carbohydrate such as maltodextrins may encourage more stable blood glucose control and maintain hepatic glycogen stores Can help with CHO loading	May induce insulin resistance
Creatine	Combined with insulin and CHO loading regimens may have anabolic contribution	Fears of renal toxicity and muscle cramping are unfounded
Chromium	Theoretical	No evidence
Antioxidants	Theoretical	No evidence

Source: From Peirce (1999), with permission from BMJ Publishing Group Ltd.

Table 40.4 Underperformance issues in athletes with type 1 diabetes

Underperformance issue	Possible causes in T1DM	Actions
Fatigue during intense exercise	Low glycogen stores: hepatic or muscle (reduced glycogen can lead to reduced power output and or hypoglycemia) Antecedent hypoglycemia may reduce counterregulatory response	Tackle postexercise refueling and overnight refueling. Insulin resistance increases in days off so may need slightly higher insulin doses May need to loosen control slightly in 48 hours before competition
Increased insulin resistance	T1DM is sometimes associated with insulin resistance. Produces paradox of hyperglycemia but glycogen depletion and reduced CHO oxidation	Check overall insulin dosing. If sport is largely anaerobic consider adding aerobic training to improve insulin sensitivity
Hyper and hypoglycemia during exercise	These are covered in the above but may have glycogen storage and utilization factors involved	Beware using too much insulin during exercise including pumps. Very little needed, especially during endurance activities
Disordered eating	Just like any athletic population there is a risk of disordered eating and body composition that will seriously impair performance	Consider Disordered Eating Questionnaire and work with psychologist

Table 40.5 Challenges of type 1 diabetes in different sports

Sport	Challenges	Actions
Strength-based explosive sports, e.g., weight lifting, martial arts, sprinting	Can produce relative insulin resistance Postexercise hyperglycemia Monitor for complication of T1DM such as renal impairment	Add aerobic conditioning Care with high excess protein ingestion and "supplements" culture
Endurance Sport (i.e., triathlon, distance running)	Difficulty in matching high energy requirements and refueling Hypoglycemia during exercise Hydration and fluid/CHO absorption	May need larger bolus injections after exercise and late evening refueling. See Tables 40.1 and 40.2 Likely to need minimal insulin and regular CHO and sports drinks just like any athlete without diabetes
Intermittent Sport (football, rugby)	May drop blood sugar concentrations quickly. Difficult to predict intensity	Have CHO gels and drinks available throughout. May need small additional insulin injection at half time
Unplanned exercise (i.e., tennis, cricket)	Starting times for matches and innings very variable and may be weather dependent	Need to ensure regular smaller meals and snacks with small top ups of short-acting insulin
Extreme Sports (motor racing) (environmental extremes)	Main problems will relate to potential for hypoglycemia	May consider insulin pump Cold weather and extreme conditions require particular care due to risks of hypoglycemia and reduced awareness

Type 2 Diabetics

T2DM occurs mainly in the adult population and is caused by insulin resistance, manifest as hyperinsulinemia and hyperglycemia. It is strongly correlated with obesity, physical inactivity, and family history and accounts for 90% of all diabetes. Typically those with T2DM are not athletes. Overall, the incidence of disease is increasing in Western societies. Obesity and inactivity are increasing in younger people too and T2DM is now being diagnosed in children and young adults (Deckelbaum & Williams, 2001). Nutrition is an important factor in the development of T2DM. Typically, excess energy is consumed with a high intake of fats, particularly saturated/trans fats.

T2DM presents as insulin resistance, which consists of an impaired response to endogenous insulin. In health, insulin acts to stimulate glucose uptake into skeletal muscle and hepatic cells and inhibits hepatic glucose production thereby reducing blood glucose levels. Insulin resistance means the mechanism of glucose uptake into hepatic and skeletal muscle cells is impaired and as pancreatic beta cells function in T2DM, as opposed to T1DM, hyperinsulinemia is the initial response to mitigate hyperglycemia secondary to insulin resistance. In T2DM, and its precursor impaired glucose tolerance (IGT), both GLUT4 translocation and the IRS phosphorylation pathway are faulty (Frosig & Richter, 2009) and do not respond to the stimulation of high circulating levels of insulin, so hyperglycemia continues. Increasingly we understand that obese individuals exhaust cellular fat metabolism capacities because of increased plasma FFA availability. This excess causes fatty acid metabolites to be incompletely stored in cells like skeletal muscle cells. It is these fatty acid metabolites that interfere with the IRS phosphorylation pathway, GLUT4 translocation, and hexokinase activity (Schenk & Horowitz, 2007). This explains why those who are obese are more likely to have IGT and progress to T2DM.

Exercise improves insulin resistance, allowing increased peripheral uptake of glucose (Praet & van Loon, 2007). It also promotes CHO oxidation and storage with decreased hepatic glucose production and increased GLUT4 translocation, increases blood flow to muscles, and causes energy expenditure promoting weight loss (Praet et al., 2006). Exercise in T2DM, therefore, is able to produce significant falls in circulating blood glucose and insulin concentrations without increasing the risk of hypoglycemia. Indeed, only a single bout of exercise lasting 20–30 minutes, endurance or resistance based, improves insulin sensitivity and stimulates cellular glucose uptake (Wallberg-Henriksson et al., 1998). Improvements can occur with endurance training at 50–70% VO_2 max and the effect of a single bout of endurance exercise on insulin sensitivity can last 24–72 hours (Praet et al., 2006). Resistance exercise increases muscle mass which provides a greater area for glucose disposal, further improving insulin sensitivity and glycemic control irrespective of age (Castaneda et al., 2002).

Several large epidemiological studies have now conclusively shown that IGT and progression to T2DM can be prevented. The Malmo study (Eriksson & Lindgarde, 1991) is the landmark study in the prevention of T2DM with lifestyle modification. For 5 years, 41 patients with T2DM and 181 patients with IGT were followed. Each participant was assigned to an intervention group, where diet and physical activity advice was given, or a control group, where no lifestyle advice was provided. Those who received lifestyle advice lost 2–4% of their body weight, while the control group gained weight. The glycemic control of the T2DM improved with lifestyle advice while 67% of the control group diabetics had worsened glucose tolerance. Importantly, lifestyle advice saw a 63% relative risk reduction of progressing to T2DM in the IGT group.

Studies confirming these findings have been repeated in Japan, China, Finland, India, and the United States. All have shown that exercise is beneficial in preventing IGT from progressing to T2DM. The Da Quig diabetes prevention study examined 577 patients with IGT for 20 years (Li et al., 2008). Patients were divided into three groups which received diet, exercise, or a combination of diet and exercise advice, respectively. The most effective groups in preventing progression to diabetes were the "exercise alone" and the "diet and exercise" groups. Indeed the exercise alone group proved the most effective intervention. The

Indian diabetes mellitus prevention study randomized a cohort of patients with IGT into four groups; a control group, a group who were given diet and exercise advice, a group who were given metformin alone, and finally a group who were given lifestyle advice and metformin (Ramachandran et al., 2006). Interestingly, the groups given lifestyle advice and a combination of lifestyle advice and metformin produced similar results. The study concluded that metformin did not further reduce progression to T2DM if sufficient exercise and dietary changes are made.

In summary, the goal with individuals with T2DM is to modify their diet to reduce energy intake and to exercise to improve insulin sensitivity. This strategy can be enough to manage the disease successfully and indeed to prevent its onset in those with IGT.

Management of Individuals with T2DM

Nutrition and Exercise

Before Exercise Individuals with T2DM are usually overweight and may have risk factors for cardiovascular disease, therefore before commencing an exercise regimen it is prudent to ensure a medical examination is performed (ADA, 2004). Diets should be changed to reduce energy intake. Specifically saturated fats and trans fats should be reduced and low glycemic index CHO be eaten rather than high glycemic index foods, both of which improve glycemic control (ADA, 2008; Willett et al., 2002). Nutritional advice is summarized in Table 40.6. Overall, more exercise performed and an improved diet will result in a negative energy balance meaning body weight, specifically fat mass, is reduced, thereby decreasing fatty acid metabolites at a cellular level improving insulin sensitivity.

Immediately before exercise, no additional CHO should be consumed to supplement a healthy diet as the goal is weight loss (ADA, 2008).

During Exercise Once again no additional CHO is recommended during exercise as the objective of this activity is weight loss. Most overweight individuals will exercise at low to moderate intensity and as a result fat oxidation will fuel continued activity more so than CHO oxidation (Maughan et al., 1997). Indeed, individuals with T2DM use up less energy than individuals without T2DM because of overall poorer aerobic capacity.

After Exercise Insulin sensitivity is improved after an acute bout of exercise. Exercise improves the efficiency of IRS phosphorylation pathway, GLUT4 translocation, and hexokinase activity thereby improving glycogen synthesis and reducing circulating plasma glucose. Improved glycogen stores will allow more endurance capacity and encourage longer exercise duration in the future. To encourage further energy utilization, CHO consumption is not advised immediately after exercise and individuals with T2DM are advised to wait till their next regular meal (Jensen, 2004).

Table 40.6 Type 2 diabetes: role of nutrition in prevention

Preventing diabetes (primary prevention).
Among individuals at high risk for developing type 2 diabetes. Structured programs that emphasize lifestyle changes that include moderate weight loss (7% body weight) and regular physical activity (150 min/week), with dietary strategies including reduced energy intake and reduced intake of dietary fat, can reduce the risk for developing diabetes and are therefore recommended.
Individuals at high risk for type 2 diabetes should be encouraged to achieve the USDA recommendation for dietary fiber (14 g fiber/1000 kcal) and foods containing whole grains.
There is no sufficient, consistent information to conclude that low-glycemic load diets reduce the risk for diabetes. Nevertheless, low-glycemic index foods that are rich in fiber and other important nutrients are to be encouraged.
Observational studies report that moderate alcohol intake may reduce the risk for diabetes, but the data do not support recommending alcohol consumption to individuals at risk of diabetes.
No nutrition recommendation can be made for preventing type 1 diabetes.
Although there are insufficient data at present to warrant any specific recommendations for prevention of type 2 diabetes in youth, it is reasonable to apply approaches demonstrated to be effective in adults, as long as nutritional needs for normal growth and development are maintained.

Source: Reproduced from ADA (2008), with permission from the American Diabetes Association.

Summary

T1DM and T2DM challenge the athlete and the multidisciplinary team that works with them. A careful understanding of the physiology underlying normal exercise metabolism and the changes that occur with diabetes plays a key role in providing a strategy of dietary interventions to match necessary changes in medications and insulin regimens. Careful planning of exercise and recovery regimens allows athletes to achieve elite performance and organizations such as the International Athletes Diabetes Association help recount different experiences of success.

References

ADA (2004) Physical activity/exercise and diabetes. *Diabetes Care* 27, s58–s62.

ADA (2008) American diabetes association—nutrition recommendations and interventions for diabetes. *Diabetes Care* 31, S61–S78.

Admon, G., Weinstein, Y., Falk, B., et al. (2005) Exercise with and without an insulin pump among children and adolescents with Type 1 Diabetes Mellitus. *Pediatrics* 116, e348–e355.

Baldi, J.C., Cassuto, N.A., Foxx-Lupo, W.T., et al. (2010) Glycemic status affects cardiopulmonary exercise response in athletes with Type I diabetes. *Medicine & Science in Sports & Exercise* 42, 1454–1459.

Burke, L. & Deakin, V. (2003) *Clinical Sports Nutrition*. The McGraw-HillCompanies, Inc, Sydney, Australia.

Burke, L.M., Collier, G.R., & Hargreaves, M. (1993) Muscle glycogen storage after prolonged exercise: effect of the glycemic index of carbohydrate feedings. *Journal of Applied Physiology* 75, 1019–1023.

Burke, L.M., Kiens, B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22, 15–30.

Bussau, V.A., Ferreira, L.D., Jones, T.W., et al. (2006) The 10-s maximal sprint—a novel approach to counter an exercise-mediated fall in glycemia in individuals with type 1 diabetes. *Diabetes Care* 29, 601–606.

Bussau, V.A., Ferreira, L.D., Jones, T.W., et al. (2007) A 10-s sprint performed prior to moderate-intensity exercise prevents early post-exercise fall in glycaemia in individuals with type 1 diabetes. *Diabetologia* 50, 1815–1818.

Castaneda, C., Layne, J.E., Munoz-Orians, L., et al. (2002) A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care* 25, 2335–2341.

Chibalin, A.V., Yu, M., Ryder, J.W., et al. (2000) Exercise-induced changes in expression and activity of proteins involved in insulin signal transduction in skeletal muscle: differential effects on insulin-receptor substrates 1 and 2. *Proceedings of the National Academy of Sciences of USA*, 97, 38–43.

DAFNE (2002) Training in flexible, intensive insulin management to enable dietary freedom in people with type 1 diabetes: dose adjustment for normal eating (DAFNE) randomised controlled trial. *BMJ* 325, 746.

Deckelbaum, R.J. & Williams, C.L. (2001) Childhood obesity: the health issue. *Obesity Research* 9, 239S–243S.

Defronzo, R.A. (2004) Pathogenesis of type 2 diabetes mellitus. *Medical Clinics of North America* 88, 787–835.

Eriksson, K.F. & Lindgarde, F. (1991) Prevention of type-2 (non-insulin-dependent) diabetes-mellitus by diet and physical exercise—the 6-year malmo feasibility study. *Diabetologia* 34, 891–898.

Francescato, M.P., Geat, M., Fusi, S., et al. (2004) Carbohydrate requirement and insulin concentration during moderate exercise in type 1 diabetic patients. *Metabolism Clinical and Experimental* 53, 1126–1130.

Frohnauer, M.K., Lin, K., & Devlin, J.T. (2000) Adjustment of basal lispro insulin in CSII to minimize glycemic fluctuations caused by exercise. *Diabetes Research and Clinical Practice* 50, S80.

Frosig, C. & Richter, E.A. (2009) Improved insulin sensitivity after exercise: focus on insulin signaling. *Obesity* 17, S15–S20.

Galassetti, P., Tate, D., Neill, R.A., et al. (2003) Effect of antecedent hypoglycemia on counterregulatory responses to subsequent euglycemic exercise in type 1 diabetes. *Diabetes* 52, 1761–1769.

Gallen, I.W., Ballav, C., Lumb, A., et al. (2010) Caffeine supplementation reduces exercise induced decline in blood glucose and subsequent hypoglycaemia in adults with Type 1 Diabetes (T1DM) treated with multiple daily Insulin Injection (MDI). *Diabetes* 59, A318–A318.

Gallen, I.W., Hume, C., & Lumb, A. (2011) Fuelling the athlete with type 1 diabetes. *Diabetes Obesity & Metabolism* 13, 130–136.

Gallen, I.A., Redgrave, A., & Redgrave, S. (2003) Olympic diabetes. *Clinical Medicine* 3, 333–337.

Goff Jr, D.C., Gerstein, H.C., Ginsberg, H.N., et al. (2007) Prevention of cardiovascular disease in persons with type 2 diabetes mellitus: current knowledge and rationale for the action to control cardiovascular risk in diabetes (ACCORD) trial. *American Journal of Cardiology*, 99, 4I–20I.

Horton, E.S. (1988) Role and management of exercise in diabetes mellitus. *Diabetes Care* 11, 201–211.

Ivy, J.L. (1991) Muscle glycogen synthesis before and after exercise. *Sports Medicine (Auckland, N.Z.)* 11, 6–19.

Jensen, J. (2004) Nutritional Concerns in the diabetic athlete. *Current Sports Medicine Reports* 3, 192–197.

Li, G.W., Zhang, P., Wang, J.P., et al. (2008) The long-term effect of lifestyle interventions to prevent diabetes in the China Da Qing Diabetes Prevention Study: a 20-year follow-up study. *Lancet* 371, 1783–1789.

Lumb, A.N. & Gallen, I.W. (2010) Diabetes management for intense exercise. *Current Opinion in Endocrinology Diabetes and Obesity* 17, 150–155.

Marliss, E. B. & Vranic, M. (2002) Intense exercise has unique, effects on both insulin release and its roles in glucoregulation—implications for diabetes. *Diabetes* 51, S271–S283.

Maughan, R.J., Gleeson, M., & Greenhaff, P.L. (1997) *Biochemistry of Exercise and Training*. Oxford University Press, New York.

Moy, C.S., Songer, T.J., Laporte, R.E., et al. (1993) Insulin-dependent diabetes-mellitus, physical-activity, and death. *American Journal of Epidemiology* 137, 74–81.

Peirce, N.S. (1999) Diabetes and exercise. *British Journal of Sports Medicine* 33, 161–172.

Praet, S.F.E. & Van LOON, L.J.C. (2007) Optimizing the therapeutic benefits of exercise in type 2 diabetes. *Journal of Applied Physiology* 103, 1113–1120.

Praet, S.F., Manders, R.J., Lieverse, A.G., et al. (2006) Influence of acute exercise on hyperglycemia in insulin-treated type 2 diabetes. *Medicine & Science in Sports & Exercise* 38, 2037–2044.

Ramachandran, A., Snehalatha, C., Mary, S., et al. (2006) The Indian Diabetes Prevention Programme shows that lifestyle modification and metformin prevent type 2 diabetes in Asian Indian subjects with impaired glucose tolerance (IDPP-1). *Diabetologia* 49, 289–297.

Robertson, R.P., Halter, J.B., & Porte, D. (1976) Role for alpha-adrenergic receptors in abnormal insulin-secretion in diabetes-mellitus. *Journal of Clinical Investigation* 57, 791–795.

Schenk, S. & Horowitz, J.F. (2007) Acute exercise increases triglyceride synthesis in skeletal muscle and prevents fatty acid-induced insulin resistance. *Journal of Clinical Investigation* 117, 1690–1698.

Sieverdes, J.C., Sui, X.M., Lee, D.C., et al. (2010) Physical activity, cardiorespiratory fitness and the incidence of type 2 diabetes in a prospective study of men. *British Journal of Sports Medicine* 44, 238–244.

Tattersall, R.B. (1995) A force of magical activity—the introduction of insulin-treatment in Britain 1922–1926. *Diabetic Medicine* 12, 739–755.

Wallberg-Henriksson, H., Rincon, J., & Zierath, J.R. (1998) Exercise in the management of non-insulin-dependent diabetes mellitus. *Sports Medicine* 25, 25–35.

WHO. (2010) *Diabetes Programme.* Available at: http://www.who.int/diabetes/en/ (accessed December 23, 2011).

WHO. (2011) *Diabetes.* Fact Sheet No. 312 (updated March 2013). Available at: http://www.who.int/mediacentre/factsheets/fs312/en/index.html (accessed May 29, 2013).

Willett, W., Manson, J., & Liu, S. (2002) Glycemic index, glycemic load, and risk of type 2 diabetes. *American Journal of Clinical Nutrition* 76, 274S–280S.

Zisman, A., Peroni, O.D., Abel, E.D., et al. (2000) Targeted disruption of the glucose transporter 4 selectively in muscle causes insulin resistance and glucose intolerance. *Nature Medicine* 6, 924–928.

Chapter 41

The Overweight Athlete

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Introduction

The description of an elite athlete as "overweight" may at first seem a misnomer. Few, if any, elite athletes have a weight or level of adiposity placing them at a risk of ill-health. However, many athletes are concerned about the effect of weight, body composition, and physique on performance. The importance of morphology to athletic performance is at one level obvious, with those in sports such as throwing, heavyweight boxing, or rowing, having pronounced muscular development to optimize strength and power while those in distance running, rhythmic gymnastics, and high jump being slender to obtain energy cost, aesthetic or biomechanical benefit. Justification for the contribution of specific physique characteristics to athletic success is often limited or absent from the scientific literature. At the elite level, competition success is decided by incredibly small margins and a range of physiological, psychological, and sport-specific factors. Designing studies sensitive and powerful enough to capture the independent effect of subtle weight or physique changes on performance is challenging through to almost impossible for some sports. Despite this, support for the importance of physique to athletic performance can be gleaned from longitudinal data on successful athletes, competition gradients within sports, and modeling or comparison of metabolic, thermoregulatory, and biomechanical advantages in athletes with differing morphology (Norton & Olds, 2001).

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This chapter will focus briefly on the available evidence supporting the relevance of attaining a desirable weight or body composition for optimizing sports performance and why this may be challenging for athletes. Dietary strategies for assisting athletes to safely manipulate weight and body composition will then be examined. Specific issues associated with making weight will be covered in Chapter 53.

Weight, Physique Attributes, and Athletic Performance

Morphological attributes generally considered important to sports performance include body mass or weight, stature, and skeletal muscle or fat mass (Khosla & McBroom, 1988). Other features such as limb or segment lengths and frame size may be important for specific sports. The reason these characteristics are considered important can generally be attributed to one or more of the following factors: reduction of energy or metabolic cost, thermoregulatory benefits, enhancement of strength, power or power-to-weight ratio, and a range of biomechanical factors (e.g., reach or stride length). In some sports, weight and physique are important for aesthetic reasons (O'Connor & Slater, 2011).

Evidence for the importance of low body mass (and short stature) in endurance running is supported by historical data through the twentieth century (Figure 41.1). The data show that mass and stature of successful marathon runners have remained relatively static over many decades, indicating these physique characteristics are success critical to this event at the elite level. Additional evidence in

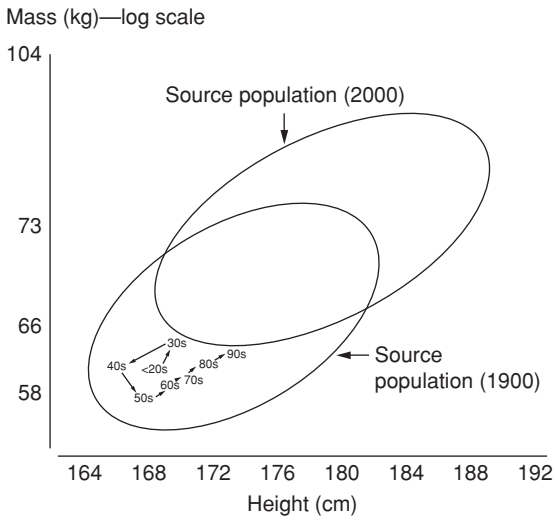

Figure 41.1 Bivariate distribution of height (cm) and mass (kg) for elite marathoners and the source population. The ellipses are 90% density ellipses for both the marathoners and the source population. Mass values are plotted on a log scale. Source: Professor Timothy Olds, University of South Australia.

distance running also comes from data demonstrating lower weight as athletic caliber increases. This observational information is supported by laboratory and field data confirming that lower body mass and fatness decrease the energy cost of running, and result in lower heat production and improved thermoregulation (O'Connor et al., 2007).

Reducing total mass, especially fat mass which is considered as "dead weight," increases power-to-weight ratio, which is important for optimum performance in many sports (e.g., jumping, gymnastics, light weight rowing, or ski jumping), especially those where athletes succeed by moving body mass further or more rapidly through space, overcoming gravitational, wind, or water resistance (O'Connor & Slater, 2011). Even when leanness is optimized, excessive skeletal muscle may render the athlete "overweight." Although theoretically providing increased power and strength, the additional lean mass may fail to deliver superior functional benefits for performance. This is likely due to increased energy cost of locomotion, less effective thermoregulation, or potentially a limitation on agility or start dynamics in "stop and go" team sports or in sprinting (see Chapter 46).

Evidence for the existence of a bias toward low mass and leanness in aesthetic sports is more difficult to justify using the scientific literature. Although points for weight or physique are not allocated for any Olympic sport, the athlete physique may sway the "trained eye" of the judge and influence scores for artistic impression. It has been argued that extremely low body mass and fat in some sports (e.g., rhythmic gymnastics) is driven primarily by appearance rather than performance enhancement. Unfortunately, physique ideals in some sports are often entrenched and difficult to change. An additional pressure on athletes in recent years also comes from benefits derived from media or sponsorship opportunities which appear to be influenced by athlete physical attractiveness, including the societal desirability of leanness (O'Connor & Caterson, 2010).

Compared with other physique characteristics such as stature, limb lengths, or bone breadths, body mass, particularly skeletal muscle, fat, and water components are relatively amenable to change. Although diet and training can significantly alter body mass and composition, genetic factors ultimately underpin the limit to which these characteristics can be manipulated.

Challenges Influencing Manipulation of Weight and Body Composition

Genetics

Genes are known to influence phenotype via many body systems including, but not limited to, energy and fuel metabolism, synthetic pathways, and behavioral characteristics (e.g., appetite, physical activity). A detailed discussion of the genetic influence on phenotype is beyond the scope of this chapter but excellent reviews are available (Bray et al., 2009; O'Rahilly et al., 2003). Numerous genes have been identified as influencing physique, and the interaction of these with environmental factors ultimately determines phenotype. Genetic factors, however, explain why athletes following similar diet and training programs respond differently or at varied rates to these interventions. Some athletes and coaches may be tempted to engage in extreme

diet or training regimens in the pursuit of weight or physique goals that are out of reach. Sometimes goals can be achieved but only with chronic energy restriction or low energy availability which has serious negative physical and psychological effects (see Chapter 5). It is important for athletes and coaches to accept that there are limits to the plasticity of body mass and composition and to seek diet and training strategies that are safe and maintain athlete health.

Athlete Environmental Challenges

Athletes in Western and increasingly in developing countries are exposed to an obesogenic environment. This challenges healthy eating practices and increases the risk of undesirable weight or fat gain. Dietary changes required for weight or fat loss can also be challenging when an athlete is consistently exposed to inappropriate food choices and under time pressure to incorporate training, work/study, family, and social commitments. Athletes living in student accommodation or away from family support may struggle to find suitable choices on dormitory menus or to purchase and prepare a healthy diet. Food preparation skills or financial constraints may especially be an issue when living away from home (Heaney et al., 2008).

Traveling to competition further challenges dietary goals as athletes may have difficulty in finding appropriate choices at competition venues or airport lounges or in selecting unfamiliar foods in foreign countries (see Chapter 34). The wide range of food choice at competition dining halls is also a significant temptation for athletes. Despite this time being critical for optimizing dietary intake, the athlete dining hall can derail preplanned weight management strategies and compromise physique goals (see Chapter 36). Finally, exposure to widespread misinformation on weight management through the Internet and mass media as well as via athlete networks provides confusing and potentially dangerous, but often alluring, guarantees of rapid weight or fat loss. Unfortunately, results are often exaggerated and negative side effects underreported. Athletes desperate to reduce weight or fat are vulnerable to these "quick fix" diets. Even if effective for short-term weight or fat loss, these programs typically fail to adequately provide for the specific energy and fuel needs of training (O'Connor & Caterson, 2010).

Growth and Pubertal Considerations

A relatively rapid change in physique occurs during puberty. Weight increases, with a greater accrual of lean mass relative to fat in boys than in girls (see Chapter 29). These physique changes can develop quite suddenly in some athletes and even if it is eventually beneficial to be taller or more muscular, it usually takes time for technical skills to adapt and performance to improve. Early puberty can be an advantage in strength and power sports, as competitive sport during adolescence is typically contested by chronological age. The increased fat mass in female athletes can be a concern in sports where extreme leanness is desirable (e.g., gymnastics, diving, figure skating, endurance running). Increasing adiposity is a normal and expected transition into adulthood, but it often initiates restricted dieting and disordered eating which may delay growth, maturation, and have negative health and performance consequences, especially in girls (see Chapters 28 and 42). During adolescence, greater independence and a preference for energy-dense, high-fat/sugar foods, often consumed at fast food outlets, can also challenge weight and physique goals as can the onset of alcohol intake which typically occurs in late adolescence, sometimes associated with the "peer bonding" experience (Mays et al., 2010).

Training Considerations

Weight management programs for athletes need to consider energy and fuel needs for training. Body weight, fat, or lean mass targets need to be achieved without compromising training and performance. An understanding of athlete energy expenditure is therefore essential, but this is challenging because of the variability from day to day, especially when training is periodized (Byrne et al., 2011). Other factors such as pre-competition tapers, the "off season" in some sports, injury, or illness can also dramatically and often abruptly reduce energy requirements. Athletes may also compensate for

high energy expenditures during heavy training periods by reducing habitual energy expenditure or including a daily nap or extending nocturnal sleep. Growth spurts in adolescence can, on the contrary, significantly increase energy requirements (O'Connor & Caterson, 2010).

Evidence also suggests that human appetite is not tightly coupled to expenditure in the short term, and some individuals possess the capacity to more closely match intake to energy needs than others (King et al., 1994). Matching energy intake to requirements also appears to be influenced by the intensity, duration, and modality of exercise, with intense exercise more likely to blunt appetite. Exposure to cold (i.e., swimming in cold water) may increase appetite. Studies demonstrate that post exercise intake can result in partial, complete, and even overcompensation of the energy expended. Gender may also play a role with evidence of greater compensation of energy after exercise in women than men (King et al., 2007). Maintenance of energy reserves in women for reproductive purposes may underpin this finding.

The fuel mix oxidized has also been reported to influence postexercise consumption, with a higher rate of carbohydrate oxidation during exercise hypothesized to drive carbohydrate intake. Often this comes with additional, and possibly excessive, fat intake via passive overconsumption when athletes access palatable meals or snacks (Tremblay et al., 1985). While there is some evidence of closer matching in those who habitually exercise (King et al., 2007), the effect of exercise on appetite, especially in highly trained athletes, is largely unexplored. In practice, the complex interplay of these factors likely contributes to individual variability in the ability of athletes to regulate appetite and achieve energy balance.

Strategies for Weight and Fat Loss

Prescription of Energy Intake

Regardless of the approach used, body weight or fat will be lost only if energy intake is less than expenditure. Additional aerobic training is often incorporated into athlete programs to increase energy expenditure, but the time required to develop technique and skill is usually substantial, so too much additional training risks athlete fatigue, delayed recovery, and possibly injury (see Exercise-Related Strategies to Promote Weight and Fat Loss). Reduction of energy intake usually also results in faster results and often, overall improvement in the food choices made by athletes are sufficient to induce the required energy deficit. Energy deficits should be targeted as mild to moderate, which equates to a 10–20% reduction from the energy intake required to maintain energy balance (American College of Sports Medicine, 2009). Gradual reduction of energy intake has a less dramatic effect on hunger and helps to identify the minimum level of restriction required to elicit desired results. Guidance from a sports dietitian or nutritionist is valuable not only for the development of an eating plan which optimizes and periodizes nutrient intake to compliment the needs of training but also for continued monitoring and adjustment of the plan in response to weight or fat losses, performance results, and athlete health.

Unfortunately, athletes and coaches may impose a severe energy restriction often via use of "fad" or popular diets to obtain rapid results. This tends to increase the risk of inadequate nutrient consumption (Williams & Williams, 2003), fatigue, delayed recovery, energy deficiency, and overtraining syndrome (see Chapters 5 and 33). Severe energy restriction may also result in lean mass loss or compromised accrual of training benefits (see Chapter 10). This approach is also more difficult to maintain in the longer term and athletes may fall into a pattern of erratic restrictive dieting or repeated weight cycling which should be avoided as it is known to have a negative impact on psychological wellbeing and may be associated with adverse health risks including obesity in later life (Saarni et al., 2006; see also Chapter 53).

Strict, chronic energy restriction is more common in sports where extreme leanness is desirable (e.g., gymnastics, figure skating, endurance running, diving) and although an issue for some male athletes, naturally higher levels of body fatness generally make the attainment of such leanness more challenging for females (see Chapter 28). Gender aside, some athletes still appear to achieve the desired

weight or fat relatively easily while others struggle. This may be due to a range of factors, but as athletes participating in skill-/aesthetic-based sports (e.g., gymnastics, diving, figure skating) have a lower capacity for fat oxidation than endurance athletes and expend substantially less energy in training, they may need to rely more on restriction of energy intake (see Chapters 12 and 49). Finally, as previously mentioned, there is a strong influence of genes but also the caliber of support available to the athlete. An athlete should not be told to restrict energy intake to induce weight or fat loss without appropriate health professional assessment, guidance, monitoring, and support.

Practically, accurate estimation of energy requirements is challenging, even with tools such as accelerometers and global positioning system monitors (Byrne et al., 2011). Although a number of useful equations and resources exist to guide estimation of both resting and activity energy requirements (Manore & Thompson, 2010), a prescription for energy intake based solely on theoretical estimates rather than an individualized, athlete-tailored approach is likely to be less successful. Information from athlete food histories, diaries, and weight loss experience is extremely useful. Although food intake may be underreported (Barnard et al., 2002), information on eating styles and food beliefs can be used together with the training program and theoretical requirements to develop an eating plan that is appropriate and acceptable to the athlete's physical needs and personal preferences. Apparent underreporting can be assessed and also provides an opportunity to probe the existence of energy deficiency (see Chapter 5) or nondisclosure of intake due to either high or erratic energy intakes (Barnard et al., 2002) or body image, behavioral/self-esteem issues. Variability in energy requirements due to periodization can be managed via the development of a "core" plan for days with little to no training and guidance for larger or additional meals or snacks timed around the suite of training sessions. This approach helps athletes to understand the need to reduce food intake when not training, injured, or on a taper but also to consume foods with the appropriate energy and macronutrient content to support activity on training days.

Low-Fat, High-Carbohydrate Diets for Weight and Fat Loss

The optimum macronutrient distribution for weight or fat loss is currently under extensive investigation in the obesity literature. Historically, the pendulum has swung toward lower and then higher carbohydrate diets over the last 40 years with moderate carbohydrate, higher protein diets currently supported by emerging research (Larsen et al., 2010). This has evolved from the 1990s where *ad libitum* high-carbohydrate, low-fat diets were popular and widely recommended. At this time, the contribution of carbohydrate (even when consumed in excess) to weight and fat gain via *de novo* lipogenesis was considered insignificant and self-limiting due to the energy cost of lipogenesis. Carbohydrate intake was considered to be tightly regulated due to limited body stores with ingestion and appetite linked to its replenishment. The well-established greater energy density of fat than carbohydrate, together with epidemiological evidence linking fat rather than energy intake to obesity prevalence, supported this high-carbohydrate, low-fat diet approach to weight management. Athletes were also influenced by this eating style which was consistent with sport nutrition recommendations for supporting optimum athletic performance (O'Connor & Caterson, 2010).

While *ad libitum* low-fat, high-carbohydrate diets consistently promoted modest weight loss, the overall results, even in the obese, were disappointing (~3 to 4 kg over 3 months) (Astrup et al., 2000). Wide dissemination of this diet prescription also failed to curtail obesity prevalence which continued to increase worldwide. Moderate weight loss, together with concerns about the type and amount of carbohydrate, particularly high fructose corn syrup used in many low-fat products (and athlete sports foods) eventually resulted in disillusionment with this approach by the late 1990s in both the general population and athletes. Despite this, for athletes who have relatively high energy expenditures and require only modest weight or fat loss, a simple focus of reducing fat intake (particularly in those consuming high-fat diets) usually results in satisfactory results without the need for systematic energy restriction. This strategy is also easy for athletes to learn and implement,

with carbohydrate and nutrient reserves well maintained and excessive hunger avoided. Guidance toward a diet that is low in fat and has an adequate, rather than unlimited, carbohydrate content is therefore a useful approach for weight management in many athletes (O'Connor & Caterson, 2010).

Reduced-Carbohydrate, Higher-Protein Diets

By the early 2000s, reduced-carbohydrate, higher-protein diets (e.g., Atkins, South Beach, Zone) had become popular in the general community and with athletes. Low-carbohydrate, high-protein diets consistently appear to result in faster weight and fat loss across the first 3–6 months in obese individuals, but thereafter weight is usually regained (Sacks et al., 2009). Immediate weight (as opposed to fat) loss results from glycogen depletion and associated water loss. However, other mechanisms hypothesized to explain the short- to medium-term efficacy of these diets include the reduction of glycemic load, decreased insulin secretion, and ketosis (O'Connor & Caterson, 2010). Insulin inhibits fat oxidation and ketosis and low-glycemic index (GI) diets (low in carbohydrate and glycemic index) minimize insulin secretion. As a result, fat oxidation and utilization is enhanced. A lower total energy consumption is also consistently observed on these diets and has been attributed to a higher protein intake which is known to enhance satiety (McMillan-Price & Brand-Miller, 2006). Novelty, simplicity, and monotony (limited food variety) (Stubbs et al., 2001) may also explain some of the benefits. Studies examining the relative effect of ketosis report that it is not associated with greater fat loss (Sacks et al., 2009).

Despite their popularity, low-carbohydrate diets are no better at maintaining weight/fat loss in obese participants than balanced macronutrient diets in the longer term (Sacks et al., 2009). There is insufficient evaluation of these diets in athletes, but concerns regarding the longer-term safety of low-carbohydrate diets also warrant consideration. Chronic ketosis is potentially harmful, and is associated with hyperlipidemia, optic neuropathy, osteoporosis, and altered cognitive function. There is also a risk of nutrient inadequacy (especially for fiber, vitamins B1, B2, C, for calcium and magnesium, and phytochemicals) primarily due to the limitation of fruit, starchy vegetables, grains, and dairy foods. Inadequate carbohydrate intake also decreases muscle glycogen stores and has been shown to impair high-intensity training, immune function, and recovery (see Chapter 39). The effect of low-carbohydrate diets on lean mass in obese participants is equivocal but is potentially detrimental in athletes (O'Connor & Caterson, 2010).

Obesity researchers have recently turned to a more moderate approach using higher protein, moderate carbohydrate diets. While not always resulting in superior weight loss, the reduction in weight and fat is generally comparable to or better than the more traditional high-carbohydrate approach. A large, multicenter study of over 700 participants using a higher protein (25% of energy), moderate carbohydrate, low GI approach showed that this combined strategy was more successful at maintaining weight loss than a higher GI carbohydrate, higher protein or lower GI carbohydrate, lower protein diet (Larsen et al., 2010). The combination of the higher protein, low-GI diet was considered to enhance satiety and improve adherence.

The protein leverage hypothesis (PLH) proposes that dilution of the ratio of dietary protein to carbohydrate or fat contributes to increased energy intake and consequent weight or fat gain (Simpson & Raubenheimer, 2005). A protein leverage effect on the regulation of energy intake in the diets of human and numerous other species has been reported (Raubenheimer & Simpson, 1997). Energy consumption is suggested to be adjusted to reach a "target" ingestion of protein independent of carbohydrate and fat; in the typical western diet, this leads to passive overconsumption of these macronutrients (Simpson & Raubenheimer, 2005). As protein typically provides a significantly lower proportion of dietary energy than either carbohydrate or fat, a small decrease in the percentage of dietary protein potentially drives additional food intake until target protein requirements are satisfied. Although the effect of exercise on protein leverage in humans is largely unexplored, increased protein requirements in athletes (see Chapter 10) possibly elevate the target. Athletes with a dietary focus on carbohydrate (or fat) at the expense of protein may be driven to consume additional

energy to satisfy protein targets. However, further research is required to confirm the PLH through additional studies in humans and subsequently, if warranted, to determine how protein leverage may be best exploited to assist weight management.

Finally, there is a paucity of weight management research in athletes, so specific benefits of macronutrient manipulation can be inferred only from the obesity literature. Athletes are clearly different from obese individuals not only from a metabolic but also from a nutritional and exercise behavior standpoint. Early performance enhancement following weight or fat loss from any restrictive diet needs to be balanced alongside the potential detrimental effects. In practice, athletes attempting to reduce body mass or fat more often adopt certain aspects of popular diet programs; with low-carbohydrate diets this is typically an increase in protein and a modest reduction in carbohydrate intake and possibly low GI rather than extreme carbohydrate restriction. Unfortunately, popular diet trends travel rapidly through athlete networks, and pose a risk to younger athletes who often have inadequate access to sport nutrition support.

Manipulation of Energy Density

A further consideration when preparing meal plans for weight management is energy density (energy content per gram of food). Athletes are often unaware of the energy density of foods and that high energy density is linked with passive overconsumption and fat gain (Rolls, 2000). Sports foods and beverages by design are energy-dense but often not micronutrient-dense. They provide a convenient and compact source of fuel to consume when energy or macronutrient needs are difficult to meet, especially during long training sessions or competition. Decreasing energy density by incorporating high-fiber foods and being strategic about use of energy-dense sports foods is recommended, especially for athletes on a tight energy budget.

Low-GI, High-Fiber Diets and Gut Microbiota

Low-GI diets enhance satiety (McMillan-Price & Brand-Miller, 2006). A recent systematic review evaluating the efficacy of the low-GI approach to obesity management reported a small positive effect for promoting weight loss (Thomas et al., 2009). Low-GI diets are associated with positive health consequences although the impact on glycogen stores and athlete performance from chronic use has not been evaluated. As with low GI, high-fiber diets are also associated with enhanced satiety and positive health benefits (Howarth et al., 2001). Many (but not all) high-fiber foods are also low in GI. High-fiber, low-GI meal plans are likely to be especially helpful for athletes who struggle with increased hunger when energy intake is restricted. Strategic use of higher-fiber, low-GI, and protein-containing snacks between meals may also be beneficial in reducing hunger at main meals.

Emerging evidence also supports the role of gut microbiota in the regulation of body weight (Greiner & Backhed, 2011; Tilg & Kaser, 2011). Although complex and in need of further research, the Western diet is high in fat and GI and low in fiber and may not promote optimum gut flora; this may influence energy extraction, transport of nutrients, and systemic inflammation which play a role in energy balance and metabolism. We extract most, but not all, of the energy we consume in the diet. Gut microbes appear to change this and a poor profile may increase the fraction that is digested and absorbed and increase risk of weight gain. As yet there is insufficient information to direct a specific dietary approach for improved weight management.

Dairy Consumption and Weight Management

The role of dairy consumption in regulation of body weight or fat has been extensively investigated. A number of large population studies support an inverse association between diary intake and body mass index (Major et al., 2008). However, evidence of a benefit from dairy product consumption on weight/fat loss has been conflicting. A recent systematic review and meta-analysis (Abargouei et al., 2012) examining the effect of dairy consumption on weight and body composition concluded that increased dairy intake (range of 550–1000 mg/day) was beneficial for reduction in body weight, body fat content, and waist circumference and retention of lean mass only when total daily energy intake

was restricted (Abargouei et al., 2012). Plausible biological mechanisms including the effect of dairy calcium on adipocyte lipid metabolism, fatty acid absorption, and post-prandial fat metabolism have been proposed. Other dairy constituents, including conjugated linoleic acid, medium chain triglycerides, and protein, may also play a positive metabolic role, while protein and calcium may exert a beneficial effect on appetite control (Dougkas et al., 2011). As adequate intake of dairy and the nutrients it provides is important for good health and bone development, particularly for achievement of peak bone mass (see Chapter 20), inclusion of dairy foods (even when fat-reduced) in energy-restricted weight management plans would seem beneficial.

Commercial Weight Loss and Meal Replacement Programs

A number of nutritionally sound, commercial weight loss programs are available in the community. The meal plans provided by these programs are designed primarily for overweight, relatively sedentary individuals, rather than for elite athletes. Some commercial programs offer adapted guidance for active persons but the staff typically possess limited sports nutrition/dietetic expertise to tailor eating programs for elite athletes. A benefit associated with some commercial programs or individual counseling from a dietitian or psychologist includes the incorporation of behavior modification techniques which have been clearly shown to enhance weight/fat loss in the obese both in the short and long term (O'Connor & Caterson, 2010). Comprehensive dietary and behavior modification guidance designed to address the athlete's weight management and sports nutrition needs from a skilled health professional would be considered best practice.

Meal replacement diets aim to reduce energy intake through meal substitution. These can range from a menu of energy-controlled foods, meals, and snacks through to liquid meals designed to totally or partially replace food consumption. This approach may appear attractive as it removes some or all of the discretionary food choice and along with this, most or all of the food preparation. The programs aim to provide products which are both palatable and satiating. They are designed primarily for sedentary to moderately active overweight or obese individuals, so the energy and nutritional profile of the meals is not adequate for elite athletes. These programs are typically a short- to medium-term weight management option as they tend to become boring and repetitive. Extreme versions (e.g., very low-energy diets) are suited only to the very obese under medical supervision and are not recommended for use in athletes (O'Connor & Slater, 2011).

Exercise-Related Strategies to Promote Weight and Fat Loss

Any diet program introduced for athlete weight management needs to ensure that sufficient energy and nutrients are consumed to sustain and optimize training. Cross-training is often used not only to develop or extend physiological attributes but also to support attainment of physique goals. Low intensity, aerobic exercise is usually incorporated to assist weight or fat loss as it increases energy expenditure and promotes adaptations that enhance fat oxidation (see Chapter 12). However, although the proportion of fat oxidized is greater at low to moderate exercise intensities, in well-trained athletes, substantial proportions (and often greater amounts) of fat can be oxidized at moderate–higher exercise intensities (below anaerobic threshold) as the total energy expenditure is higher (Romijn et al., 1993). Some athletes also attempt to achieve improved fat oxidation though "train low" or "fasted exercise" (Stannard et al., 2010). Training in a fasted or glycogen-depleted state needs to be undertaken with care and is not recommended for developing athletes (see Chapter 13).

Exercise intensity may also influence appetite regulation (King et al., 1994). Unexpected greater fat loss after higher intensity exercise training programs has been reported (Knechtle et al., 2008; Trapp et al., 2008), even when the training energy expenditure was approximately half that of a comparison group performing low-intensity exercise (Tremblay et al., 1994). There are a wide range of considerations for cross-training but it should be

carefully programmed to avoid overtraining and injury and tailored to the needs of individual athletes. Anecdotal evidence suggests that some exercise modalities (e.g., running) are more effective for fat loss than others (e.g., swimming) although this has not been clearly established in the literature. High-impact modalities such as running may in particular increase the risk of injury.

Clinical and Practical Aspects for Weight Management in Athletes

Although dieting and losing weight appear to be commonplace in the community, the recommendation for an athlete to undertake weight or fat loss should be given serious consideration. Restrictive diets may result in a range of negative consequences for athletes, including inadequate nutrient intake/deficiency, decreased immunocompetence (see Chapter 39), or a pathway toward disordered eating (see Chapter 42). This can even be the case when an athlete is provided with professional advice and support. Energy restriction increases risk for energy deficiency and this may result in menstrual dysfunction and osteopenia in female athletes. Osteopenia increases the risk for stress fractures which at a minimum will result in some training interruption through to recurrent incidence which can be career ending. Reestablishment of bone integrity may not be possible and predisposes the athlete to osteoporosis in later life (see Chapters 5 and 20).

In athletes where there a clear benefit of lower body weight and fat for competition, this can often be periodized to minimize the time and extent of restriction. Delaying the more restrictive aspects of weight management in younger, developing athletes until they have moved closer to peak bone mass and are more mature and better able to cope with both the physical and psychological risks is recommended. If rigorous dieting is required at an early age, consideration should also be given to whether the athlete possesses the right genetic predisposition to attain and then maintain the desired physique goal. Monitoring athlete progress is also critical from both a physical (including physique assessment, see Chapter 6) and psychological perspective. Although athletes are often interested in supplements which will facilitate weight or fat loss there is limited evidence of efficacy (see Chapter 23).

References

Abargouei, A.S., Janghorbani, M., Salehi-Marzijarani, M., et al. (2012) Effect of dairy consumption on body composition in adults: a systematic review and meta-analysis of randomized controlled trials. *International Journal of Obesity* 36, 1485–1493.

American College of Sports Medicine (2009) Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41, 709–731.

Astrup, A., Grunwald, G.K., Melanson, E.L., et al. (2000) The role of low fat diets in body weight control: a meta-analysis of ad libitum dietary intervention studies. *International Journal of Obesity* 24, 1545–1552.

Barnard, J.A., Tapsell, L.C., Davies, P.S.W., et al. (2002) Relationship of high energy expenditure and variation in dietary energy with reporting accuracy on 7 day food records and diet history in a group of healthy adult volunteers. *European Journal of Clinical Nutrition* 56(4), 358–367.

Bray, S., Hagberg, M.J., Pérusse, L., et al. (2009) The human gene map for performance and health-related phenotypes. The 2006–2007 update. *Medicine & Science in Sports & Exercise* 41(1), 35–72.

Byrne, N.M., Shultz, S.P., & Hills, A.P. (2011) Field assessment of physical activity and energy expenditure among athletes. In *Nutritional Assessment of Athletes*, 2nd edn (eds. J.A. Driskell & I. Wolinsky), pp. 183–212. CRC Press, Boca Raton, FL.

Dougkas, A., Reynolds, C.K., Givens, I.D., et al. (2011) Associations between dairy consumption and body weight: a review of the evidence and underlying mechanisms. *Nutrition Research Reviews* 15, 1–24.

Greiner, T. & Bäckhed, F. (2011) Effects of gut microbiota on obesity and glucose homeostasis. *Trends in Endocrinology and Metabolism* 22(4), 117–123.

Heaney, S., O'Connor, H., Naughton, J., et al. (2008) Towards an understanding of the barriers to good nutrition for elite athletes. *International Journal of Sports Science Coaching* 3(3), 391–401.

Howarth, N.C., Saltzman, E., & Roberts, S.B. (2001) Dietary fiber and weight regulation. *Nutrition Reviews* 59, 129–139.

Khosla, T. & McBroom, V.C. (1988) Age, height and weight of female Olympic finalists. *British Journal of Sports Medicine* 9, 96–99.

King, N.A., Burley, V.J., & Blundell, J.E. (1994) Exercise-induced suppression of appetite: effects on food intake and implications for energy balance. *European Journal of Clinical Nutrition* 48, 715–724.

King, N.A., Cauldwell, P., Hopkins, M., et al. (2007) Metabolic and behavioral compensatory responses to exercise interventions: barriers to weight loss. *Obesity* 15, 1373–1383.

Knechtle, B., Schwanke, M., Knechtle, P., et al. (2008) Decrease in body fat during an ultra endurance triathlon is associated with race intensity. *British Journal of Sports Medicine* 42, 609–613.

Larsen, T.M., Dalskov, S., van Baak, M., et al. (2010) Diets with high or low protein content and glycemic index for weight-loss maintenance. *New England Journal of Medicine* 363, 2102–2113.

Major, G.C., Chaput, J.-P., Ledoux, M., et al. (2008) Recent developments in calcium related obesity research. *Obesity Reviews* 9, 428–445.

Manore, M. & Thompson, J. (2010) Energy requirements of the athlete: assessment and evidence of energy efficiency. In *Clinical Sports Nutrition*, 4th edn (eds. L. Burke & V. Deakin), pp. 98–115. McGraw-Hill, Sydney, Australia.

Mays, D., DePadila, L., Thompson, N.J., et al. (2010) Sports participation and problem alcohol use. A multi-wave national sample of adolescents. *American Journal of Preventive Medicine* 35(5), 491–498.

McMillan-Price, J. & Brand-Miller, J. (2006) Low glycemic index diets and body weight regulation. *International Journal of Obesity* 30, S40–S46.

Norton, K. & Olds, T. (2001) Morphological evolution of athletes over the twentieth century: causes and consequences. *Sports Medicine* 31(11), 763–783.

O'Connor, H. & Caterson, I. (2010) Weight loss and the athlete. In *Clinical Sports Nutrition*, 4th edn (eds. L. Burke & V. Deakin), pp. 116–148. McGraw-Hill, Sydney, Australia.

O'Connor, H. & Slater, G. (2011) Losing, gaining and making weight for athletes. In *Sport and Exercise Nutrition* (eds. S. Lanham-New, S. Stear, M. Sherriffs, et al.), pp. 210–232. Wiley-Blackwell, West Sussex, UK.

O'Connor, H.T., Olds, T., & Maughan, R. (2007) Physique and performance for track and field. *Journal of Sport Science* 25(Suppl. 1), S49–S60.

O'Rahilly, S., Farooqi, S., Yeo, G.S.H., et al. (2003) Minireview: human obesity—lessons from monogenic disorders. *Endocrinology* 144, 3757–3764.

Raubenheimer, D. & Simpson, S.J. (1997) Integrative models of nutrient balancing: application to insects and vertebrates. *Nutrition Research Reviews* 10, 151–179.

Rolls, B.J. (2000) The role of energy density in overconsumption of fat. *Journal of Nutrition* 130, 268S–271S.

Romijn, J.A., Coyle, E.F., Sidossis, L., et al. (1993) Regulation of endogenous fat and carbohydrate metabolism in relation to exercise intensity. *American Journal of Physiology* 265, E380–E391.

Saarni, S.E, Rissaen, A., Sarna, S., et al. (2006) Weight cycling of athletes and subsequent weight gain in middle age. *International Journal of Obesity* 30, 1639–1644.

Sacks, F.M., Bray, G.A., Carey, V.J., et al. (2009) Comparison of weight loss diets with different compositions of fat, protein and carbohydrates. *New England Journal of Medicine* 360, 859–873.

Simpson, S.J. & Raubenheimer, D. (2005) Obesity: the protein leverage hypothesis. *Obesity Reviews* 6, 133–142.

Stannard, S.R., Buckley, A.J., Edge, J.A., et al. (2010) Adaptations to skeletal muscle with endurance exercise training in the acutely fed versus overnight fasted state. *Australian Journal of Science and Medicine in Sport* 13, 465–469.

Stubbs, R.J., Johnstone, A.M., Mazlan, N., et al. (2001) Effect of altering the variety of sensorially distinct foods, of the same macronutrient content, on food intake and body weight in men. *European Journal of Clinical Nutrition* 55, 19–28.

Thomas, D., Elliott, E.J., & Baur, L. (2009) Low glycemic index or low glycemic load diets for overweight and obesity. John Wiley & Sons, Ltd, Chichester, West Sussex.

Tilg, H. & Kaser, A. (2011) Gut microbiome, obesity and metabolic dysfunction. *Journal of Clinical Investigation* 121(6), 2126–2132.

Trapp, E.G., Chisholm, D.J., Freund, J., et al. (2008) The effects of high-intensity intermittent exercise training on fat loss and fasting insulin levels of young women. *International Journal of Obesity* 32, 684–691.

Tremblay, A., Déspres, J.-P., & Bouchard, C. (1985) The effects of exercise-training on energy balance and adipose tissue morphology and metabolism. *Sports Medicine* 2, 223–233.

Tremblay, A., Simoneau, J.A., & Bouchard, C. (1994) Impact of exercise intensity on body fatness and skeletal muscle metabolism. *Metabolism* 43, 814–818.

Williams, L. & Williams, P. (2003) Evaluation of a tool for rating popular diet books. *Nutrition and Dietetics* 60, 185–197.

Chapter 42

Eating Disorders in Male and Female Athletes

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Introduction

The components for success in competition for both male and female athletes have been spread by the motto of the modern Olympic Games: "Citius, Altius, Fortius," translated into "Swifter, Higher, Stronger." Each of these components is based on the athlete's capacity for cardiorespiratory and muscular endurance, muscle strength, power, flexibility, agility, speed, and mental strength. However, for some physically active males and females, rigorous exercise and a competitive lifestyle may expose them to some threats to their health and well-being. Thus, participation in sports does not produce only an array of health benefits. Circumstances that may adversely affect the athlete's short- and long-term health include, e.g., low energy availability, disordered eating (DE) behaviors, and eating disorders (EDs). These conditions were first identified in female athletes, but research has shown that male athletes are also susceptible to the same challenges (Glazer, 2008).

In the following sections, we will define the DE spectrum and describe the prevalence of EDs among female and male athletes. Thereafter, we will discuss the etiology of EDs among athletes followed by a description of health- and performance-related consequences of EDs. Finally, we will discuss the possibilities of prevention, as well as management, of EDs among athletes.

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Definitions of the DE Spectrum

Among athletes, a spectrum of DE behaviors, like preoccupation with body weight and shape, food restriction, dieting, binge eating, vomiting, and abuse of diuretics, laxatives, and diet pills, seems to exist (Sundgot-Borgen & Torstveit, 2010). The purpose of DE behavior is usually to achieve a low body weight to compensate for strong body dissatisfaction (American Psychiatric Association [APA], 1994). Athletes with DE often feel continuously fat, and the DE behaviors may become more intensified, to a degree where the athlete meets the criteria for a clinical ED (Sundgot-Borgen, 1994).

The clinical EDs include anorexia nervosa (AN), bulimia nervosa (BN), and eating disorder not otherwise specified (EDNOS) (APA, 2000).

When the first prevalence studies showed the presence of EDs among athletes, it was common to classify EDs into only AN and BN. Due to the fact that athletes constitute a unique population, however, an attempt was made to identify the group of athletes that did not meet the DSM-IV criteria for AN or BN, but showed significant symptoms of EDs. These athletes were classified as having a subclinical ED termed anorexia athletica (Sundgot-Borgen, 1993). A few years after the introduction of the anorexia athletica definition in the sports medicine field, the EDNOS category was included in the DSM-IV criteria (APA, 1994), and most athletes

Table 42.1 Diagnostic criteria for anorexia nervosa (American Psychiatric Association, 2000)

Table not available in this digital edition.

Table 42.2 Diagnostic criteria for bulimia nervosa (American Psychiatric Association, 2000)

Table not available in this digital edition.

meeting the criteria for anorexia athletica also fulfill the EDNOS criteria. The clinical EDs have many features in common, and patients frequently move between them. The DSM-IV diagnostic criteria for AN, BN, and EDNOS are listed in Tables 42.1, 42.2, and 42.3, and a short description of these three disorders follows below.

Anorexia Nervosa (AN): AN is the most extreme restrictive eating behavior, where an individual continues to starve and feel fat despite being more than 15% below an ideal body weight. AN is also characterized by amenorrhea in post-menarche girls (APA, 2000) (Table 42.1).

Bulimia Nervosa (BN): A bulimic behavior refers to a cycle of food restriction or fasting followed by inappropriate compensatory behavior such as self-induced vomiting, abuse of laxatives, diuretics, or other medications, fasting or excessive exercise. Individuals suffering from BN are usually within a normal body weight range (APA, 2000) (Table 42.2).

Eating Disorder Not Otherwise Specified (EDNOS): The EDNOS category refers to disorders of eating that do not meet the criteria for any specific ED. For example, that the individual displays all the characteristics of AN, but has regular menses. Or that all the criteria for BN are present except that the binge eating and inappropriate compensatory behaviors have occurred less frequently than twice per week in the last three months (APA, 2000). This category acknowledges the existence and importance of a variety of eating disturbances (Table 42.3).

Prevalence of Eating Disorders Among Athletes

For about 20 years ago, weight loss practices were found to be a part of the participation of many

Table 42.3 Diagnostic criteria for eating disorders not otherwise specified (American Psychiatric Association, 2000)

Table not available in this digital edition.

women in exercise and sports, and several studies reported that ED symptoms or EDs were common in female athletes and even more prevalent than among nonathletic controls (Sundgot-Borgen, 1993). In recent years, several studies indicate that the challenge with EDs among athletes is still present, and that the prevalence is not restricted to female elite athletes, but applies also for male athletes, among athletes participating in recreational sports, among high school and college athletes, and among fitness instructors (Byrne & McLean, 2002; Glazer, 2008; Höglund & Normen, 2002; Holm-Denoma et al., 2009; Sundgot-Borgen & Torstveit, 2004).

Prevalence Among Female Athletes

The prevalence of DE behavior and EDs among female athletes has been estimated to range from 1% to 78% (Byrne & McLean, 2001; Smolak et al., 2000). The wide range in prevalence among female athletes may be explained by a number of methodological factors, such as different definitions, different groups of athletes (i.e., age, performance level), and different sport disciplines being studied. It has been claimed that the prevalence seems to be higher in elite athletes than in athletes at lower competitive levels and controls (Byrne & McLean, 2001, 2002; Sundgot-Borgen, 1993; Sundgot-Borgen & Torstveit, 2004). Interestingly, a recent systematic review including female athletes (not only elite athletes) and nonathletes aged between 12 and 35 years old concluded that most studies (12 of 22 studies) showed no significant difference between the athlete group and the control group regarding the presence of DE (Coelho et al., 2010). These results were based on studies in 11 countries, and most studies used self-reporting questionnaires to screen for DE. An important consideration was, however, that even if it was suggested that female athletes have a similar risk of developing DE to that of nonathletes, the athletes seemed to be in more severe stages of the disorder, e.g., showing a higher frequency of menstrual dysfunction (Coelho et al., 2010). Furthermore, among the five studies that extended the methods from using only questionnaires to adding clinical interviews to diagnose EDs, three found a higher prevalence in the athlete group than in the control group (Byrne & McLean, 2002; Sundgot-Borgen, 1993; Sundgot-Borgen & Torstveit, 2004).

It is further important to add that some prevalence studies have looked into different sport groups and concluded that there seem to be some differences in the prevalence of EDs depending on the type of sport/event and competitive level. In 2008, a controlled study using clinical interviews to diagnose EDs found a higher prevalence rate of EDs among athletes in leanness sports (47%) than among athletes in non-leanness sports (20%) and among nonathletic controls (21%) (Torstveit et al., 2008). It should be noted that this study failed to find any significant difference between the elite athletes as a whole and the control group when age was controlled for (Torstveit et al., 2008). Other studies have confirmed these findings, concluding that EDs seem to be more prevalent in leanness sports compared with non-leanness sports (Byrne & McLean, 2001; Sundgot-Borgen, 1993; Sundgot-Borgen & Torstveit, 2004). Hence, even if some findings indicate no significant differences between athlete groups and nonathletic groups, DE and EDs appear to be more frequent among female athletes competing in sports

focusing on leanness and/or a low weight than among athletes competing in sports where these factors are considered less important.

An interesting question is whether there are any trends in the prevalence of EDs over time. Limited longitudinal research has been done, but we have some experiences based on studies on health-related issues in female elite athletes and matched nonathletic controls in Norway, since the beginning of the 1990s. To our knowledge, this type of long-term follow-up studies on the prevalence of EDs has not been performed elsewhere. During the period from 1990 to 2002, new groups of Norwegian elite athletes were included. However, in all studies (Sundgot-Borgen, 1993; Sundgot-Borgen & Torstveit, 2004; Torstveit et al., 2008), the total population of female elite athletes, in an almost identical age range (12–39 years), was included. Based on these three Norwegian studies, an increase in the prevalence of clinical EDs was observed during the investigated period for female elite athletes as a group ($p < 0.0001$). The prevalence of EDs had increased gradually from the beginning of the 1990s to 2002 in both female athletes and nonathletes, with the largest increase observed during the last 5 years of the period. In the athletes, the prevalence of EDs had increased from 20% to 28% ($p = 0.0007$) and in the controls from 5% to 21% ($p < 0.001$) in this period. More individual and group-based longitudinal studies over time are, however, needed to increase the knowledge related to risk factors and the true trend in prevalence of EDs.

Prevalence Among Male Athletes

There has been a widely held assumption that DE and EDs are rare among men and boys, and even that male athletes are not affected by this disorder. The female to male ratio from population-based studies is estimated to be in the range of 10:1 for both AN and BN (APA, 1994). A limited number of studies, however, in the last 15 years have shown that DE and EDs are not only affecting female athletes, but also male athletes in several sport groups (DiGioacchino et al., 2002; Martinsen et al., 2010; Riebl et al., 2007; Rosendahl et al., 2009; Sundgot-Borgen & Torstveit, 2004). In a Norwegian study investigating the total population of both male and female elite athletes using both questionnaire screening and clinical interview to diagnose possible EDs, the authors found the prevalence rate of EDs among elite athletes and controls to be 8% and 0.5% among men and 20% and 9% among women (Sundgot-Borgen & Torstveit, 2004). In the same study, the prevalence of EDs among male elite athletes was reported to be 22% in gravitational sports, 18% in weight class sports, 9% in endurance sports, 5% in ball-game sports, 4% in technical sports, and finally no cases of EDs in aesthetic, power, or motor sports were found (Sundgot-Borgen & Torstveit, 2004).

To sum up, there are still too few controlled studies that offer evidence of an association between DE/EDs and female athletes, and there are a very limited number of controlled studies investigating the prevalence of DE/EDs in male athletes. In addition, methodological weaknesses exist related to a number of the published prevalence studies. Therefore, the true prevalence of EDs in the athletic population is not known.

Risk Factors for Eating Disorders Among Athletes

Many athletes are dieting to enhance performance, and dieting, weight losses, frequent weight fluctuation, and restrained eating have been shown to be very common entry points for developing an ED (Sundgot-Borgen, 1994). It is believed that the pathogenesis of EDs is multifactorial, with cultural, individual, family, and genetic/biochemical factors each playing roles (Fisher, 2006). Why some athletes cross the line from dieting and use of extreme weight loss methods to serious, diagnosable EDs is not known. However, it may be possible that when elite athletes face the paradigm of their sport there might be a pressure to reduce weight and body fat, and this behavior can create a culture for DE.

Many authors have found relationships between sports participation and EDs or DE (Byrne & McLean, 2002; Krentz & Warschburger, 2011; Sundgot-Borgen & Torstveit, 2004; Torstveit et al., 2008), but different studies present, to some extent, contradictory results due to athletic performance level, kind of sport, and

methodology used. Consequently, it is difficult to draw too strong conclusions about this issue. However, because of additional stress associated with the athletic environment, elite athletes appear to be more vulnerable to EDs than the general population (Sundgot-Borgen, 1994; Thompson & Sherman, 2010). Some risk factors that have been discussed are restrained eating and training, frequent weight-cycling, early start of sport-specific training, personality factors, injuries, overtraining, and the impact of coaching behavior (Smolak et al., 2000; Sundgot-Borgen, 1994). The pressure to reduce weight has been the common explanation for the increased prevalence of eating-related problems among athletes. The important factor may, however, not be dieting *per se*, but rather the situation in which the athlete is encouraged to lose weight, the phrasing of the message given, and whether the athlete received professional guidance or not. In addition to the pressure to reduce weight, athletes often have little time to achieve their goals, and they may have to lose weight rapidly to make the weight to be selected for, or to remain on the team. As a result, they often experience frequent periods of restrictive dieting or weight cycling (Sundgot-Borgen, 1994). Such weight fluctuation has been suggested to be an important risk or trigger factor for the development of EDs in athletes (Sundgot-Borgen, 1994).

Several of the psychological characteristics of elite athletes correspond with the character traits in people diagnosed with a clinical ED. Extreme performance orientation, high degree of perfectionism, a tendency to obsessive thoughts, and low self-esteem are among those discussed in the literature. These properties, with the possible exception of the low self-esteem, are assumed also to be significant in order to perform at a high athletic level (Sundgot-Borgen & Torstveit, 2004).

It is important to have in mind that to examine the true risk and trigger factors of participation in sports and the development of EDs, controlled, longitudinal studies are needed. In order to identify at-risk athletes as early as possible, knowledge of signs and symptoms is necessary. An overview of physical/medical and psychological/behavioral characteristics of DE/EDs among athletes is presented in Table 42.4.

Health and Performance Consequences

For both female and male athletes, the desire to excel in sports has led to various methods of training, conditioning, and dietary alterations in an attempt to improve their performance. Case reports have shown that the attempt to fulfill an athletic potential may lead to extreme behaviors that for some athletes result in very undesirable and unhealthy consequences. Hence, EDs may cause serious medical problems and, in some circumstances, even be fatal.

A recent meta-analysis found an overall elevated mortality rate for patients with all types of ED, with the highest risk of death in those with AN, with a weighted annual mortality rate of 5 per 1000 persons per year. In patients with EDNOS, the annual mortality rate was found to be 3 per 1000 persons per year, and finally 1.7 per 1000 persons per year in patients with BN (Arcelus et al., 2011). Mortality rates of EDs among athletes are not known. However, a number of deaths among top-level athletes representing gymnastics, running, alpine skiing, and cycling have been reported in the media. Five athletes (5.4%) of those diagnosed with EDs in a Norwegian study (Sundgot-Borgen, 1994) reported suicide attempts.

The physiological and medical complications and the effect on performance that are associated with EDs depend upon the severity and/or duration and frequency of energy restriction, the amount of weight loss, rate and composition of weight loss, and the electrolyte imbalance induced by dehydration or purging. We have found no studies that specifically have examined the short- and long-term health effect of extreme dieting and DE in elite athletes. A limited number of studies have looked at the female athlete triad disorders and the increased risk of injuries (Thein-Nissenbaum et al., 2011). However, it is reasonable to expect that the similar health consequences reported among nonathletes will apply also to athletes.

Medical Consequences

Physiological and medical complications involve the cardiovascular, gastrointestinal, endocrine (including decreased metabolic rate), reproductive,

Table 42.4 Physical/medical and psychological/behavioral characteristics (signs and symptoms) of disordered eating/eating disorders among athletes

Signs and symptoms
Physical/medical characteristics
Low energy availability
Irregular menstruation
Noticeable weight loss or constant weight fluctuation
Excessive training
Frequent injuries and lengthened recovery
Overuse injuries and stress fractures
Muscle weakness, cramps, or both
Fatigue beyond that normally expected in training and competition
Symptoms of overtraining (can also be psychological)
Gastrointestinal problems (i.e., constipation, bloating, diarrhea)
Hypothermia
Dehydration
Electrolyte abnormalities
Dental and gum problems
Cardiac arrhythmias
Anemia
Delayed growth
Hypoestrogenemia
Hypercortisolemia
Infertility (lasting?)
Possible increased breast, cervical, ovarian cancer risk
Elevated plasma cholesterol, triglycerides, and low density lipoprotein (LDL)
Reduced endothelium-dependent vasodilatation
Skeletal demineralization
Psychological/behavioral characteristics
Restrictive eating and/or binging and purging[a] (some have secretive eating)
Anxiety, both related and unrelated to sport performance
Depression
Avoidance of eating and eating situation
Dissatisfaction with body image
Extreme performance orientation
Self-critical, especially concerning body weight, composition, and performance
Low self-esteem, poor coping skills, compulsiveness, perfectionism, and self-critical
Mood swings
Claims of feeling fat despite being thin
Compulsive and excessive training, also when injured
Insomnia
Resistance to weight gain or weight maintenance recommended by sport support staff
Excessive use of the restroom
Substance abuse–-whether legal, illegal, prescribed, or over-the-counter drugs, medications, or other substances
Bathroom visits immediately following meals
Reduced social activities
Poor concentration, fatigue

Source: Revised based on Thompson and Sherman (2010) and Sundgot-Borgen (2002).
[a]Laxatives, vomiting, and/or excessive exercising.

skeletal, renal, and central nervous systems, including psychological stress and depressions (Nattiv et al., 2007). Whereas most complications of AN occur as a direct or indirect result of starvation, complications of BN also relate to binge eating and purging (Thompson & Sherman, 2010). The loss of fluids and electrolytes during purging can induce serious medical problems, including dehydration, acid–base abnormalities, and cardiac rhythm disturbances. Furthermore, the negative effects of rapid weight loss (e.g., fasting, dehydration) and longer periods of restricted energy intake on performance, growth, cognitive function, and health have been discussed (Beals, 2004; O'Connor & Caterson, 2006).

Consequences for Performance

The effects of EDs on sports performance depends on the type of ED, as well as the duration and the severity of the ED. Endurance performance is likely to be impaired if the liver and muscle glycogen levels are lower or if the athlete is dehydrated or anemic. Dehydration is common in both AN and BN, and acute dehydration can lead to loss of motor skills and coordination. A reduced blood volume in the dehydrated state may impair thermoregulatory capacity during exercise, which may lead to impaired performance. Electrolyte disturbances are probably detrimental to muscle function and, over time, may lead to a loss of muscle mass and decreased strength and power (Rankin, 2006).

It should also be mentioned that some of those athletes who severely restrict their energy intake for short periods and lose weight do experience an initial, albeit, transient improvement in performance. This may be related to the initial physiological and psychological consequences of starvation (Beals, 2004). With weight loss, athletes may feel lighter, and experience a psychological boost, particularly, if they believe that lighter means improved performance (Beals, 2004). The result is a stimulatory effect on the central nervous system, which will evoke a sense of excitability and exhilaration in the energy-restricting athlete. In addition, the decrease in body weight may induce a transient, direct increase in performance through the reduction in weight itself or indirectly by increasing aerobic capacity per kilogram body weight. The latter factor is crucial for performance in endurance events.

In 1992, a relationship between DE, amenorrhea, and osteoporosis in women participating in leanness sports was discovered, and this was referred to as the female athlete triad (the Triad). In 2007, a revised position statement of the American College of Sports Medicine suggested that each clinical condition of the Triad comprises the pathological end of a spectrum of interrelated, subclinical conditions between health and disease (e.g., a spectrum of energy availability from optimal energy availability to low energy availability, with or without an ED) (Nattiv et al., 2007). Dieting athletes may experience low energy availability (see Chapter 5), may slip into DE, which in turn can lead to a serious ED, disruption of the normal menstrual cycle, and, eventually, imbalance in bone remodeling leading to osteopenia or osteoporosis. Although any one of these problems can occur in isolation, the emphasis on weight loss and/or low energy availability in individuals at risk can start a cycle in which all three diseases occur in sequence (Nattiv et al., 2007). In recent years, studies have found the presence of the Triad in elite athletes representing both leanness and nonleanness sports (Torstveit & Sundgot-Borgen, 2005), in female college athletes (Beals & Hill, 2006) and high school athletes (Nichols et al., 2006), and even in women who are not competing in sports (Torstveit & Sundgot-Borgen, 2005). A controlled study of the entire population of female elite athletes in Norway demonstrated that 4.3% of female elite athletes met the criteria for the Triad. When evaluating the presence of two of the components of the Triad, prevalence ranged from 5% to as high as 27% in elite athletes (Torstveit & Sundgot-Borgen, 2005).

There has also been some speculation that a male athlete triad exists, especially since some recent studies have found low bone density values in male athletes (Hetland et al., 1993; Rector et al., 2008; Smathers et al., 2009). Two studies have investigated male cyclists. Smathers et al. (2009) found that 9% of male competitive cyclists and 3% of age- and body mass-matched controls were classified as osteoporotic, while as many as 25% of the cyclists compared with 10% of the controls were classified as osteopenic. Furthermore, Rector et al.

(2008) found that 63% of their group of recreational male cyclists had osteopenia of the spine or hip, compared with 19% of a comparison group of runners. The authors concluded that, after controlling for age, body weight, and bone-loading history, cyclists were seven times more likely to have osteopenia of the spine than runners. Reduced bone mass and increased bone turnover have also been found in male long distance runners compared with controls (Hetland et al., 1993). The pathophysiological explanation is not clear in these studies, and more research is needed to explain this possible phenomenon of a male athlete triad.

Possible performance consequences of DE/EDs in athletes are listed in Table 42.5.

Management of Eating Disorders Among Athletes

Specific strategies to prevent, detect, and treat EDs in athletes can include surveillance, research, medical care, and public and professional education.

Table 42.5 A presentation of possible performance-related consequences associated with disordered eating/eating disorders

Performance-related consequences
Decreased nerve conduction velocity
Decreased muscle contraction rate
Decreased reaction time
Atrophy and loss of lean body mass
Decreased strength and power
Decreased aerobic endurance
Decreased blood flow to skeletal muscle
Decreased delivery of oxygen to muscle
Increased recovery time
Impaired oxidative metabolism in skeletal muscle
Increased number of training days missed due to musculoskeletal injuries or infections
Decreased self-esteem, fear of failure
Decreased concentration
Light-headedness
Fatigue

Source: Revised based Torstveit (2004) and Sundgot-Borgen (2002).

In terms of education, possible target groups are coaches, trainers, parents, athletes, peers, athletic administrators, officials of sport governing bodies, and healthcare professionals who work with athletes (Nattiv et al., 2007).

The prevention and treatment of athletes with EDs can be organized into three categories. Primary prevention involves education and instruction designed to prevent the onset of EDs. Secondary prevention focuses on early identification of at-risk athletes and follow-up treatment, while tertiary prevention includes treatment of those athletes who have developed EDs.

Primary Prevention

The goal of primary prevention should be to protect young athletes from factors that can predispose them to the development of DE or EDs. Due to the fact that body shape and weight obsession, dieting, and development of EDs can start as early as puberty, primary prevention should be initiated as early as primary or junior high school. Information about proper nutrition and healthy living habits, and a focus on a positive self-image seem to be essential. In addition, the athletic environment should avoid focus on unhealthy attitudes and behaviors toward body weight and performance. Coaches must be educated and made aware of their responsibility when working with young female athletes. So-called "casual" comments about an athlete's body shape and size with respect to performance, or demanding that the athlete lose weight in relation to growth and development may be destructive and must never occur. In the twenty-first century, athletes, coaches, parents, and health professionals ought to realize the potential consequences of extreme dieting.

It is important to dispel existing myths and misunderstandings about the association between energy/nutrient intake, dieting, body weight, body composition, menstruation, and athletic performance. Focus should be placed on the importance of acquiring a healthy and performance-enhancing diet, as well as development and maintenance of a positive self-image, thereby helping the athletes to set realistic goals for body composition, training

volume, and finally athletic performance (Nattiv et al., 2007). Athletics, in general, and every specific sport in particular should, in its own way, contribute toward primary prevention of DE/EDs. Change of rules in some sports may be one possible strategy. In order to prevent some athletes from striving toward unrealistic weight goals in order to perform better, introduction or change of rules regarding age and/or BMI limits for competition, weigh-in times, or proper clothing or equipment, may be considered.

Secondary Prevention

Secondary prevention should aim to limit the progression and shorten the duration of DE symptoms. Identification of athletes at risk and an early intervention are essential because EDs are identical to most unwanted situations in that they become harder to treat as they progress.

Different screening instruments have been developed over time to make it easier to identify people at risk of developing EDs or people who already have an ED, and as a help to diagnose these disorders. Measurement methods to identify DE include self-report, observation, symptom checklists, standardized test meals, and clinical interviews. A combination of standardized surveys and clinical interviews is regarded as the optimum method to identify prevalence of EDs among athletes (Sundgot-Borgen & Torstveit, 2004; Torstveit et al., 2008), and it is claimed that true AN or BN only can be diagnosed through clinical interview, and that the self-administered or self-report questionnaires, above all, serve to detect minor deviant eating behaviors (Ruiz-Lazarao et al., 2010).

Among the many existing screening instruments, the Eating Disorders Inventory (EDI) and the Eating Attitudes Test (EAT) seem to be the most commonly used to identify DE. In athletes, a variety of different screening instruments and questions have been used. In a recent systematic review investigating 22 prevalence studies on DE in female athletes, the authors found that nine studies used the EAT questionnaire (eight used the short version and one used the long version), five studies used the EDI (four used a selection of the subtests and one used the extended version), three studies used the Eating Disorder Examination Questionnaire (EDE-Q), and finally one study used the Female Athlete Screening Tool (FAST) (Coehlo et al., 2010). Among the five studies using interviews to diagnose EDs, four applied a semi-structured clinical interview (the Eating Disorder Examination, EDE) and one applied a structured interview (Composite International Diagnostic Interview, CIDI) (Coelho et al., 2010).

To detect an athlete with EDs can be challenging because the disorders are not always readily apparent. Furthermore, some athletes will attempt to conceal the symptoms. Unfortunately, the symptoms of EDs are too often ignored or overlooked by coaches, health personnel, or others in contact with the athletes, possibly due to a lack of knowledge about the symptoms of DE/EDs, an extreme performance-oriented approach, or a more or less conscious fear of discovering an ED in one of "their" athletes.

Therefore, professionals working with athletes should acquire knowledge about the possible risk factors, and early signs and symptoms of DE/EDs (Table 42.4), the medical- and performance-related consequences of DE/EDs (Table 42.5), as well as how to approach the problem if it occurs (see section under Tertiary Prevention). Each team coach should work closely with sports medicine professionals to get help and guidance with weight problems, nutritional factors, injury prevention, menstrual issues, and body composition measurements.

Recommendations for Health Professionals

Unfortunately, it is rather rare that an athlete will admit an eating problem during the first consultation. Generally, the athlete may describe symptoms that do not necessarily point to an ED, such as headaches, constipation, diarrhea, sleeping problems, breathing problems, dizziness, sadness, or tiredness. He or she may complain that others make suspicious comments about their eating behavior or their lost weight or related issues. Regardless of which complaints/symptoms are presented during the first consultation, the health professional should search for possible underlying causes. Questions about food intake, dieting methods, and training should be straightforward and concrete. When discussing potential EDs, the athlete may feel less

threatened if the discussion focuses on the past (Drinkwater et al., 2005). When the consequences amenorrhea (female athletes)/low testosterone levels (male athletes) and loss of bone mineral density are also suspected, one should ask the athlete questions about current and previous weight, diet, menstruation, and his or her training program. Table 42.6 presents questions that can be included in the consultation.

Tertiary Prevention

Treatment of an athlete with EDs should utilize a multidisciplinary intervention, which normally includes a physician, a gynecologist, a nutritionist, a physical therapist, an exercise scientist, and, in some cases, a psychiatrist or a psychologist. In addition, coaches and parents may be part of the treatment team if they have a positive relationship with the athlete. The younger the athlete, the more the involvement of the family is recommended. The primary goals of treatment should be to optimize energy availability, to control and manage the athlete's DE behavior, to restore normal hormone levels, and to monitor and treat other medical complications resulting from the ED (Drinkwater et al., 2005; Sundgot-Borgen & Torstveit, 2010; Torstveit, 2004). In Table 42.7, an example of the anthropometric, biochemical, clinical, dietary, and environmental assessment is presented.

When treating an athlete with an ED, it is important to establish that each treatment group member

Table 42.6 Questions that can be included in the consultation (Drinkwater, 2005; Torstveit, 2004)

Questions about food	Questions about weight	Questions about menstruation (for females)	Questions about training and injuries
Do you feel that you have a "relaxed" relation to food?	What have your highest and lowest body weight been during the last year?	When did you experience your first menstruation?	Please describe your normal training volume (frequency, intensity, duration)
Please describe your eating habits[a]	What would you say your "competition" body weight is?	Have your periods been regular since you first began menstruating?	Have you altered training methods, volume, or intensity recently (or in the past)?
How many meals do you eat per day?	Have you lost weight recently? How did you achieve the weight loss?	What is the longest period of time you have experienced lack of menstrual bleeding?	Do you practice any exercise in addition to your sport-specific training?
Are there any types of food you try to avoid or do you have any "forbidden" foods?	Are you comfortable with your current body weight?	When was your last period?	Have you had any problems with overuse injuries? What type of injuries?
Can you tell me what you ate and drank yesterday?	Are there other people who are especially interested in your body weight?	How do you feel about having/not having menstruation?[b]	Have you ever had a stress fracture or a normal fracture?
Question the athlete about purging methods (preferably referring to the past)		Do you currently use oral contraceptive pills or other contraceptives? Have you used them in the past?	When experiencing an injury, did you take time to recover? How did you feel about that?

AN, anorexia nervosa; BN, bulimia nervosa; ED, eating disorder.

[a]It can be difficult for an individual with eating problems to spontaneously list what he/she has eaten. A person with AN avoids fat and is often a vegetarian. Eating habits are characterized by the same restrictive intake of "acceptable" foods each day. A person with BN constantly attempts to postpone energy consumption then overeats in the afternoon and evening.

[b]A female athlete with an ED prefers not to menstruate (many feel it is a "defeat" to have such a high percent body fat that one has regular menstruation).

Table 42.7 The anthropometric, biochemical, clinical, dietary, and environmental assessment of athletes with disordered eating/eating disorders by health professionals (Adapted from N. Meyer, unpublished table)

ABCDEs	Measures	Comments
Anthropometric	• Body height • Body mass • Body composition • Girth and breadths	Valid and reliable methods of body composition should be sought (e.g., DXA, skinfold assessment using the International Society for Advancement of Kinanthropometry (ISAK) standards; 4 component models assessing fat, fat-free, and lean tissue mass, and total body water; measurement of hydration status recommended for all anthropometric assessments). Careful reflection required whether assessment of body mass and composition may trigger more problems.
Biochemical	• Complete blood count • Complete metabolic panel • Lipid panel • Iron profile • Thyroid function (e.g., TSH and T_3) • Females: Estradiol, progesterone, prolactin, LH, and FSH • Males: Testosterone • Cortisol • 25 (OH) Vitamin D • Urine analysis • Pregnancy test	In females with menstrual dysfunction, prolactin needs to be assessed to rule out pituitary tumor; if ovarian cysts and oligomenorrhea, androgens should be assessed.
Clinical	• History • Physical exam • Medications • Dietary supplements	Medical history should include previous DE and EDs. If pre-participation, physical, general medical history, including menstrual history/status, bone health, history of stress fracture and other injuries, osteoporosis. Screening for DE and EDs with screening tools or clinical interview and identification of physical signs and symptoms (e.g., general (weight fluctuations, fatigue, appetite, sleep, edema); skin (dryness, callus on hands); hair (loss, lanugo); gastrointestinal symptoms (constipation, diarrhea, pain, reflux, teeth, sore mouth/tongue); cardiopulmonary (heart rate, sleep apnea, blood pressure); renal (polydipsia, color); musculoskeletal (pain, injuries, bone mass); immune (frequent illness); other (parotid artery enlargement); weight fluctuations (pressure to lose weight, highest/lowest body mass at current height)). Medications (oral contraceptives, antidepressants, thyroid medication). Dietary and sport supplements (e.g., vitamins, mineral, energy-containing supplements, and ergogenic aids, stimulants).
Dietary	• Quantity • Quality • Timing • Energy expenditure	Energy intake; total daily energy expenditure; resting metabolic rate; exercise energy expenditure; energy availability; energy density; macro- (expressed in g/kg/day) and micronutrients; fluid balance and hydration; including sweat rate; food restrictions; allergies, intolerance; scary foods; nutrient and fluid timing; carbohydrate availability during intense training; carbohydrate and fiber related to appetite; recovery nutrition; competition preparation and fueling; travel nutrition; and appetite issues during travel or intense training. Dietary assessment methods: Consider validity and reliability as well as additional burden and stress on a DE/ED athlete when using diaries and food logs.

(Continued)

Table 42.7 *(Continued)*

ABCDEs	Measures	Comments
Environmental	• Culture of sport • Annual training plan and peaking • Travel • Work/school • Family/home • Experience in sport	At risk for DE/ED? Target-specific issues such as weight-making or body image depending on sport; evaluation of training/competition plan in discussion with coach; countries at-risk for inadequate food access and food safety concerns; work/school schedules and time for food preparation, eating, recovery; level of athlete and experience.

DE, disordered eating; ED, eating disorders.

has a clear understanding of their responsibilities and that the group members communicate well. As long as there are no underlying complex conflicts contributing to cause the ED, regular and long-term follow-up by a nutritionist is usually sufficient. Often, though, complicating medical factors are present and therefore the physician in cooperation with the coach remains central in the follow-up process. The physician should be responsible for providing guidelines with respect to training and participation in competition during the treatment period, and should consider an athlete suffering from EDs as an injured athlete. Athletes with severe EDs should not be allowed to compete.

In terms of possible treatment initiatives for the athlete with DE behavior and/or EDs, we refer to recent publications dealing with these issues (Bonci et al., 2008; Sundgot-Borgen & Torstveit, 2010; Thompson & Sherman, 2010).

Summary and Recommendations

It is important to note that DE should not be used as a reason to disqualify otherwise healthy athletes from athletic participation. Sports and physical activity, in general, are healthy for females and males at all ages, if carried out appropriately, and should be promoted for both health benefits and enjoyment. However, nutritional behavior, exercise volume, and menstrual history (for females) need to be reviewed and normalized in all athletes. EDs and/or DE behaviors should not be considered as normal responses to exercise. Education and counseling regarding factors associated with EDs should be provided to both male and female athletes, parents, coaches, health personnel, and others in contact with male or female athletes. Finally, potential treatment initiatives should be multidisciplinary in origin and be initiated as early as possible.

References

American Psychiatric Association (APA) (1994) Eating disorders. In *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn, pp. 539–550. APA, Washington, DC.

American Psychiatric Association (APA) (2000) Eating disorders. In *Diagnostic and Statistical Manual of Mental Disorders*, 4th edn, Text Revision, pp. 583–595. APA, Washington, DC.

Arcelus, J., Mitchell, A.J., Wales, J., et al. (2011) Mortality rates in patients with anorexia nervosa and other eating disorders. A meta-analysis of 36 studies. *Archives of General Psychiatry* 68(7), 724–731.

Beals, K.A. (2004) Effects of disordered eating. In *Disordered Eating among Athletes–-A Comprehensive Guide for Health Professionals*, Chapter 5, pp. 67–98. Human Kinetics, Champaign, IL.

Beals, K.A. & Hill, A.K. (2006) The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 16(1), 1–23.

Bonci, C.M., Bonci, L.J., Granger, L.R., et al. (2008) National athletic trainers' association position statement: preventing, detecting, and managing disordered eating in athletes. *Journal of Athletic Training* 43(1), 80–108.

Byrne, S. & McLean, N. (2001) Eating disorders in athletes: a review of the literature. *Journal of Science and Medicine in Sport* 4, 145–159.

Byrne, S. & McLean, N. (2002) Elite athletes: effects of the pressure to be thin. *Journal of Science and Medicine in Sport* 5, 80–94.

Coelho, G.M., Soares Ede, A., & Ribeiro, B.G. (2010) Are female athletes at increased risk for disordered eating and its complications? *Appetite* 55(3), 379–387.

DiGioacchino DeBate, R., Wethington, H., & Sargent, R. (2002) Sub-clinical eating disorder characteristics among male and female triathletes. *Eating and Weight Disorders* 7(3), 210–220.

Drinkwater, B., Loucks, A., Sherman, R., et al. International Olympic Committee Medical Commission Working Group Women in Sport (2005) *Position Stand on the Female Athlete Triad*. Available at: http://www.olympic.org/Documents/Reports/EN/en_report_917.pdf (accessed May 29, 2013).

Fisher, M. (2006) Treatment of eating disorders in children, adolescents, and young adults. Pediatrics in Review 27(1), 5–16.

Glazer, J.L. (2008) Eating disorders among male athletes. *Current Sports Medicine Reports* 7(6), 332–337.

Hetland, M.L., Haarbo, J., & Christiansen, C. (1993) Low bone mass and high bone turnover in male long distance runners. *Journal of Clinical Endocrinology and Metabolism* 77(3), 770–775.

Höglund, K. & Normén, L. (2002) A high exercise load is linked to pathological weight control behavior and eating disorders in female fitness instructors. *Scandinavian Journal of Medicine & Science in Sports* 12(5), 261–275.

Holm-Denoma, J.M., Scaringi, V., Gordon, K.H., et al. (2009) Eating disorder symptoms among undergraduate varsity athletes, club athletes, independent exercisers, and nonexercisers. *International Journal of Eating Disorders* 42(1), 47–53.

Krentz, E.M. & Warschburger, P. (2011) Sports-related correlates of disordered eating in athletic sports. *Psychology of Sport and Exercise* 12, 375–382.

Martinsen, M., Bratland-Sanda, S., Eriksson, A.K., et al. (2010) Dieting to win or to be thin? A study of dieting and disordered eating among adolescent elite athletes and nonathlete controls. *British Journal of Sports Medicine* 44(1), 70–76.

Nattiv, A., Loucks, A.B., Manore, M.M., et al. (2007) American college of sports medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise* 39(10), 1867–1882.

Nichols, J.F., Rauh, M.J., Lawson, M.J., et al. (2006) Prevalence of the female athlete triad syndrome among high school athletes. *Archives of Pediatrics & Adolescent Medicine* 160(2), 137–142.

O'Connor, H. & Caterson, I. (2006) Weight loss and the athlete. In *Clinical Sports Nutrition*, 3rd edn (eds. L. Burke & V. Deakin), pp. 135–174. McGraw-Hill Australia Pty Ltd.

Rankin, J.W. (2006) Making weight in sports. In *Clinical Sports Nutrition*, 3rd edn (eds. L. Burke & V. Deakin), pp. 175–199. McGraw-Hill Australia Pty Ltd.

Rector, R.S., Rogers, R., Ruebel, M., et al. (2008) Participation in road cycling vs running is associated with lower bone mineral density in men. *Metabolism* 57(2), 226–232.

Riebl, S.K., Subudhi, A.W., Broker, J.P., et al. (2007) The prevalence of subclinical eating disorders among male cyclists. *Journal of the American Dietetic Association* 107(7), 1214–1217.

Rosendahl, J., Bormann, B., Aschenbrenner, K., et al. (2009) Dieting and disordered eating in German high school athletes and non-athletes. *Scandinavian Journal of Medicine & Science in Sports* 19(5), 731–739.

Ruiz-Lázaro, P.M., Comet, M.P., Calvo, A.I., et al. (2010) Prevalence of eating disorders in early adolescent students. *Actas Espanolas Psiquiatria* 38(4), 204–211.

Smathers, A.M., Bemben, M.G., & Bemben, D.A. (2009) Bone density comparisons in male competitive road cyclists and untrained controls. *Medicine & Science in Sports & Exercise* 41(2), 290–296.

Smolak, L., Murnen, S.K., & Ruble, A.E. (2000) Female athletes and eating problems: a meta-analysis. *International Journal of Eating Disorders* 27, 371–380.

Sundgot-Borgen, J. (1993) Prevalence of eating disorders in elite female athletes. *International Journal of Sport Nutrition* 3, 29–40.

Sundgot-Borgen, J. (1994) Risk and trigger factors for the development of eating disorders in female elite athletes. *Medicine & Science in Sports & Exercise* 26, 414–419.

Sundgot-Borgen, J. (2002) Disordered eating. In *The Female Athlete* (eds. M.L. Ireland & A. Nattiv), pp. 237–248. Saunders, Philadelphia, PA.

Sundgot-Borgen, J. & Torstveit, M.K. (2004) Prevalence of eating disorders in elite athletes is higher than in the general population. *Clinical Journal of Sport Medicine* 14(1), 25–32.

Sundgot-Borgen, J. & Torstveit, M.K. (2010) Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine & Science in Sports* 20(2), 112–121.

Thein-Nissenbaum, J.M., Rauh, M.J., Carr, K.E., et al. (2011) Associations between disordered eating, menstrual dysfunction, and musculoskeletal injury among high school athletes. *Journal of Orthopaedic and Sports Physical Therapy* 41(2), 60–69.

Thompson, R.A. & Sherman, R.T. (2010) *Eating Disorders in Sport*, pp. 284. Routledge, Taylor & Francis Group, New York.

Torstveit, M.K. (2004) The female athlete triad in Norwegian elite athletes and non-athletic controls. Identification and prevalence of disordered eating, menstrual dysfunction and osteoporosis. Doctoral dissertation, pp. 1–116. Norwegian School of Sport Sciences.

Torstveit, M.K. & Sundgot-Borgen, J. (2005) The female athlete triad exists in both elite athletes and controls. *Medicine & Science in Sports & Exercise* 37(9), 1449–1459.

Torstveit, M.K., Rosenvinge, J., & Sundgot-Borgen, J. (2008) Prevalence of eating disorders and the predictive power of risk factor models in female elite athletes: a controlled study. *Scandinavian Journal of Medicine & Science in Sports* 18 (1), 108–118.

Chapter 43

Importance of Gastrointestinal Function to Athletic Performance and Health

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Introduction

Gastrointestinal (GI) function can influence health and physical performance. The GI tract provides a selective barrier to nutrients and other ingested substances, its function limiting or accelerating the arrival of ingested components into the bloodstream and hence delivery to organs and tissues. An application of knowledge concerning GI function underpins the optimum use of nutritional adjuncts to aid performance. Normal GI function, including selective membrane transport, motility, and a healthy microflora, is essential in maintaining health. Athletic competition and the environments in which it is conducted can compromise the integrity and/or functionality of the GI tract (Kayser, 1994; van Wijk et al., 2011). GI dysfunction can reduce nutrient availability and associated symptoms can be debilitating. For a competitive athlete, both can reduce performance capacity.

Taking exercise has been touted to aid digestion. This adage can be interpreted to mean that exercise accelerates the passage of a meal through the GI tract. In this chapter, the evidence supporting this statement will be examined and mechanisms of action, specifics of the imposed load, and effects of the environment in which it is conducted are explored. Specific nutrients can affect GI function and GI function can alter specific nutrient absorption. As a result, substrate oxidation and immune function can be influenced. This has particular relevance for athletes with high energy demands and those traveling for competition who may be exposed to exotic (pathogenic) microbial organisms.

Therefore, the specific aims of this chapter are to:

1. review the state of knowledge concerning GI function, how it is regulated with respect to exercise, nutrient intake and other factors associated with athletic endeavors, and
2. catalogue exercise-associated GI dysfunction, environmental influences, mechanisms and evidence regarding preventive measures and treatment.

Part I: GI Function and Nutritional Provision during Exercise

Effects of Exercise on GI Function

Gastric Emptying Gastric emptying has been monitored by various techniques, all having

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particular limitations, some more invasive than others or not tolerated by all individuals. For solid, as well as semisolid meals, technetium labeling and stationary imaging by gamma camera is the "gold standard" (Urbain & Charkes, 1995). Intubation and removal of stomach contents over time has been commonly used to monitor the emptying of fluids. A modification of a technique using phenol red dye was developed allowing sequential sampling and calculation of secretion, thus net beverage emptying (Beckers et al., 1991). When interpreting older gastric emptying studies errors can be made if the volume of secretion is unknown and it is assumed that the total stomach volume is ingested material and thus a slower emptying is reported, particularly in beverages which promote a high rate of secretion. Other techniques involve ingesting a tracer and monitoring the appearance in blood or breath. It is important to understand that the appearance of label may not represent the digestion/metabolism of all meal components, if only a certain compound or nutrient within the meal is labeled, e.g., isotopically.

Low intensity exercise, such as an after-dinner walk, increases gastric emptying (Neufer et al., 1989b). The effect of the intensity of physical exercise is graded in an inverted-U fashion: moderately intensive efforts have little (Rehrer et al., 1989) and less consistent effects (Neufer et al., 1989b) while very intensive (80–90% VO_2 max, maximal oxygen consumption, typically intermittent) efforts decrease gastric emptying (Leiper et al., 2001) (Figure 43.1). Some of the inconsistency at moderate intensities may be a result of methodological differences or lack of power to detect small changes. It appears to be the relative rather than the absolute intensity that is most important, probably due to corresponding sympathetic/parasympathetic effects (Wang et al., 2010). Numerous GI peptides also alter gastric motility, although motility is mainly affected by feeding state (Hellström et al., 2006). Endogenous opioids and other neuropeptides may also be implicated. Even at maximal exercise intensities, the consistent, statistically significant delay in gastric emptying that is observed is not large enough to deter ingestion of fluid when losses are of a magnitude to impair performance. It would, however, indicate that an athlete involved

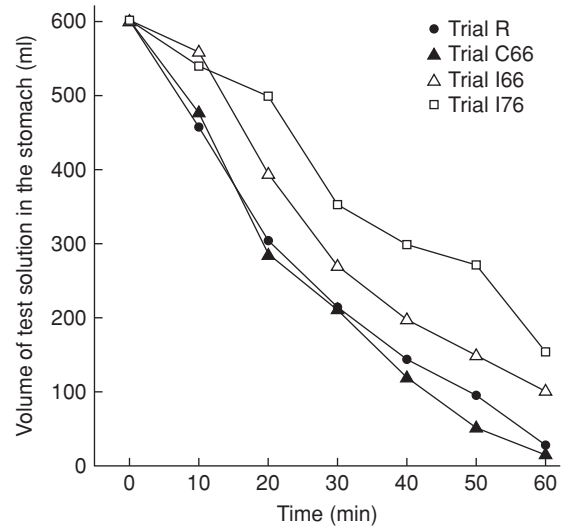

Figure 43.1 The median volume of a carbohydrate–electrolyte beverage remaining after ingestion at rest (R) or during continuous cycling at 66% VO_2 max (C66) or at 66% VO_2 max interspersed with blocks of three 30-second sprints at 100% of VO_2 max (I66) or at 75% VO_2 max interspersed with blocks of three 30-second sprints at 100% of VO_2 max (I75). From Leiper et al. (2001).

in a sport that demands maximum efforts should begin competition well hydrated, anticipate needs and, when appropriate, supplement in advance.

The mode of exercise appears not to be of particular importance. The majority of gastric emptying studies involved ambulation, walking or running, and/or cycling. In studies in which the same subjects repeated similar trials with both cycling and running, no difference in gastric emptying was observed (Houmard et al., 1991). To our knowledge, the effects of arm exercise or swimming have not been evaluated.

Rehrer et al. (1989) did not observe a difference in emptying of either water or carbohydrate (CHO) beverages at rest or during exercise (50% and 70% VO_2 max) between trained cyclists and untrained individuals. Carrio et al. (1989), however, observed faster gastric emptying at rest in marathon runners than in untrained individuals. Meal differences may account for the inconsistency; Carrio et al. evaluated the emptying of a solid, egg-based meal. Whether it was the nature of the meal, the higher energy

content, the protein and fat in the solid meal, or differences in adaptation to running versus cycling training remains unknown. Although gastric emptying during exercise at a given relative intensity (e.g., %VO_2 max %HR max) does not appear to differ in trained versus untrained, at a given absolute intensity (e.g., running 14 km/h) the untrained individual would be at a higher relative intensity and may be more likely to be affected.

There are large interindividual differences in gastric emptying rate. Nevertheless, from the few data collected comparing males and females, it appears that, in general, males have faster emptying rates, probably related to estradiol and progesterone effects on the GI tract smooth muscle decreasing motility (Datz et al., 1987). The effects of increasing age on gastric emptying are inconsistent but it appears that CHO fluids empty more slowly in older (~70 years) than younger (~20 years) individuals (O'Donovan et al., 2005).

Hyperthermia, accompanied by hypohydration, during exercise also reduces gastric emptying rate (Neufer et al., 1989a). It appears that hypohydration alone does not impair gastric emptying (Ryan et al., 1998) but rather it is the combined effects of exercise in the heat and hypohydration on thermal strain, possibly related to a greater reduction in blood flow to the gut. This information might suggest to an athlete that fluid ingestion should commence before hypohydration has occurred. Psychological stress can also delay gastric emptying (Beaumont, 1838), which might lead to greater delay during competition than in controlled laboratory studies.

Intestinal Absorption and Secretion The most widely recognized method to assess the intestinal absorption of varying fluids is the triple lumen perfusion method, which allows for quantification of absorption of water and nutrients and secretion of water, thereby deriving net flux. This method is difficult to apply during exercise, although some have been able to do this. It also typically measures absorption over only a short segment of the small intestine and, although it can reliably compare flux from fluids of varying composition, it does not represent absorption across the whole intestine. Furthermore, the infusion rate is typically held constant and, thus, may not reflect variation in gastric emptying rate that occurs with varying composition. The method also fails to account for changes in the composition of fluids after oral ingestion. Meal labeling and imaging techniques can be used, as described above, producing a product of both gastric emptying and intestinal absorption.

Results from studies of physical exertion on intestinal absorption are inconsistent. Neither Fordtran and Saltin (1967) using the triple lumen perfusion technique and passively absorbed markers, nor Gisolfi et al. (1991), also with the perfusion technique, with imposed exercise up to ~70% VO_2 max, observed a consistent effect of exercise on absorption, with wide interindividual variation. Maughan et al. (1990) did, however, observe progressive decrease in deuterium accumulation in the blood after ingestion of a D_2O containing solution as exercise intensity increased from 42% to 80% VO_2 max. The rate of tracer accumulation is a result of gastric emptying as well as absorption, and may be more affected by the former. Barclay and Turnberg (1988) also found an exercise effect on absorption at an unknown relative intensity in untrained individuals. It may be that intestinal absorption is reduced during exercise only when the exercise intensity and/or environment is such that the blood flow to the GI tract is reduced to a degree that O_2 supply is also reduced.

Motility and GI Transit GI motility drives transit through myoelectric activity, modulated by enteric and autonomic nerves. Numerous studies have been conducted to examine motility and transit over varying portions or the whole GI tract. In line with exercise effects on gastric emptying, moderately intense exercise has been shown to enhance myoelectric activity (Lu et al., 2000), motility and transit, while intense exercise (from ~85% to 90% VO_2 max) has been shown to decrease motility (Brown et al., 1994). Undertaking repeated activity (training), be it endurance (Cordain et al., 1986) or resistance-type (Koffler et al., 1992) exercise, decreases colorectal transit time, whereas imposed inactivity in those previously active increases (nearly doubling) colonic transit time (Liu et al., 1993). This effect may contribute to the inverse relationship between

physical activity and colon cancer risk (Thune & Lund, 1996). As with gastric emptying, colonic transit varies widely between individuals but, in general, it is faster in males than females (Degen & Phillips, 1996).

Blood Flow and Intestinal Permeability Exercise decreases splanchnic blood flow (Rehrer et al., 2001; Rowell et al., 1964) (Figure 43.2). This is most likely due to increased sympathetic drive with increasing exercise intensity. Moderate decreases in blood flow (up to ~40%) are compensated by an increase in O_2 extraction but, as exercise can reduce splanchnic blood flow by up to ~80%, O_2 supply to the gut can be impaired. The combined effects of exercise with hyperthermia and dehydration can reduce blood flow to a greater extent. Nonsteroidal anti-inflammatory drugs (NSAIDs)/aspirin also decrease visceral and renal perfusion during exercise (Walker et al., 1994), and can increase intestinal permeability (Ryan et al., 1996).

Exercise, particularly endurance running, has been observed to decrease intestinal permeability (Øktedalen et al., 1992). This represents a compromise in the selective nature of the gut–blood barrier. There is a graded effect of exercise intensity on permeability that becomes significant at ~80% VO_2 max (Pals et al., 1997). It is assumed that this follows decreased blood flow. With severe ischemia, tissue hypoxia can occur which, via altered energy supply, can disturb membrane function and eventually cause cell death.

Effects of CHO Type and Concentration

Gastric Emptying/Secretion CHO concentration is one of the strongest regulators of gastric emptying (Rehrer et al., 1989; Vist & Maughan, 1994) (Figure 43.3). Receptors in the small intestine feed back to inhibit gastric emptying, although the delay is such that, although fluid delivery is reduced, CHO delivery is still greater than with less concentrated solutions (Figure 43.4). CHO type also plays a role, due to effects on osmolarity and viscosity. Use of polymers of increasing chain length will reduce osmolarity at a given concentration. This has particular relevance for beverages containing more than about 13–16% CHO, i.e., when osmolarity is above about 800–1000 mOsmol/l, as high luminal

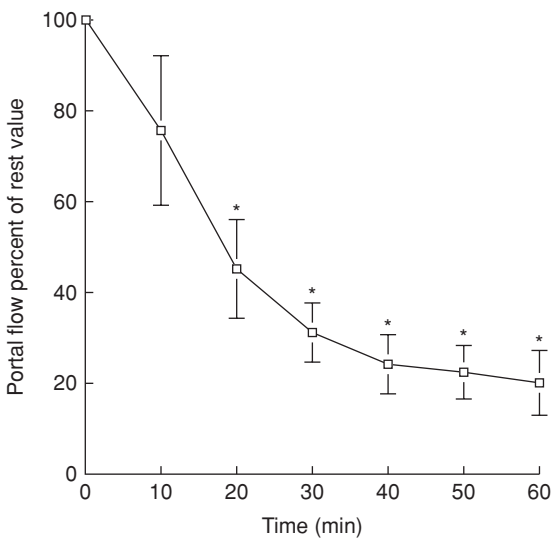

Figure 43.2 Portal vein flow (mean ± SE) at rest and during 70% VO_2 max cycling. A decrease in flow over time was observed (ANOVA, $p = 0.0001$). *Flow less than at rest (Fisher's PLSD, $p < 0.05$). From Rehrer et al. (2001).

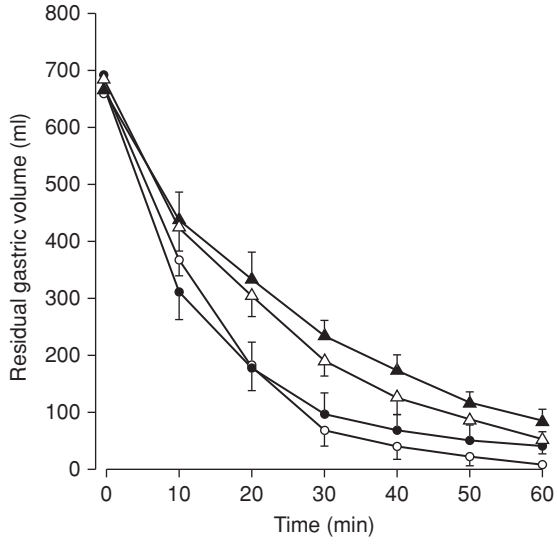

Figure 43.3 Total volume in the stomach after ingesting 600 ml of water (○) or of a 20 g/l (●), 40 g/l (△), or 60 g/l (▲) glucose solution. From Vist and Maughan (1994).

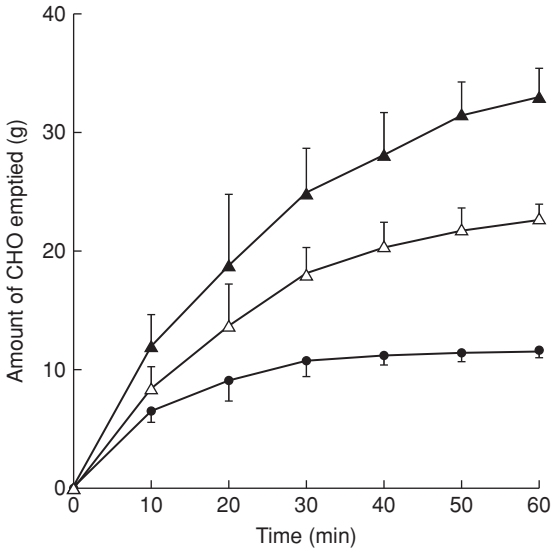

Figure 43.4 The amount of glucose delivered to the small intestine after ingesting 600 ml of 20 g/l (●), 40 g/l (△), or 60 g/l (▲) glucose solution. From Vist and Maughan (1994).

osmolarity leads to net water secretion. Recent research observations also indicate that gastric emptying and delivery of both fluid and CHO can be enhanced by using multiple types of CHO (Jeukendrup & Moseley, 2010) (Figure 43.5).

Intestinal Absorption/Secretion Increasing CHO concentration increases CHO absorption, but raises osmolarity and, as in the stomach, strong hyperosmotic solutions increase water flux into the lumen and thereby reduce the net absorption rate of water. Recent research examining the effects of combining different types of CHO has also demonstrated that the absorption rate of both fluid and CHO can be enhanced (Jeukendrup & Moseley, 2010) and that the oxidation rate of exogenous CHO is increased (Jentjens et al., 2004). This is due to the recruitment of different transporter systems involved in the absorption of different CHO forms (Jeukendrup & Moseley, 2010) (Figure 43.5).

Ingesting CHO enhances blood flow to the splanchnic region, but when combined with exercise this response is ameliorated (Rehrer et al., 2005).

Effects of Other Nutrients, Ingredients, and Meal or Beverage Characteristics

Volume Volume strongly influences gastric emptying (Noakes et al., 1991). Increasing the volume either in a single bolus or by repeated ingestion after an initial bolus enhances the gastric emptying rate. At a certain point, although the absolute amount

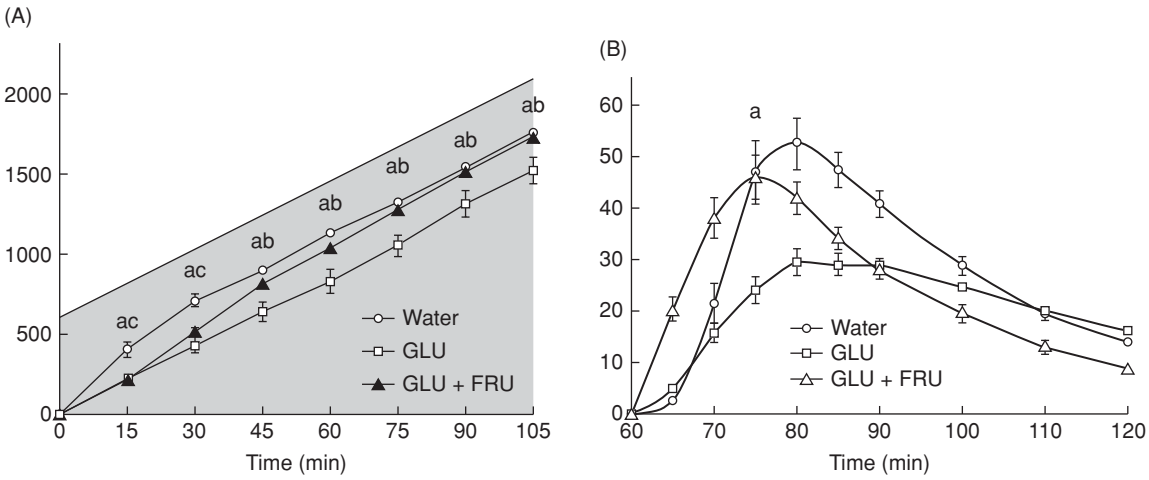

Figure 43.5 (A) Cumulative gastric emptying (mean ± SE) of 600 ml at onset of exercise and 203 ml/15 min thereafter of water, 8.6% glucose (GLU), and 8.6% glucose + fructose, 2:1 ratio (GLU + FRU). "a" denotes water different from GLU; b, GLU different from GLU + FRU; c, water different from GLU + FRU. (B) Corrected (δ per mil vs. PDB) breath $^{13}CO_2$ (mean ± SE) following ingestion of 150 ml ^{13}C-acetate with water, 8.6% GLU and 8.6% GLU + FRU. "a" denotes GLU + FRU time to peak significantly earlier. From Jeukendrup and Moseley (2010).

emptied still increases, a smaller proportion of that ingested is emptied, and larger gastric residues can lead to discomfort during exercise (Mitchell & Voss, 1991). Although tolerance varies between individuals, the fluid ingestion rate that triggers distress appears for most to be around 1000–1200 ml/h (Mitchell & Voss, 1991). It is possible that this is adaptable. Lambert et al. (2008) observed increasing stomach comfort with repeated running trials when fluid intake matched sweat volume (mean around 1250 ml), but no alteration in emptying rate was observed. For an overview of the gastric emptying characteristics of selected sport drinks see Murray et al. (1999). A review of some of the more important regulatory components and characteristics not covered previously follows.

Addition of Protein or Fat The addition of protein or fat inhibits gastric emptying in proportion to the energy density (Calbet & MacLean, 1997), thus reducing the rate of water availability and any CHO contained in the meal. The latter can thus influence the blood glucose response and potentially the rate of downstream metabolism of exogenous CHO.

Solids versus Liquids Liquids empty from the stomach more quickly than solids (Notivol et al., 1984) and, in a graded fashion, the smaller the particle size the quicker the emptying (Holt et al., 1982).

Beverage Temperature Beverage temperature has little effect on gastric emptying, particularly within the range of temperatures at which a beverage is typically ingested, as there is rapid equilibration to body temperature. Temperature may alter palatability and provide a small thermoregulatory effect (see Chapter 14).

Minor Ingredients At concentrations typically contained in sports drinks, sodium, combined with CHO, does not appear to have a significant effect on gastric emptying. Although Na^+ is co-transported with glucose across the intestinal membrane, it is not necessary to add sodium to a beverage to optimize absorption of water or CHO as it can be secreted. At very high concentrations, such as that used in sodium loading pre-hydration regimens, NaCl can result in increased gastric secretions and discomfort, and therefore some of the sodium is typically provided by Na citrate (Greenleaf et al., 1997).

Caffeine, in addition to being an ergogenic aid, has been observed to increase glucose absorption across the intestine without altering orocecal transit time (van Nieuwenhoven et al., 2000) and can enhance exogenous CHO oxidation (Yeo et al., 2005).

Carbonation does not affect the gastric emptying of a CHO beverage during exercise (Ryan et al., 1991). Anecdotal accounts of athletes letting carbonated beverages "go flat" before ingestion indicates that they may still not be ideal, possibly due to ease of consumption or increased eructation.

Buffers can enhance gastric secretions and their inclusion in sports drinks may increase gastric residue (Rehrer et al., 1993). Numerous other additives and preservatives may be present in beverages and an individual's tolerance may vary.

Part II: GI Dysfunction during Exercise

Although light/moderate levels of physical activity improve GI symptoms in functional GI disorders (Johannesson et al., 2011), higher exercise levels can induce adverse GI symptoms in healthy individuals. GI disturbances that limit exercise performance and participation are experienced by recreational and elite endurance athletes, but their causes are not well understood.

Prevalence and Type of GI Symptoms

The reported prevalence of GI symptoms among athletes varies widely but their frequency and severity are dependent upon intensity and duration of exercise, and are more frequent in hot environments. Variation in incidence may reflect the imprecise classification systems used to assess symptoms. Furthermore, the use of self-reported questionnaires to identify the frequency of symptoms may be subject to reporting bias; individuals who suffer from GI symptoms during exercise may be more likely to respond.

A recent study suggests that a similar percentage of individuals suffer GI discomfort during running and cycling (Pfeiffer et al., 2012). Generally, a higher prevalence of GI disturbances among long-distance runners has been reported, perhaps due to the differing physiological demands or mechanical

differences of these exercise modes (see Mechanical Factors). In support of this, triathletes report that most GI symptoms occur during the running phase (Wright et al., 2011). Other risk factors include younger age (ter Steege et al., 2008; Wright et al., 2011), female gender (ter Steege et al., 2008), and training status (Peters et al., 1999) although the last is not universally reported (Wright et al., 2011). These factors may partially account for published differences in prevalence.

GI Symptoms

GI symptoms can be classified into upper and lower GI symptoms (Table 43.1). Exercise mode may be an important determinant of the site of GI discomfort; runners tend to experience lower GI symptoms such as abdominal cramps and diarrhea (termed "runner's trots"; Fogoros, 1980), while cyclists generally experience upper GI symptoms such as belching.

Most GI symptoms during exercise are mild and transient, resolving without intervention. Although uncommon, more serious complications such as ischemic colitis ("runner's colitis") can occur with endurance exercise and may require clinical intervention. Regardless of severity, GI symptoms experienced during exercise can impair individual performance. It is necessary to understand the causes and identify individuals at risk to enable athletes to exercise to their maximum potential. In addition, occult blood loss can also occur; this does not cause symptoms, but consequent anemia or iron deficiency may impair performance.

Table 43.1 Commonly experienced upper and lower gastrointestinal symptoms. The site of origin can be broadly subdivided into upper (gastroesophogeal) and lower (intestinal)

Upper	Lower
• Nausea	• Bloating
• Vomiting	• Abdominal cramps
• Belching	• Diarrhea
• Reflux	• Flatulence
• Anorexia	• Urge to defecate

Mechanisms

Underlying mechanisms are speculative due to the difficulty in physiological measurement during exercise and the transient, probably multifactorial nature of GI symptoms. Several hypotheses have been proposed.

Mechanical Factors Running appears to cause the highest frequency of GI symptoms such as diarrhea, thus it is likely that factors related to the mechanics of running underpin these symptoms. During running, acceleration/deceleration of the body is more than twice as high as in cycling (Rehrer & Meijer, 1991) and the movement of the gut may contribute to GI symptoms. GI bleeding after exercise is surprisingly common in distance runners (incidence ranging from 8% to 85%; Sanchez et al., 2006), but is usually transient and occult. It is probably due to mechanical trauma because the prevalence is low in cyclists and absent in walkers (de Oliveira & Burini, 2009). Postural factors may explain the predominance of upper GI symptoms among cyclists because of the forward position adopted toward the handlebars increasing intra-abdominal pressure (Ho, 2009).

Physiological Mechanisms Blood flow to the GI tract decreases during strenuous exercise. During endurance events, this persists over time, rendering the mucosa susceptible to ischemic injury (de Oliveira & Burini, 2009). GI symptoms may be caused by ischemia. More severe clinical cases of ischemic colitis may follow from hypotension/hypovolemia consequent upon dehydration or bleeding (Elder et al., 2009). Some degree of dehydration is likely in the exercising endurance athlete, especially in the heat, predisposing to ischemia in the colon in particular. Prior training at altitude may exacerbate this via hypercoagulability secondary to polycythemia as described in one report (Lucas & Schroy, 1998). This suggests that exercising in extreme environments (heat, altitude) presents additional physiological challenges (acute and chronic) to GI function. Prolonged (10–17 hours) exercise showed changes in pre- to post-race splanchnic hemodynamics (measured by Doppler ultrasound) and GI symptoms (Wright et al., 2011), although there were no differences between

symptomatic and asymptomatic groups suggesting factors other than impaired blood flow cause GI discomfort during prolonged exercise. However, measurements of splanchnic hemodynamics were made ~1.5 hours after the race and a rapid recovery of splanchnic perfusion after hypoperfusion with 60 minutes of intensive cycling has been observed (van Wijk et al., 2011). Although duration of exercise was much longer in the study by Wright et al. (2011), it is possible that differences between groups, were absent by time of measurement due to the time lag.

Maximal acute aerobic exercise and strenuous long-duration exercise can both result in endotoxemia. Inappropriate luminal endotoxin access to the circulation may contribute to diarrhea, vomiting, and nausea experienced by athletes during exercise (de Oliveira & Burini, 2009). Supplementation with ascorbic acid (vitamin C) has been reported to abolish this suggesting a free-radical-mediated event (Ashton et al., 2003).

It has been suggested that "runner's trots" have a colonic origin. Increases in motility and transit have been described, but other research suggests that diarrhea is not related to accelerated colonic transit with similar (Kayaleh et al., 1996; Rao et al., 2004) or decreased transit times in symptomatic athletes (van Nieuwenhoven et al., 2004).

Nutrition/Dehydration High CHO intakes during exercise have been correlated with nausea and flatulence but symptoms are usually mild, so athletes should not limit CHO ingestion when it can enhance performance. CHO combinations may improve GI comfort as well as increase exogenous CHO oxidation (Jentjens et al., 2004), with an optimum fructose to maltodextrin ratio of 0.8 (O'Brien & Rowlands, 2011). Although high CHO intake may be linked to GI distress during exercise, athletes who adhere to current recommendations are less likely to suffer from GI distress.

Content and timing of prior food ingestion can influence the incidence of GI symptoms during long-duration events. All individuals who experienced intestinal cramps during a triathlon reported having eaten a fiber-rich meal prior to the race, compared with just 10% of those individuals who reported no symptoms (Rehrer et al., 1992). Exercise undertaken in the postprandial state can worsen symptoms of gastroesophogeal reflux (Parmalee-Peters & Moeller, 2004) with symptoms further exacerbated by high intensity exercise.

Fermentable oligosaccharides, disaccharides, monosaccharides, and polyols, (FODMAPs) are short-chain carbohydrates, such as fructose, that are widespread in the diet and poorly absorbed in the small intestine. FODMAPs can lead to alterations in fluid content and colonic bacterial fermentation triggering functional gut symptoms in some healthy people and especially in those with irritable bowel syndrome. Restricting FODMAP intake can reduce symptoms (Gibson & Shepherd, 2010). Present in everyday foods such as milk, yoghurt, and fruit, FODMAPs may also be present in sports drinks, powders, and gels.

Pharmaceutical Triggers Medications or supplements may contribute to GI symptoms during exercise. NSAIDs are commonly used to reduce pain and inflammation, but exercise-induced dehydration can potentially exacerbate problems associated with NSAID use, including decreased splanchnic blood flow, increased intestinal permeability, and GI bleeding (Sanchez et al., 2006).

Legal ergogenic aids are used by athletes even in short-duration, high intensity exercise. Sodium bicarbonate ingestion can enhance performance by increasing extracellular buffering capacity but can result in GI symptoms including nausea, vomiting, and diarrhea (Burke & Pyne, 2007). A recent study suggests these symptoms can be minimized by ingesting sodium bicarbonate with a high carbohydrate meal 2–2.5 hours prior to exercise (Carr et al., 2011).

Other One of the strongest predictors of GI complaints during exercise is a prior history of GI distress (Pfeiffer et al., 2012). The anatomy of the colon is very variable, so some areas may be more vulnerable to ischemia than others. Reports of ischemic colitis are relatively uncommon because athletes cease exercising before progressing that far. Some might use discomfort as a gauge for how hard they are exercising and thus may ignore warning symptoms of more serious GI dysfunction.

GI dysfunction in visitors to high altitude is commonplace and may be partly due to different food

habits and poor sanitation. Common symptoms of nausea and vomiting may relate to acute mountain sickness (AMS). High altitude anorexia is also a symptom of AMS but can persist when other symptoms of AMS have resolved. This may relate to changes in appetite regulatory hormones. Exercising at altitude may therefore present a dual burden of hypoxia and exercise, both eliciting GI symptoms.

Clark (2011) reported a case of a marathon runner who had previously undergone bariatric surgery. Given the increasing numbers of these procedures, we may see increasing reports of GI problems during exercise consequent upon bariatric surgery.

Management of GI Symptoms While fixed intrinsic factors such as younger age and female gender may predispose to GI distress, extrinsic factors such as environmental conditions, exercise mode/intensity, and nutrition can be managed by an individual. If an athlete seeks medical advice, it is essential to identify possible other causes and eliminate any pathology before attributing to exercise. Most gastroenterologists are unaccustomed to evaluating athletes, while exercise physiologists may not have the clinical expertise or appropriate diagnostic techniques at their disposal.

There are various methods (nonpharmacological and pharmacological) utilized by athletes in an attempt to eliminate or minimize symptoms they experience.

Appropriate Training Although recreational and elite athletes are equally likely to suffer GI complaints, appropriate training for recreational athletes is advisable because training can reduce extreme vascular shifts away from the gut; thus, in trained individuals, splanchnic blood flow is greater at any given workload (Murray, 2006). However, trained individuals may exert themselves to a greater extent during training and competition than less trained individuals and, thus, could experience comparable reductions in gut blood flow.

Nutraceutical/Pharmacological Treatment Probiotics are "live microorganisms which when administered in adequate amounts confer a health benefit on the host" (WHO, 2001) in foods or preparations. The most common are lactic acid producing bacteria from the *Lactobacillus* and *Bifidobacterium* genera. Probiotics may offer some relief from some GI problems and may be prophylactic. From a systematic review of 63 studies on the use of probiotics to treat infectious diarrhea, it was concluded that probiotics shortened the duration of diarrhea (by ~25 hours), reduced the risk of it lasting ≥4 days by 59%, and also reduced stool frequency (Allen et al., 2010). In another review of 16 studies, it was concluded that high doses (5–40 billion CFU/day) of *Lactobacillus rhamnosus* and/or *Saccharomyces boulardii* may reduce the incidence of diarrhea after antibiotic treatment (Johnston et al., 2011). Studies are needed to assess whether these will affect exercise-induced bowel disturbance. Prebiotics (nondigestible, fermentable CHO, typically oligosaccharides) are also used to enhance "healthy" intestinal flora. In mice, an increase in gut microbes, decrease in gut permeability, cytokines and markers of inflammation, and oxidative stress were observed in association with enhanced GLP-2 (a proglucagon-derived peptide) (Cani et al., 2009). Effects of probiotics on general immunity are less consistent. Although there are general population studies indicating that probiotics enhance resistance to upper respiratory tract infections, studies with athletes are less convincing (for review and a discussion of mechanisms of action, see West et al. (2009) and Figure 43.6). The efficacy of particular strains, prophylactically or in response to infection, dosage, timing, and duration deserve further attention before

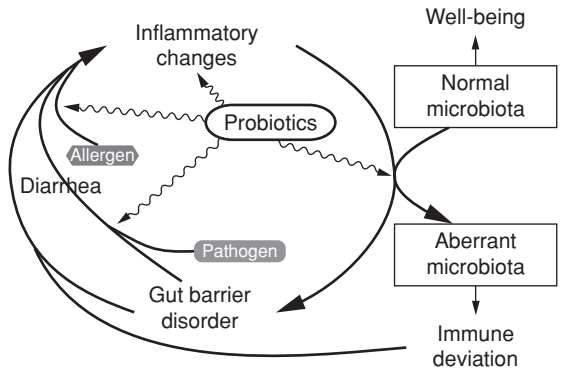

Figure 43.6 Targets for probiotic therapy in clinical conditions with impaired mucosal barrier function, particularly manifested by infectious and inflammatory disease. From Saarela et al. (2002).

exact recommendations can be made. Nevertheless, it appears that for the traveling athlete at risk of developing gastroenteritis, a supply of probiotics, and possibly pre-loading prior to travel, could reduce the duration of, if not prevent, infection.

Recent research has also shown that 14 days of supplementation with bovine colostrum can reduce the increase in gut permeability after acute, vigorous exercise possibly by mechanisms including reducing temperature-induced apoptosis (Marchbank et al., 2011). Although research is limited, this study suggests that colostrum supplementation may be beneficial in maintaining gut stability during exercise and could help prevent GI discomfort.

For elite athletes who recurrently suffer diarrhea associated with competition, the use of medication such as loperamide, which suppresses motility, is an accepted method of prevention. Loperamide and oral rehydration solutions are also helpful in episodes of traveler's diarrhea or viral gastroenteritis, although loperamide should be avoided in bacterial dysentery or enterocolitis due to more pathogenic organisms such as *Salmonella*, *Shigella*, or *Campylobacter.*

Nutrition and Hydration An awareness of nutrition/hydration appropriate to the environmental conditions is important to minimize dehydration and hypovolemia, which can further reduce gut blood flow during exercise. Athletes often have to compromise between fluid delivery and carbohydrate delivery. The type and concentration of CHO should be formulated to keep osmolarity as low as possible, particularly when splanchnic blood flow may be compromised.

Summary/Recommendations

Knowledge of GI function and limitations thereof provide the basis for optimizing nutritional supplementation for the competitive athlete. Gastric emptying varies with beverage or meal characteristics and can be influenced by exercise intensity. The effects of volume to stimulate and increasing CHO concentration to inhibit are key regulatory factors. Carbohydrate type not only can influence GI function via altering the osmolality for a given concentration but by mixing different CHO that have varying transporters, the intestinal absorption, gastric emptying, and oxidation can be enhanced above that observed with a single CHO. When competition is such that endogenous glycogen reserves can be limiting for performance and a high rate of CHO oxidation is demanded, mixing CHO may help maximize the rate of digestion and oxidation. Increasing CHO concentration, although delaying rate of fluid emptied, also enhances CHO availability. With increasing concentration, osmolarity also increases and stimulates GI secretions, which can reduce net water uptake. With these two pieces of information, one can conclude that to maximize CHO availability and oxidation it would be prudent to have one of the CHO be a long-chain polymer which for a given concentration has a lower osmolarity. To maximize fluid (water) digestion and absorption, a lower CHO concentration and a low osmolarity are advised.

Although high intensity exercise does decrease gastric emptying and may decrease absorption, the magnitude of effect would not warrant avoiding fluid intake during exercise that substantially depletes reserves. As blood flow to the splanchnic region is reduced during intensive exercise, and dehydration/hyperthermia can further reduce blood flow and has been shown to decrease gastric emptying rate, it is advisable to anticipate losses and consume fluids before severe hypohydration occurs.

Reduced blood flow to the splanchnic region has been implicated as a major mechanism underpinning the GI symptoms that are commonly experienced by athletes during exercise. Exercise mode might also predict the severity and type of GI symptom (upper/lower) experienced. Most symptoms are transient and resolve shortly after cessation of exercise, but they can be debilitating, particularly during competition.

Although certain factors related to the mechanics of individual sports cannot be modified to minimize GI symptoms during exercise, other factors can be adapted in an attempt to minimize GI symptoms. Appropriate training is important, as is proper acclimatization to the environmental conditions. Athletes should be aware of foods that may trigger GI symptoms and should plan a personalized

nutrition strategy. Some individuals may have to minimize or avoid FODMAPs to minimize gut symptoms. Athletes are advised to plan and practice their individualized nutrition/hydration strategies during training to enable them to establish the most effective types and timings of food/drink ingestion that aid performance and minimize GI discomfort. Normal dietary habits may be hard to maintain when training or competing at altitude, or in countries where there may be risk of GI illnesses due to consumption of water/food where there are poor hygiene standards. Therefore, athletes should plan ahead and be aware of what foods are available, be wary of whether drinks/foods contain water from local supply and if necessary take foods with them so their diet while away can be supplemented with nonperishable foodstuffs they are accustomed to. Probiotics may provide some benefit and shorten the duration of symptoms. The use of legal ergogenic aids and pharmaceuticals prior to exercise can cause or exacerbate GI symptoms, and should also be trialed in training.

Application of nutritional principles based on knowledge of GI function and environmental and physiological constraints is a key element in delivering optimal performance. Further developments in optimizing nutrient delivery should prove possible as our understanding of nutrient sensing and transport systems develops. A key area will be to understand and characterize physiological variations in individual athletes, so that personalized advice and tailored regimens can be rationally designed. Coupled with a more detailed understanding of the physiological and environmental factors that lead to GI symptoms and distress, it should prove possible to enhance human performance while minimizing the negative impact of exercise and training on GI function.

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References

Allen, S.J., Martinez, E.G., Gregorio, G.V., et al. (2010) Probiotics for treating acute infectious diarrhoea. *Cochrane Database of Systematic Reviews* 11 (Art. No.: CD003048). doi:10.1002/14651858.CD003048.pub3

Ashton, T., Young, I.S., Davison, G.W., et al. (2003) Exercise-induced endotoxemia: the effect of ascorbic acid supplementation. *Free Radical Biology & Medicine* 35, 284–291.

Barclay, G.R. & Turnberg, L.A. (1988) Effect of moderate exercise on salt and water transport in the human jejunum. *Gut* 29, 816–820.

Beaumont, W. (1838) *Experiments and Observations on the Gastric Juice and the Physiology of Digestion*. MacLachlan & Stewart, Edinburgh, UK.

Beckers, E.J., Rehrer, N.J., Saris, W.H., et al. (1991) Daily variation in gastric emptying when using the double sampling technique. *Medicine & Science in Sports & Exercise* 23, 1210–1212.

Brown, B.P., Ketelaar, M.A., Schulze-Delrieu, K., et al. (1994) Strenuous exercise decreases motility and cross-sectional area of human gastric antrum. *Digestive Diseases and Sciences* 39, 940–945.

Burke, L.M. & Pyne, D.B. (2007) Bicarbonate loading to enhance training and competitive performance. *International Journal of Sports Physiology and Performance* 2, 93–97.

Calbet, J.A. & Maclean, D.A. (1997) Role of caloric content on gastric emptying in humans. *Journal of Physiology* 498(Pt 2), 553–559.

Cani, P.D., Possemiers, S., Van de Wiele, T., et al. (2009) Changes in gut microbiota control inflammation in obese mice through a mechanism involving GLP-2-driven improvement of gut permeability. *Gut* 58, 1091–1103.

Carr, A.J., Slater, G.J., Gore, C.J., et al. (2011) Effect of sodium bicarbonate on [HCO3-], pH, and gastrointestinal symptoms. *International Journal of Sports Nutrition and Exercise Metabolism* 21, 189–194.

Carrio, I., Estorch, M., Serra-Grima, R., et al. (1989) Gastric emptying in marathon runners. *Gut* 30, 152–155.

Clark, N. (2011) Case study: nutrition challenges of a marathon runner with a gastric bypass. *International Journal of Sports Nutrition and Exercise Metabolism* 21, 515–519.

Cordain, L., Latin, R.W., & Behnke, J.J. (1986) The effects of an aerobic running program on bowel transit time. *Journal of Sports Medicine and Physical Fitness* 26, 101–104.

Datz, F.L., Christian, P.E., & Moore, J. (1987) Gender-related differences in gastric emptying. *Journal of Nuclear Medicine* 28, 1204–1207.

Degen, L.P. & Phillips, S.F. (1996) Variability of gastrointestinal transit in healthy women and men. *Gut* 39, 299–305.

de Oliveira, E.P. & Burini, R.C. (2009) The impact of physical exercise on the gastrointestinal tract. *Current Opinion in Clinical Nutrition and Metabolic Care* 12, 533–538.

Elder, K., Lashner, B., & Al Solaiman, F. (2009) Clinical approach to colonic ischemia. *Cleveland Clinic Journal of Medicine* 76, 401–409.

Fogoros, R. (1980) 'Runner's trots'. Gastrointestinal disturbances in runners. *Journal of the American Medical Association* 243, 1743–1744.

Fordtran, J.S. & Saltin, B. (1967) Gastric emptying and intestinal absorption during prolonged severe exercise. *Journal of Applied Physiology* 23, 331–335.

Gibson, P. & Shepherd, S. (2010) Evidence-based dietary management of functional gastrointestinal symptoms: The FODMAP approach. *Journal of Gastroenterology and Hepatology* 25, 252–258.

Gisolfi, C.V., Spranger, K.J., Summers, R.W., et al. (1991) Effects of cycle exercise on intestinal absorption in humans. *Journal of Applied Physiology* 71, 2518–2527.

Greenleaf, J.E., Looft-Wilson, R., Wisherd, J.L., et al. (1997) Pre-exercise hypervolemia and cycle ergometer endurance in men. *Biology of Sport* 14, 103–114.

Hellström, P.M., Grybäck, P., & Jacobsson, H. (2006) The physiology of gastric emptying. *Best Practice & Research Clinical Anaesthesiology* 20, 397–407.

Ho, G. (2009) Lower gastrointestinal distress in endurance athletes. *Current Sports Medicine Reports* 8, 85–91.

Holt, S., Reid, J., Taylor, T.V., et al (1982) Gastric emptying of solids in man. *Gut* 23, 292–296.

Houmard, J.A., Egan, P.C., Johns, R.A., et al. (1991) Gastric emptying during 1 h of cycling and running at 75% VO_2 max. *Medicine & Science in Sports & Exercise* 23, 320–325.

Jentjens, R.L.P.G., Achten, J. & Jeukendrup, A.E. (2004) High oxidation rates from combined carbohydrates ingested during exercise. *Medicine & Science in Sports & Exercise* 36, 1551–1558.

Jeukendrup, A.E. & Moseley, L. (2010) Multiple transportable carbohydrates enhance gastric emptying and fluid delivery. *Scandinavian Journal of Medicine & Science in Sports* 20, 112–121.

Johannesson, E., Simrén, M., Strid, H., et al. (2011) Physical activity improves symptoms in irritable bowel syndrome: a randomised controlled trial. *American Journal of Gastrotenterology* 106, 915–922.

Johnston, B.C., Goldenberg, J.Z., Vandvik, P.O., et al. (2011) Probiotics for the prevention of pediatric antibiotic-associated diarrhea. *Cochrane Database of Systematic Reviews* 11 (Art. No.: CD004827). doi:10.1002/14651858.CD004827.pub3

Kayaleh, R., Meshkinpour, H., Avinashi, A., et al. (1996) Effect of exercise on mouth-to-cecum transit in trained athletes: a case against the role of runners' abdominal bouncing. *Journal of Sports Medicine and Physical Fitness* 36, 271–274.

Kayser, B. (1994) Nutrition and energetic of exercise at altitude. Theory and possible practical implications. *Sports Medicine* 16, 309–323.

Koffler, K.H., Menkes, A., Redmond, R.A., et al. (1992) Strength training accelerates gastrointestinal transit in middle-aged and older men. *Medicine & Science in Sports & Exercise* 24, 415–419.

Lambert, G.P., Lang, J., Bull, A., et al. (2008) Fluid tolerance while running: effect of repeated trials. *International Journal of Sports Medicine* 29, 878–882.

Leiper, J.B., Broad, N.P., & Maughan, R.J. (2001) Effect of intermittent high-intensity exercise on gastric emptying in man. *Medicine & Science in Sports & Exercise* 33, 1270–1278.

Liu, F., Kondo, T., & Toda, Y. (1993) Brief physical inactivity prolongs colonic transit time in elderly active men. *International Journal of Sports Medicine* 14, 465–467.

Lu, C.-L., Shidler, N., & Chen, J.D.Z. (2000) Enhanced postprandial gastric myoelectrical activity after moderate-intensity exercise. *American Journal of Gastroenterology* 95, 425–431.

Lucas, W. & Schroy, P. (1998) Reversible ischemic colitis in a high endurance athlete. *American Journal of Gastroenterology* 93, 2231–2234.

Marchbank, T., Davison, G., Oakes, J.R., et al. (2011) The nutriceutical bovine colostrum truncates the increase in gut permeability caused by heavy exercise in athletes. *American Journal of Physiology—Gastrointestinal and Liver Physiology* 300, G477–G484.

Maughan, R., Leiper, J., & Mcgaw, B. (1990) Effects of exercise intensity on absorption of ingested fluids in man. *Experimental Physiology* 75, 419–421.

Mitchell, J.B. & Voss, K.W. (1991) The influence of volume on gastric emptying and fluid balance during prolonged exercise. *Medicine & Science in Sports & Exercise* 23, 314–319.

Murray, R. (2006) Training the gut for competition. *Current Sports Medicine Reports* 5, 161–164.

Murray, R., Bartoli, W., Stofan, J., et al. (1999) A comparison of the gastric emptying characteristics of selected sports drinks. *International Journal of Sport Nutrition* 9, 263–274.

Neufer, P., Young, A., & Sawka, M. (1989a) Gastric emptying during exercise: effects of heat stress and hypohydration. *European Journal of Applied Physiology and Occupational Physiology* 58, 433–439.

Neufer, P.D., Young, A.J., & Sawka, M.N. (1989b) Gastric emptying during walking and running: effects of varied exercise intensity. *European Journal of Applied Physiology & Occupational Physiology* 58, 440–445.

Noakes, T.D., Rehrer, N.J., & Maughan, R.J. (1991) The importance of volume in regulating gastric emptying. *Medicine & Science in Sports & Exercise* 23, 307–313.

Notivol, R., Carrio, I., Cano, L., et al. (1984) Gastric emptying of solid and liquid meals in healthy young subjects. *Scandinavian Journal of Gastroenterology* 19, 1107–1113.

O' Brien, W.J. & Rowlands, D.S. (2011) Fructose-maltodextrin ratio in a carbohydrate–electrolyte solution differentially affects exogenous carbohydrate oxidation rate, gut comfort, and performance. *American Journal of Physiology. Gastrointestinal and Liver Physiology* 300, G181–G189.

O'donovan, D., Hausken, T., Lei, Y., et al. (2005) Effect of aging on transpyloric flow, gastric emptying, and intragastric distribution in healthy humans—impact on glycemia. *Digestive Diseases and Sciences* 50, 671–676.

Øktedalen, O., Lunde, O.C., Opstad, P.K., et al. (1992) Changes in the gastrointestinal mucosa after long-distance running. *Scandinavian Journal of Gastroenterology* 27, 270–274.

Pals, K.L., Chang, R.-T., Ryan, A.J., et al. (1997) Effect of running intensity on intestinal permeability. *Journal of Applied Physiology* 82, 571–576.

Parmalee-Peters, K. & Moeller, J.L. (2004) Gastroesophageal reflux in athletes. *Current Sports Medicine Reports* 3, 107–111.

Peters, H. P., Bos, M., Seebregts, L., et al. (1999) Gastrointestinal symptoms in long-distance runners, cyclists, and triathletes: prevalence, medication, and etiology. *American Journal of Gastroenterology* 94, 1570–1581.

Pfeiffer, B., Stellingwerff, T., Hodgson, A.B., et al. (2012) Nutritional intake and gastrointestinal problems during competitive endurance events. *Medicine & Science in Sports & Exercise* 44, 344–351.

Rao, K. A., Yazaki, E., Evans, D. F., et al. (2004) Objective evaluation of small bowel and colonic transit time using pH telemetry in athletes with gastrointestinal symptoms. *British Journal of Sports Medicine* 38, 482–487.

Rehrer, N.J. & Meijer, G.A. (1991) Biomechanical vibration of the abdominal region during running and

bicycling. *Journal of Sports Medicine and Physical Fitness* 31, 231–234.
Rehrer, N.J., Beckers, E., Brouns, F., et al. (1989) Exercise and training effects on gastric emptying of carbohydrate beverages. *Medicine & Science in Sports & Exercise* 21, 540–549.
Rehrer, N.J., van Kemenade, M., Meester, W., et al. (1992) Gastrointestinal complaints in relation to dietary intake in triathletes. *International Journal of Sport Nutrition* 2, 48–59.
Rehrer, N.J., Beckers, E.J., Brouns, F., et al. (1993) Effects of electrolytes in carbohydrate beverages on gastric emptying and secretion. *Medicine & Science in Sports & Exercise* 25, 42–51.
Rehrer, N.J., Smets, A., Reynaert, H., et al. (2001) Effect of exercise on portal vein blood flow in man. *Medicine & Science in Sports & Exercise* 33, 1533–1537.
Rehrer, N.J., Goes, E., Dugardeyn, C., et al. (2005) Effect of carbohydrate on portal vein blood flow during exercise. *International Journal of Sports Medicine* 26, 171–176.
Rowell, L.B., Blackmon, J.R., & Bruce, R.A. (1964) Indocyanine green clearance and estimated hepatic blood flow during mild to maximal exercise in upright man. *The Journal of Clinical Investigation* 43, 1677–1690.
Ryan, A.J., Navarre, A.E., & Gisolfi, C.V. (1991) Consumption of carbonated and noncarbonated sports drinks during prolonged treadmill exercise in the heat. *International Journal of Sport Nutrition* 1, 225–239.
Ryan, A.J., Chang, R.T., & Gisolfi, C.V. (1996) Gastrointestinal permeability following aspirin intake and prolonged running. *Medicine & Science in Sports & Exercise* 28, 698–705.
Ryan, A.J., Lambert, G.P., Shi, X., et al. (1998) Effect of hypohydration on gastric emptying and intestinal absorption during exercise. *Journal of Applied Physiology* 84, 1581–1588.
Saarela, M., Lahteenmaki, L., Crittenden, R., et al. (2002) Gut bacteria and health foods—the European perspective. *International Journal of Food Microbiology* 78(1–2), 99–117.
Sanchez, L.D., Tracy, J.A., Berkoff, D., et al. (2006) Ischemic colitis in marathon runners: a case-based review. *Journal of Emergency Medicine* 30, 321–326.
ter Steege, R.W., Van der Palen, J., & Kolkman, J.J. (2008) Prevalence of gastrointestinal complaints in runners competing in a long-distance run: an internet-based observational study in 1281 subjects. *Scandinavian Journal of Gastroenterology* 43, 1477–1482.
Thune, I. & Lund, E. (1996) Physical activity and risk of colorectal cancer in men and women. *British Journal of Cancer* 73, 1134–1140.
Urbain, J.L. & Charkes, N.D. (1995) Recent advances in gastric emptying scintigraphy. *Seminars in Nuclear Medicine* 25, 318–325.
van Nieuwenhoven, M.A., Brummer, R.M., & Brouns, F. (2000) Gastrointestinal function during exercise: comparison of water, sports drink, and sports drink with caffeine. *Journal of Applied Physiology* 89, 1079–1085.
van Nieuwenhoven, M.A., Brouns, F., & Brummer, R.-J. M. (2004) Gastrointestinal profile of symptomatic athletes at rest and during physical exercise. *European Journal of Applied Physiology* 91, 429–434.
van Wijk, K., Lenaerts, K., van Loon, L.J., et al. (2011) Exercise-induced splanchnic hypoperfusion results in gut dysfunction in healthy men. *PLoS One* 6, e22366.
Vist, G.E. & Maughan, R.J. (1994) Gastric emptying of ingested solutions in man: effect of beverage glucose concentration. *Medicine & Science in Sports & Exercise* 26, 1269–1273.
Walker, R.J., Fawcett, J.P., Flannery, E.M., et al. (1994) Indomethacin potentiates exercise-induced reduction in renal hemodynamics in athletes. *Medicine & Science in Sports & Exercise* 26, 1302–1306.
Wang, Y., Kondo, T., Suzukamo, Y., et al. (2010) Vagal nerve regulation is essential for the increase in gastric motility in response to mild exercise. *Tohoku Journal of Experimental Medicine* 222, 155–163.
West, N.P., Pyne, D.B., Peake, J.M., et al. (2009) Probiotics, immunity and exercise: a review. *Exercise Immunology Review* 15, 107–126.
WHO (2001) Health and Nutritional Properties of Probiotics in Food including Powder Milk with Live Lactic Acid Bacteria. Report of a Joint FAO/WHO Expert Consultation on Evaluation of Health and Nutritional Properties of Probiotics in Food Including Powder Milk with Live Lactic Acid Bacteria (October 2001). Food and Agriculture Organization, World Health Organization.
Wright, H., Collins, M., Villiers, R.D., et al. (2011) Are splanchnic haemodynamics related to the development of gastrointestinal symptoms in ironman triathletes? A prospective cohort study. *Clinical Journal of Sports Medicine* 21, 325–338.
Yeo, S.E., Jentjens, R.L.P.G., Wallis, G.A., et al. (2005) Caffeine increases exogenous carbohydrate oxidation during exercise. *Journal of Applied Physiology* 99, 844–850.

Chapter 44

Hyponatremia of Exercise

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Introduction

From the advent of competitive sport until 1969, athletes were advised to avoid drinking any fluids during exercise since it was believed that fluid ingestion would impair exercise performance, in particular, by causing gastrointestinal distress, the so-called "stitch." Athletes in those years actively followed that advice, priding themselves on their ability to run even 26-mile (42-km) marathon races without drinking (Noakes, 2003a). Furthermore, the rules then governing competitive distance running restricted the frequency with which athletes had access to fluid, so that athletes were allowed their first access to fluid only after they had already run 10 miles (16 km) with subsequent access restricted to every 5 miles (8 km).

A South African publication in that year (Wyndham & Strydom, 1969) proposed that the avoidance of drinking during exercise constituted "criminal folly" since it would lead inevitably to dehydration-induced heat stroke. One result was that three decades later athletes were being advised to drink "beyond thirst" by ingesting "as much as tolerable" during exercise (Convertino et al., 1996). The medical consequences of this change in drinking behavior were initially suggested already in 1981 when the first reported case of exercise-associated hyponatremic encephalopathy (EAHE) was reported in a 46-year-old female marathon runner in the 90 km Comrades ultramarathon in South Africa. She presented with the symptoms and signs typical of EAHE as listed in Table 44.1 (Noakes et al., 1985).

In 1991, we provided definitive evidence that EAHE is due to abnormal fluid retention in those who overdrink during prolonged exercise and in which a sodium deficit plays no part (Irving et al., 1991). We established this by showing that the sodium deficits developed by seven athletes who developed EAHE during a 90-km ultramarathon were no greater than the deficits developed by a control group of runners who did not develop exercise-associated hyponatremia (EAH) or EAHE. Naturally, we presumed that this definitive evidence that EAHE is caused purely by an aberrant behavior—drinking to excess during prolonged exercise—would be eagerly embraced by the global medical community and would lead to the immediate disappearance of EAHE as a serious medical threat during exercise. For in our opinion, all this required was that athletes be warned to avoid drinking too much during exercise. Instead, the opposite happened.

For shortly after the publication of this compelling evidence, influential sporting organizations began to advise athletes to drink "as much as tolerable" during exercise without properly warning of the proven dangers of overdrinking. As a result, more than 12 documented deaths from EAHE, an

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Table 44.1 Diagnostic features of exercise-associated hyponatremia (EAH) and hyponatremic encephalopathy (EAHE)

EAH	EAHE
Symptoms	
Nausea	Headache
Vomiting	Altered level of consciousness
Unexplained fatigue	Coma
Impaired exercise performance	Convulsion/seizure
Signs and clinical findings	
Evidence of weight gain	Evidence of weight gain
Serum [Na^+] <135 mmol/l	Serum [Na^+] <130 mmol/l Cerebral edema Pulmonary edema

entirely preventable condition, have been reported in the scientific literature since 1991 (Noakes, 2003b). In addition, I have traced more than 1600 cases of EAHE requiring hospitalization that were reported in the scientific or lay media (Noakes, 2012; Noakes & Vlismas, 2011).

These data confirm that after 1991, EAH and EAHE became the commonest conditions, by far, causing the hospitalization of athletes involved in endurance exercise. Yet, this fact was never properly acknowledged. Instead, hubristic statements from influential scientists promoted the mantra that "dehydration" is the greatest risk for ill-health during prolonged exercise: "The greatest threat to health and well-being during prolonged exercise, especially when performed in the heat, is dehydration" (Gisolfi, 1996); "If strenuous exercise is performed by hypohydrated persons, the medical consequences can be devastating" (Sawka & Montain, 2000); and "an effective hydration strategy can mean the difference between life and death" (Galloway, 1999, p. 188). The latter is of course true but perhaps not in the way the author originally intended it. In fact, the greatest danger to the athlete's health became the use or misuse that he or she makes of the fluids provided at the numerous refreshment stations in these marathon and ultramarathon races.

For there is no historical evidence that the absence of regular drinking during exercise produced any adverse consequences. The first purported case of heat injury in a marathon runner who did not drink during competition—Jim Peters in the 1954 Commonwealth (Empire) Games marathon in Vancouver—may not have been due to heat stroke (Noakes et al., 2008). In contrast, a series of publications in the 1960s showed that marathon runners who drank little or nothing seemed to do rather well, often winning races despite finishing with advanced levels of "dehydration" and high core body temperatures (Buskirk & Beetham, 1960; Muir et al., 1970; Pugh et al., 1967). This outcome would be expected if humans evolved their modern form specifically because of a superior capacity to run prolonged distances in (dry) heat in an arid environment with little or no fluid replacement (Bramble & Lieberman, 2004; Carrier, 1984; Liebenberg, 2006; Nolte et al., 2011).

Humans Evolved as Runners Able to Exercise for Prolonged Periods in Dry Heat with Minimal Fluid Replacement

A novel theory first proposed in the early 1900s (Morris, 1900; Read, 1925) holds that humans evolved our particular biological features—including long legs and short arms; relative hairlessness associated with an unmatched ability to lose heat by sweating; the capacity to run by rotating the upper and lower bodies in opposite directions—because all these adaptations gave early humans the critical biological advantage over other nonsweating mammals with whom we shared the hot African savannah more than 2 million years ago (Heinrich, 2010). Thus, it is proposed that our early hominid ancestors discovered that they could capture nonsweating mammals, especially swifter antelope, by chasing them in the midday heat. Their profuse capacity for sweating allowed early hominids safely to regulate their body temperatures whereas the pursued antelope's brain temperature would finally become too high causing it to stop running and seek shade. The animals actively choose the shade because the hunts occur in extreme heat and in midday. The art of hunting is to be able to track an animal that you cannot see and to choose to follow one

particular animal and to stop running. The theory is that the high-energy diet provided by these antelope allowed the rapid growth of the human brain and hence the development of *Homo sapiens*.

Naturally, the best hunters would be those who were neither overcome by thirst nor incapacitated by the loss of a large volume of body water caused by the heavy sweating during the 3–6 hours required for a successful hunt (Liebenberg, 2006). Like all the great runners of the past (Noakes, 2003a), the most successful hunters would have drunk little since carrying water would have impeded their progress and reduced their probability of success whilst hunting.

Promotion of the Contrary Belief that Humans Are Poorly Adapted to Exercise in the Heat

Two separate events that happened between 1965 and 1969 led to a contrary perception that humans are poorly adapted for exercise in the heat, and are at risk of death from heat stroke if they sweat profusely and do not drink copious amounts of fluid during exercise thereby developing "dangerous dehydration."

First was the development of the world's first sports drink at the University of Florida, beginning in 1965. The drink was developed by a renal physician, Dr Robert Cade, who was certain that fluid ingestion during exercise would prevent the development of "heat" illness, including heat stroke and heat cramps during exercise. But the drink became an overnight commercial success, especially in the United States, because of its alleged capacity to enhance the performance of American football teams, first collegiate and later professional, in the fourth quarter of football matches (Rovell, 2005). Since the product contained only sodium, carbohydrate, and flavorant it did not need to undergo extensive safety and efficacy testing that would have been the case had it been a pharmaceutical product. Yet bizarrely the product was marketed on the basis of medical claims, specifically that it could prevent a variety of medical conditions including exercise-induced muscle cramping and "heat illness," including heat stroke.

Second, a group of South African researchers showed an apparently causal relationship between fluid ingestion during exercise, the prevention of "dehydration," and lower body temperatures in athletes competing in a series of 20-mile (32-km) running races (Wyndham & Strydom, 1969). These researchers concluded that athletes who do not drink sufficient fluids during marathon running are at risk of developing heat stroke and that the international ruling then in place which restricted fluid availability during marathon running was "criminal folly."

Together, these concepts contributed to the novel idea that athletes needed to drink "as much as tolerable" during exercise in order to maximize their performance and to ensure that they do not die from heat stroke (Armstrong et al., 1996; Convertino et al., 1996). Product marketing (Noakes, 2004; Noakes & Speedy, 2007) rapidly led to the universal acceptance that not to drink "as much as tolerable" during exercise was extremely unwise. Soon it became the accepted truism that any weight loss during exercise is detrimental to both health and performance (Armstrong et al., 1996; Convertino et al., 1996).

The defining event, which invited a reassessment of this new "truth," occurred on June 1, 1981 when an athlete competing in the 56-mile (90-km) Comrades Marathon in South Africa was admitted to hospital in an unconscious state having developed a grand mal epileptic seizure. Her serum sodium concentration on hospital admission was 115 mmol/l confirming a diagnosis of EAHE. She regained consciousness after 2 days and was discharged from hospital 4 days later. Subsequent investigation over the next 2 years uncovered three additional cases of EAH and EAHE in South African ultra endurance races. Their case reports were subsequently published in 1985 in a paper entitled "Water intoxication: A possible complication during endurance exercise" (Noakes et al., 1985). In time the term EAH would supercede that of water intoxication (Hew-Butler et al., 2005). The paper drew the following conclusion: "The etiology of this condition appears to be voluntary hyperhydration with hypotonic solutions combined with moderate sweat sodium chloride

losses … advice (on fluid replacement) should be tempered with the proviso that the intake of hypotonic fluids in excess of that required to balance sweat and urine losses … may be hazardous in some individuals."

Next, Dr Anthony Irving hospitalized 8 runners who developed EAHE at the 1988 Comrades Marathon, in order to study their fluid and sodium balance during recovery (Irving et al., 1991). He showed that all these participants had drunk to excess during the race, finishing these events with a fluid excess that ranged between 1.2 and 5.9 kg (3–13 pounds) (Figure 44.1—right columns). In contrast, athletes who did not develop EAH or EAHE lost about 2 liters of fluid during exercise.

However, there was no evidence that subjects with EAHE had incurred a greater sodium deficit than the control runners who completed these races with normal serum sodium concentrations (Figure 44.1—left columns). Thus, we concluded that EAH "results from fluid retention in subjects who ingest abnormally large fluid volumes during prolonged exercise" since this study found "that each of eight subjects who collapsed with the hyponatremia of exercise (mean plasma sodium concentration 122.4 ± 2.2 mM) were fluid overloaded by an amount ranging from 1.22 to 5.92 liters. These fluid volumes are conservative because no allowance was made for insensible water losses during recovery" and that "sodium chloride losses alone cannot explain the hyponatremia of exercise" (Irving et al., 1991). There was also a striking linear relationship with a negative slope between the post-race serum sodium concentrations and the extent of the fluid excesses incurred during exercise by these eight runners (Figure 44.2).

Our finding that subjects with EAHE did not have larger sodium deficits than controls is historically important because this evidence was originally ignored. Instead, theoretical arguments without experimental support (Armstrong, 2000; Montain et al., 2001, 2006) were advanced to explain why some might develop EAH or EAHE as the result of unreplaced sodium deficits incurred during prolonged exercise (Noakes, 2012). However, no study

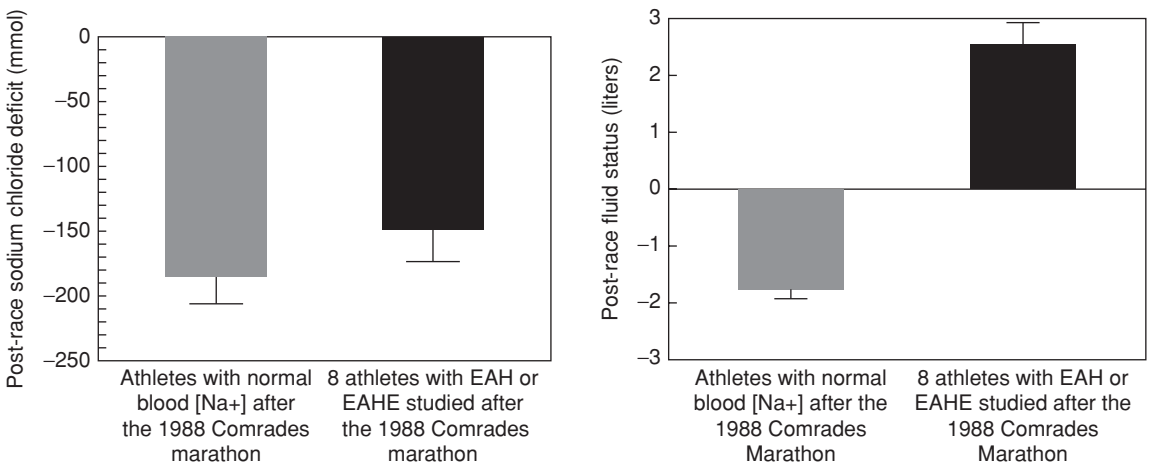

Figure 44.1 The study of Irving et al. (1991) compared sodium chloride (left panels) and fluid (right panels) balance in athletes who finished the 90-km Comrades Marathon with either normal or reduced (EAH and EAHE) serum sodium chloride concentrations (left panel). Whereas sodium chloride losses (left panels) were the same in both groups of athletes, those who maintained their serum sodium concentrations during the race lost about 2 liters of fluid (extreme right panel). In contrast, athletes who developed EAH or EAHE finished with fluid excesses that ranged from 1.2 to 5.9 liters.

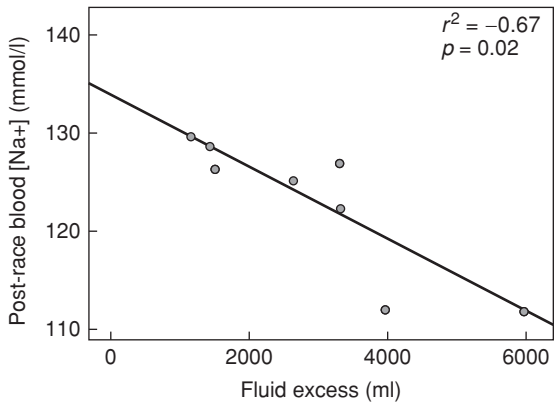

Figure 44.2 There was a striking linear relationship with a negative slope between the post-race serum sodium concentrations of eight ultramarathon runners with EAHE and EAH and the extent of the fluid excess that each incurred during that race.

has yet been able to document a sodium deficit in patients with EAH or EAHE, whereas many studies have either confirmed our original finding (Almond et al., 2005; Dugas & Noakes, 2005; Noakes et al., 2004; Speedy et al., 2000a, 2000c) or shown that sodium chloride ingestion during exercise does not influence the serum sodium concentration in those who do not overconsume fluids during exercise (Hew et al., 2006; Speedy et al., 2002). Since EAH and EAHE are due to abnormal fluid retention, only drinks containing sodium at a concentration well in excess of that found in blood (>140 mmol/l) would theoretically play some role in the prevention of these conditions (Weschler, 2005). In reality, such drinks are unpalatable and likely to induce nausea and vomiting.

Unfortunately, our original findings had little practical impact. For 10 years after the publication of our original paper, a number of influential organizations produced drinking guidelines that promoted the concept that only if athletes drank "as much as tolerable" could they exercise safely (American College of Sports Medicine et al., 2000; Armstrong et al., 1996; Casa et al., 2000; Convertino, et al., 1996). These guidelines failed to emphasize the established finding that the overconsumption during exercise of any fluids, regardless of their sodium concentrations, could have fatal consequences (Noakes, 2004).

This danger was first properly acknowledged only after the 2002 Boston Marathon at which the next two critical events occurred. First, Dr Cynthia Lucero died as a result of drinking "large quantities" of an electrolyte-containing sports drink during the race (Smith, 2002). Second, residents from the Children's Hospital at Harvard Medical School chose that same race to study the factors associated with EAH and EAHE in runners in that specific race (Almond et al., 2005). That study found that 13% of a sample of finishers in that race developed EAH. This compares with the absence of a single recorded case ever of EAH in 42-km marathon runners in South Africa and New Zealand (Noakes & Speedy, 2007).

The authors proved that the same four risk factors previously surmised (Noakes, 1992, 2003b), predicted the risk of EAH during the marathon race:

- Substantial weight gain (odds ratio, 4.2);
- Consumption of more than 3 liters of fluids during the race, consumption of fluids every mile, a racing time of >4 hours (odds ratio, 7.4);
- Female sex;
- Low body mass index.

In 2005, the First International Consensus Conference on EAH concluded that EAH is caused by excessive fluid consumption to which a sodium deficit plays little if any role (Hew-Butler et al., 2005). A subsequent collaborative research paper combining this information (Noakes et al., 2005) concluded that three factors cause EAH:

1. Voluntary overdrinking caused almost certainly by behavioral conditioning in those who have been instructed to drink "as much as tolerable" in order "to stay ahead of thirst" during exercise.
2. A failure to suppress the secretion of the water retaining hormone anti-diaretic hormone (ADH) in the face of an increased total body water content—the Syndrome of Inappropriate ADH secretion (SIADH). As a result the kidney retains fluid

leading to an increase in total body water content, a lowering of the blood sodium concentrations leading ultimately to EAH and EAHE.

3. Relocation of internal sodium stores. There is growing evidence that the sodium present in the body is not located only in the extracellular compartment. Rather, it seems that there may be a substantial amount of sodium stored in an osmotically inactive form (Na) within certain cells. Extracellular sodium may then be added to that store in the process known as inactivation of osmotically active sodium (Na^+). Alternatively, osmotically inactive sodium may be activated, transported outside the cell, and so added to that already present in an osmotically active form in the extracellular fluid.

We also found that approximately 70% of the athletes in our study who had gained weight during exercise (because they both overdrank and failed appropriately to suppress ADH secretion) did not develop EAH (Noakes et al., 2005). Since these athletes could not have ingested enough sodium during exercise to maintain the serum sodium concentration in the face of a large increase in total body water, they must have relocated Na from an osmotically inactive internal body store. Alternatively, abnormal osmotic inactivation of extracellular Na^+ will worsen EAH in those who overdrink during exercise and who fail appropriately to suppress ADH secretion.

The point is that EAH and EAHE will only ever occur in those who are predisposed because (i) they fail appropriately to suppress ADH secretion in response to overdrinking, which causes an expansion of the total body water. (ii) Either they are unable to mobilize Na^+ from internal stores of osmotically inactive Na or (iii) they inactivate osmotically active circulating Na^+ present in the extracellular space, storing it as Na in an intracellular site. The abnormality clearly develops only during exercise—that is, the SIADH component—it is not present at rest.

It therefore follows that the real etiology of EAH can only be studied in persons who exhibit all these abnormalities. Attempting to show that, for example, sodium ingestion can prevent EAH by studying subjects who are not predisposed to its development because they lack these biological variants will not likely uncover the truth.

Finally, studies funded by the sports drink industry and published in 2008 (Baker et al., 2008) and 2009 (Stachenfeld & Taylor, 2009) (Figures 44.3 and 44.4) confirmed our original findings and those of Speedy et al. (1999, 2000b, 2000c). All these findings had established that EAH is due to fluid retention (Figure 44.3) and is unrelated to the extent of sodium losses during exercise (Figure 44.4), and cannot therefore be prevented by the ingestion of sodium during exercise by those who choose to overdrink.

Instead, the simple preventive measure is that athletes should not drink to excess during exercise: they should simply drink to thirst.

Practical Considerations

1. EAH is an iatrogenic disease caused by a false understanding of human physiology. As a result of our evolutionary history, humans are highly

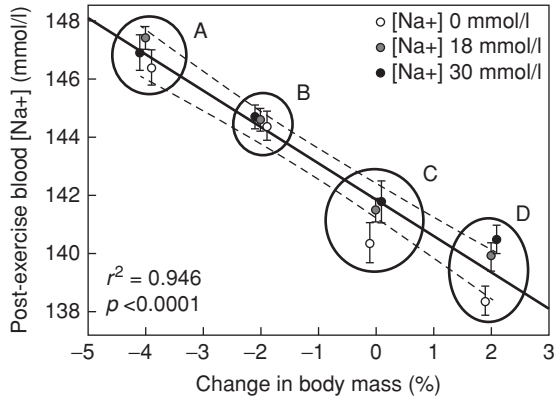

Figure 44.3 The study of Baker et al. (2008) showed that the change in serum sodium concentrations in those who drank solutions with different sodium concentrations (0 vs. 18 vs. 30 mmol/l) at different rates during prolonged exercise was a function of their change in body mass. As a result, 95% of the variance in serum sodium concentrations in that study was predicted by the change in body mass alone. This carefully conducted laboratory study therefore confirmed the finding of our 1991 field study (Irving et al., 1991). Figure 44.2 and 44.3 show an essentially identical finding.

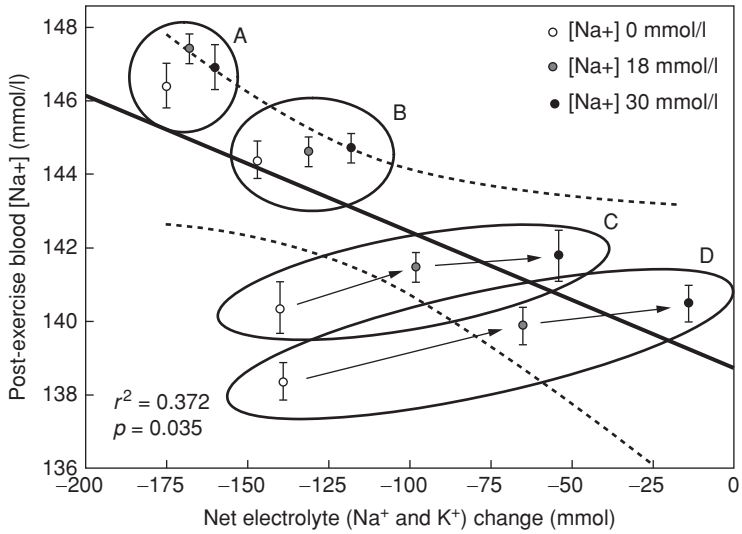

Figure 44.4 The study of Baker et al. (2008) showed that there was a weak inverse relationship between the magnitude of the sodium deficit that developed during exercise and the extent to which the serum sodium concentrations fell in those who drank different volumes of fluid with different sodium concentrations during exercise. As a result serum sodium concentrations fell the most in those with the smallest sodium deficits at the end of exercise. Drinking increasingly more concentrated sodium chloride solutions (30 vs. 18 vs. 0 mmol/l) had only a small and nonsignificant effect (arrows) in reducing the extent to which the serum sodium concentrations fell in those who overdrank during exercise (Groups C and D). There was no effect of sodium ingestion on the serum sodium concentration in those who lost weight during exercise (Groups A and B). Group A drank sufficient fluid to lose 4% of body weight (BW) during exercise, group B drank only enough to lose 2% BW, group C drank to maintain BW and group D drank "ahead of thirst" in order to increase their BW by 2%.

adapted for exercise in the heat and do not need to drink to excess to optimize their performance during exercise. Nor is there any evidence that high rates of fluid ingestion are required to prevent ill-health during exercise. When athletes drank little during exercise, they did not develop EAH and EAHE. Only after the introduction of the drinking guidelines that encouraged athletes to drink "as much as tolerable" in order to prevent a novel medical disease—"dehydration"—and to optimize athletic performance, did the prevalence of EAH and EAHE suddenly increase. This change coincided with mass participation in endurance events, creating both a market for sports drinks and a large number of underprepared participants.

2. EAHE is not a benign condition. At least 12 deaths from the condition have been described in the scientific literature (Noakes, 2003b); additional cases have been reported in the media. All these deaths were foreseeable and preventable.
3. Not everyone who overdrinks during exercise will develop EAH or EAHE. Rather, it appears that only those with genetic variants that cause (i) the oversecretion of ADH even when they are overhydrated, (ii) the relocation of extracellular Na^+ to an intracellular osmotically inactive Na store, or (iii) the inability to activate the reverse process in the face of a large increase in total body water, are at risk of developing EAH and EAHE.
4. Prevention of EAH and EAHE is simple. It requires only that athletes drink according to thirst before, during, and after exercise or according to a plan that does not allow for weight gain during the event. However, whereas experienced athletes will have the opportunity to develop such a plan in the longer training sessions, novices generally do not run far enough in

training to learn how much they should be drinking. Thus novices need to be even more certain to drink only to the dictates of thirst. Race organizers could assist in preventing overdrinking by reducing the number of refreshment stations provided in endurance events like marathons and ultramarathons. There is no proven need to have such stations more than every 5 km in marathon and longer races.

5. Some patients with EAH or EAHE have died because of inappropriate treatment—specifically the provision of hypotonic or isotonic intravenous fluids in large volumes—for the treatment of "dehydration." Yet, these athletes were overhydrated because they had drunk to excess during exercise and had retained that fluid because of SIADH. Fluid restriction in mild cases or the provision of moderate volumes of hypertonic (3–5%) saline solutions and most definitely not the provision of isotonic or hypotonic NaCl solutions in large volumes, is the basis for the safe treatment of EAHE.
6. A fatal outcome can be prevented by a high level of clinical suspicion. It requires clinicians to understand that an altered level of consciousness is *not* caused by the mild levels of "dehydration" experienced by athletes competing in modern athletic events (Noakes et al., 2005) in which fluid is readily available during exercise. Typically, an altered level of consciousness in athletes in whom there is no other obvious cause (such as a cardiac arrest or a cerebrovascular accident), will be due to either heatstroke or EAHE. Heatstroke can be excluded by the measurement of a rectal temperature less than about 41°C in which case EAHE becomes the most likely diagnosis in athletes who do not exhibit other overt clinical signs indicating intracerebral or cardiovascular pathology. EAHE often presents initially as confusion and withdrawal, similar to that seen in persons with mild head injury (concussion).
7. Treatment is simple and life saving (Table 44.2). The key is that no fluids with a NaCl concentration less than 3% (i.e., hypertonic saline) should be used. For severe cases of EAH, 3–5% NaCl solutions given intravenously at slow rates are life saving (Hew-Butler et al., 2005).

Table 44.2 Key factors in the prevention of EAH and fatal outcomes in EAHE

1. Teach athletes to drink according to the dictates of thirst before, during, and after exercise.
2. Physicians treating collapsed athletes with an altered level of consciousness should not infuse any fluids intravenously until a diagnosis of EAHE has been excluded.
3. Once a diagnosis of EAHE has been established, only hypertonic (3% or greater) saline solutions should be used for intravenous therapy.

The adoption of these guidelines has minimized the incidence of EAH and its potential fatal complication, EAHE, in New Zealand and South Africa.

Summary

There is and never was a need for EAH or EAHE to occur. There is no need ever for a fatal outcome. Deaths are always due to ignorance—ignorance on the part of athletes, their coaches, their doctors, race organizers, and powerful commercial interests, all of which have contributed to the encouragement of overdrinking during exercise. The ignorance extends to those emergency ambulance personnel and emergency care physicians who believe that "dehydration" can cause an altered level of consciousness including the development of coma and who, as a result, have treated overhydrated athletes suffering from EAH and EAHE, with the rapid infusion of large volumes of iso- or hypotonic NaCl solutions. The only effect of such treatment is to acutely increase intracerebral pressure, with the production of cerebellar coning, respiratory arrest, and brain death. If athletes drank only to thirst and if all clinicians treating collapsed marathon and ultramarathon runners understood that only profound overhydration and no level of dehydration can cause a loss of consciousness as a result of EAHE, this condition would become the medical curiosity that is all it deserves to be, not the potential killer that it was allowed to become.

References

Almond, C.S., Shin, A.Y., Fortescue, E.B., et al. (2005) Hyponatremia among runners in the Boston Marathon. *New England Journal of Medicine* 352(15), 1550–1556.

American College of Sports Medicine, American Dietetic Association, & Dietitians of Canada (2000) Joint position statement: nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 32(12), 2130–2145.

Armstrong, L.E. (2000) *Performing in Extreme Environments*. Human Kinetics Publishers, Champaign, IL.

Armstrong, L.E., Epstein, Y., Greenleaf, J.E., et al. (1996) American College of Sports Medicine position stand. Heat and cold illnesses during distance running. *Medicine & Science in Sports & Exercise* 28(12), i–x.

Baker, L.B., Lang, J.A., & Kenney, W.L. (2008) Quantitative analysis of serum sodium concentration after prolonged running in the heat. *Journal of Applied Physiology* 105(1), 91–99.

Bramble, D.M. & Lieberman, D.E. (2004) Endurance running and the evolution of Homo. *Nature* 432(7015), 345–352.

Buskirk, E.R. & Beetham, W.P.J. (1960) Dehydration and body temperature as a result of marathon running. *Medicina Sportiva* XIV(9), 493–506.

Carrier, D.R. (1984) The energetic paradox of human running and hominid evolution. *Current Anthropology* 25(4), 483–495.

Casa, D.J., Armstrong, L.E., & Hillman, S.K. (2000) National Athletic Trainers Association position statement: fluid replacement for athletes. *Journal of Athletic Training* 35, 212–224.

Convertino, V.A., Armstrong, L.E., Coyle, E.F., et al. (1996) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 28(10), i–ix.

Dugas, J.P. & Noakes, T.D. (2005) Hyponatraemic encephalopathy despite a modest rate of fluid intake during a 109 km cycle race. *British Journal of Sports Medicine* 39(10), e38.

Galloway, S.D. (1999) Dehydration, rehydration, and exercise in the heat: rehydration strategies for athletic competition. *Canadian Journal of Applied Physiology* 24(2), 188–200.

Gisolfi, C.V. (1996) Fluid balance for optimal performance. *Nutrition Reviews* 54(4), S159–S168.

Heinrich, B. (2010) Endurance predator. In *The Anthropology of Sport and Human Movement* (eds. R.R. Sands & L.R. Sands), pp. 95–101. Lexington Books, Lanham MD.

Hew, T.D., Sharwood, K.A., Speedy, D.B., et al. (2006) *Ad libitum* sodium ingestion does not influence serum sodium concentrations during an Ironman triathlon. *British Journal of Sports Medicine* 40(3), 255–259.

Hew-Butler, T.D., Almond, C.S., Ayus, J.C., et al. (2005) Consensus Document of the 1st International Exercise-Associated Hyponatremia (EAH) Consensus Symposium, Cape Town, South Africa 2005. *Clinical Journal of Sports Medicine* 15(4), 207–213.

Irving, R.A., Noakes, T.D., Buck, R., et al. (1991) Evaluation of renal function and fluid homeostasis during recovery from exercise-induced hyponatremia. *Journal of Applied Physiology* 70(1), 342–348.

Liebenberg, L. (2006) Persistence hunting by modern hunter-gatherers. *Current Anthropology* 47(6), 1017–1025.

Montain, S.J., Sawka, M.N., & Wenger, C.B. (2001) Hyponatremia associated with exercise: risk factors and pathogenesis. *Exercise and Sport Sciences Reviews* 29(3), 113–117.

Montain, S.J., Cheuvront, S.N., & Sawka, M.N. (2006) Exercise associated hyponatraemia: quantitative analysis to understand the aetiology. *British Journal of Sports Medicine* 40(2), 98–105.

Morris, C. (1900) *Man and His Ancestors*. Macmillan and Co., New York.

Muir, A.L., Percy-Robb, I.W., Davidson, I.A., et al. (1970) Physiological aspects of the Edinburgh Commonwealth Games. *Lancet* 2(7683), 1125–1128.

Noakes, T.D. (1992) The hyponatremia of exercise. *International Journal of Sport Nutrition* 2(3), 205–228.

Noakes, T.D. (2003a) *Lore of Running*. Human Kinetics Publishers, Champaign, IL.

Noakes, T.D. (2003b) Overconsumption of fluids by athletes. *British Medical Journal* 327(7407), 113–114.

Noakes, T.D. (2004) Can we trust rehydration research? In *Philosophy and Sciences of Exercise, Sports and Health* (ed. M. McNamee), pp. 144–168. Taylor & Francis Books, Abingdon, Oxfordshire, UK.

Noakes, T.D. (2012) *Waterlogged: The Serious Problem of Overhydration in Endurance Sports*. Human Kinetics, Champaign, IL.

Noakes, T.D. & Speedy, D.B. (2007) Lobbyists for the sports drink industry: an example of the rise of "contrarianism" in modern scientific debate. *British Journal of Sports Medicine* 41(2), 107–109.

Noakes, T.D. & Vlismas, M. (2011) *Challenging Beliefs: Memoirs of a Career*. Zebra Press, Cape Town, South Africa.

Noakes, T.D., Goodwin, N., Rayner, B.L., et al. (1985) Water intoxication: a possible complication during endurance exercise. *Medicine & Science in Sports & Exercise* 17(3), 370–375.

Noakes, T.D., Sharwood, K., Collins, M., et al. (2004) The dipsomania of great distance: water intoxication in an Ironman triathlete. *British Journal of Sports Medicine* 38(4), E16.

Noakes, T.D., Sharwood, K., Speedy, D., et al. (2005) Three independent biological mechanisms cause exercise-associated hyponatremia: evidence from 2,135 weighed competitive athletic performances. *Proceedings of the National Academy of Sciences of USA* 102(51), 18550–18555.

Noakes, T.D., Mekler, J., & Pedoe, D.T. (2008) Jim Peters' collapse in the 1954 Vancouver Empire Games marathon. *South African Medical Journal* 98(8), 596–600.

Nolte, H.W., Noakes, T.D., & Van Vuuren, B. (2011) Trained humans can exercise safely in extreme dry heat when drinking water ad libitum. *Journal of Sports Science* 29(12), 1233–1241.

Pugh, L.G., Corbett, J.L., & Johnson, R.H. (1967) Rectal temperatures, weight losses, and sweat rates in marathon running. *Journal of Applied Physiology* 23(3), 347–352.

Read, C. (1925) *The Origin of Man and of His Superstitions*. University Press, Cambridge.

Rovell, D. (2005) *First in Thirst—How Gatorade Turned the Science of Sweat into a Cultural Phenomenon*. Amacom, New York.

Sawka, M.N. & Montain, S.J. (2000) Fluid and electrolyte supplementation for exercise heat stress. *American Journal of Clinical Nutrition* 72(2 Suppl.), 564S–572S.

Smith, S. (2002) Marathon runner's death linked to excessive fluid intake. Boston Globe (Aug 13) p. A1.

Speedy, D.B., Noakes, T.D., Rogers, I.R., et al. (1999) Hyponatremia in ultradistance triathletes. *Medicine & Science in Sports & Exercise* 31(6), 809–815.

Speedy, D.B., Noakes, T.D., Rogers, I.R., et al. (2000a) A prospective study of exercise-associated hyponatremia in two ultradistance triathletes. *Clinical*

Journal of Sports Medicine 10(2), 136–141.
Speedy, D.B., Rogers, I.R., Noakes, T.D., et al. (2000b) Diagnosis and prevention of hyponatremia at an ultradistance triathlon. *Clinical Journal of Sports Medicine* 10(1), 52–58.
Speedy, D.B., Rogers, I.R., Noakes, T.D., et al. (2000c) Exercise-induced hyponatremia in ultradistance triathletes is caused by inappropriate fluid retention. *Clinical Journal of Sports Medicine* 10(4), 272–278.
Speedy, D.B., Thompson, J.M., Rodgers, I., et al. (2002) Oral salt supplementation during ultradistance exercise. *Clinical Journal of Sports Medicine* 12(5), 279–284.
Stachenfeld, N.S. & Taylor, H.S. (2009) Sex hormone effects on body fluid and sodium regulation in women with and without exercise-associated hyponatremia. *Journal of Applied Physiology* 107(3), 864–872.
Weschler, L.B. (2005) Exercise-associated hyponatraemia: a mathematical review. *Sports Medicine* 35(10), 899–922.
Wyndham, C.H. & Strydom, N.B. (1969) The danger of an inadequate water intake during marathon running. *South African Medical Journal* 43(29), 893–896.

PART 6

SPORT-SPECIFIC NUTRITION: PRACTICAL ISSUES

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Chapter 45

Strength and Power Events

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Introduction

Strength and power athletes compete in a variety of individual and team sports and are difficult to define as a group. The strength and power athlete competes in sporting activities that may take as little as a few seconds to complete, could last longer than several minutes, and/or could be intermittent in nature. Some athletes compete once per day, while others may have multiple races, events, or matches throughout the day. Additionally, strength and power athletes between and within sports can have markedly different body compositions, with vast differences in lean and/or fat mass. For instance, lightweight wrestlers may have very high lean mass and low total body fat, super heavyweight wrestlers may have more lean mass, but also more fat mass, and Sumo wrestlers may have even higher lean and fat mass. The current Mr. Olympia is a 175 cm (5 foot 9 inch) professional bodybuilder, who competes at nearly 110 kg (240 pounds) with less than 6% body fat, while an offensive lineman in professional American (gridiron) football may average 193 cm (6 foot 4 inch) and 140 kg (308 pounds) with 25% body fat (Kraemer et al., 2005). Thus, there is not a single type of strength and power athlete, but a group of diverse individuals. The nutritional needs of strength and power athletes are therefore unique to the goals of the individual and the specific sport.

While the specific nutritional requirements of the strength and power athlete are no doubt sport- and athlete-specific, these athletes do have similar overall objectives when it comes to dietary needs: (1) maintain good health, (2) provide energy for training and competition, (3) support recovery and adaptation from training and competition, and (4) safe and effective use of performance-enhancing substances. This chapter will highlight what is known about current dietary practices of strength and power athletes, offer insight into how these athletes can optimize nutrition for performance, and identify areas of concern.

Energy Intake

As a large number of strength and power athletes are attempting to gain or maintain a higher than normal lean mass, energy intake is not commonly a concern. A recent review of elite strength and power athletes from track and field, weightlifting, and bodybuilding demonstrates wide variability in energy intake from 6100 to 22,400 kJ/day (1457–5350 kcal/day) (reviewed in Slater & Phillips, 2011). Much of this variability appears to stem from the phase during which data were collected (i.e., training vs. competition), which is particularly evident in competitive bodybuilders. When energy intake is normalized for body mass, strength and power athletes appear to be ingesting adequate energy (117–238 kJ/kg/day

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or 28–57 kcal/kg/day), although athletes on the lower end of this range may be ingesting less than what is currently advised and less than what endurance athletes consume. Slater and Philips (2011) have suggested that traditional sport nutrition guidelines may not hold true for large strength and power athletes, who may require energy intake recommendations that are allometrically scaled rather than expressed as a ratio score relative to body mass. As strength and power athletes are a diverse group of individuals, there are those who may not achieve adequate energy intake. These athletes include those who are restricting energy intake in an attempt to lower body fat for a weight class (e.g., wrestling, Olympic weightlifting) or those who compete in sports with an aesthetic or image element (e.g., bodybuilding, sprint swimming, gymnastics).

Macronutrient Intake

Carbohydrate

No matter the sport, strength and power athletes rely heavily on carbohydrate to provide fuel for both training and competition. High intensity/supramaximal exercise lasting from a few seconds to 10 minutes relies heavily on the anaerobic and oxidative metabolism of carbohydrate to support ATP production. Even brief or intermittent high-intensity/supramaximal exercise results in large decreases in muscle glycogen, including during 30 seconds of maximal running (↓ 25%) (Cheetham et al., 1986), resistance exercise (↓ 26%) (Tesch et al., 1986), and 6 seconds of maximal cycling (↓ 14%) (Gaitanos et al., 1993). Some strength and power athletes with a basic understanding of exercise physiology may believe that the creatine/phosphorylcreatine energy system is responsible for the majority of ATP production during their training and competition, and be unconcerned with carbohydrate intake, but this is erroneous. It is known that very low carbohydrate intake impairs the performance of intense exercise. Additionally, many strength and power athletes follow a protein-centered diet (>20% of energy intake), which can displace dietary carbohydrate, potentially reducing performance and impairing recovery.

Reportedly, daily carbohydrate intake in strength and power athletes is highly variable and ranges from 3 to 7 g/kg/day (reviewed in Slater & Phillips, 2011). As with energy intake, part of this variability can be explained by training phase (e.g., off-season training, competition), but there appear to be sport-specific differences as well. Weightlifters and throwers appear to be on the lower end of the range (3–5 g/kg/day), while bodybuilders report higher carbohydrate intakes (4–7 g/kg/day), which are often in combination with a low-fat diet. Strength and power athletes generally report carbohydrate intakes lower than those of endurance athletes (7–10 g/kg/day). It has not been shown that the "lower" carbohydrate intake of strength and power athletes, relative to endurance athletes, is harmful to performance. Also, data that demonstrate a beneficial effect of a chronically high carbohydrate diet (7–10 g/kg/day) in strength and power athletes are not available.

The benefits of carbohydrate consumption before and during exercise on endurance exercise performance are well known, but less is known about acute carbohydrate ingestion and strength and power outcomes. Some investigators have shown increased work capacity following carbohydrate ingestion before or during resistance exercise (1 g/kg before; 0.5 g/kg during) (Haff et al., 2001), while others have found no effect (Kulik et al., 2008). As with endurance exercise, the benefits of carbohydrate consumption before and during exercise on strength and power performance are likely influenced by exercise duration, intensity, and volume of training; number of races/matches per day; and recovery time. More research needs to be conducted in this area to develop appropriate recommendations for strength and power athletes. Given the reliance on carbohydrate for fuel, postexercise glycogen synthesis to support performance in subsequent events, should be a priority for strength and power athletes. It is recommended that athletes consume about 1.2 g/kg of carbohydrate immediately after exercise. While this will optimize glycogen resynthesis, the addition of protein in the post-exercise recovery period (discussed in Protein, and see also Chapter 7) is also recommended.

Protein

A well-known and long-standing characteristic of the diet of strength and power athletes is a high protein intake. Athletes do have elevated protein needs, and the recommended intake is generally about 1.6–1.7 g/kg/day, or about twice what is recommended for sedentary individuals. Reportedly, strength and power athletes commonly achieve recommended levels to support muscle recovery and adaptation (reviewed in Slater & Phillips, 2011), with many ingesting far greater amounts (up to 3.2 g/kg) (Chen et al., 1989). Although disputed by athletes, it appears that excess dietary protein intake above 1.7 g/kg/day results in increased amino acid catabolism and protein oxidation without additional hypertrophic benefits (Moore et al., 2009).

While the quantity of daily protein intake does not appear to be an issue with most strength and power athletes, the dose, type, and timing of protein ingested may have important implications for recovery and adaptation. It is suggested that, following resistance exercise, 20 g of intact protein maximally stimulates muscle protein synthesis, while amounts in excess of this amount increase protein oxidation (Moore et al., 2009). Thus, strength and power athletes may consider the ingestion of no more than 20 g of protein per meal or snack, spread out over several meals throughout the day. However, the absolute amount of protein to ingest after exercise is difficult to establish, as larger athletes may require a larger amount of protein because of their greater muscle mass. When considering the type of protein to ingest, proteins of a high quality (8–10 g of essential amino acids per 20 g) are best, but rate of digestion matters as well. Data from Phillips and others show that postexercise milk protein consumption results in greater hypertrophy in response to chronic resistance training (Hartman et al., 2007) and greater net protein balance and synthesis rate following resistance exercise when compared with soy (Wilkinson et al., 2007). Additionally, this same group has shown that rapidly digested proteins (e.g., whey), which result in a rapid aminoacidemia, ingested at rest and after exercise, cause a greater increase in protein synthesis than slow-digesting proteins (e.g., casein, soy) (Tang et al., 2009). One practical issue that stems from the use of supplemental protein is that many athletes consume "meal replacement powders" instead of protein powders. While protein powders are essentially protein and flavoring, meal replacement supplements are a high-quality protein supplement with carbohydrate, fat, and high doses of vitamins and minerals. Two to three servings of some meal replacement powders per day will provide an athlete with micronutrient levels above the recommended upper tolerable limit.

The ingestion of about 20 g of rapidly digested protein after exercise in combination with postexercise carbohydrate consumption serves to maximize both postexercise protein and glycogen synthesis. As strength and power athletes may have greater absolute protein needs, and are often trying to maintain or achieve higher than normal lean mass levels, replacing some carbohydrate with protein in the immediate postexercise recovery period is valuable, especially as this appears not to reduce glycogen resynthesis. Relative to body mass, the commonly recommended levels to maximize glycogen and protein synthesis after exercise are 0.8 g/kg of carbohydrate combined with 0.4 g/kg of protein, though this will perhaps give more protein than is necessary.

Fat

Many strength and power athletes consume levels of total fat that exceed recommendations for good health (up to 47% of energy) (Chen et al., 1989). While it cannot be stated that this is harmful to performance or unhealthy during an athlete's competitive career, there are some concerns. First, the consumption of high fat/high protein foods likely displaces carbohydrate from the diet. This matters more for some strength and power athletes, especially those who compete in multiple races/matches per day and those that compete in longer duration events. Second, poor diet, along with increases in body mass index/body fat increases risk of morbidity and mortality. Strength and power athletes have recently come under scrutiny in terms of their health status and disease risk. When compared with a reference population, professional American football (gridiron) players had a lower occurrence of impaired fasting glucose, similar dyslipidemia, but a higher prevalence of hypertension

(Tucker et al., 2009). It could be hypothesized that the similar blood lipid profile between groups, despite the athletes being significantly larger (BMI 31 vs. 26 kg/m^2; waist circumference 97 vs. 86 cm; waist to hip ratio 0.88 vs. 0.85; all $p < 0.001$), is a result of the higher levels of physical activity. If so, when an athlete's sporting career ends, and physical activity decreases significantly, disease risk may increase. Also, in this cohort, blood pressure was significantly greater in the athletes regardless of body size (127/75 vs. 112/72 mmHg; $p < 0.001$) (Tucker et al., 2009). In a study of elite collegiate American (gridiron) football players, the prevalence of obesity (body fat ≥25%), insulin resistance, and metabolic syndrome was 21%, 21%, and 9%, respectively. Linemen, the largest athletes in the study, accounted for all of the obese subjects, 68% of the insulin resistant subjects, and 100% of the subjects with metabolic syndrome (Borchers et al., 2009). As it is unlikely that these athletes will do what is necessary to improve their health during their competitive career, especially if they perceive a loss of body size or a change in dietary behaviors as harmful to performance, educational programs must be developed to help these strength and power athletes as they transition out of sport.

Water

Endurance performance decrements induced by hypohydration are well documented, but far less research is available regarding strength and power performance. Additionally, some strength and power athletes may consider that their sporting events do not last long enough to induce or suffer ill effects from hypohydration. However, it appears that resistance exercise performance (Kraft et al., 2010) and mean and total peak cycling power (Jones et al., 2008) are reduced with modest hypohydration (3% of body mass). Thus, strength and power athletes should be encouraged to begin training and competition properly hydrated to prevent performance decrements (see Chapter 15).

Micronutrients

Due to a large energy/food intake, many strength and power athletes achieve or exceed recommended levels of micronutrient intake. However, there are a few concerns. On average, strength and power athletes achieve adequate micronutrient intake, but a number of athletes appear to ingest inadequate amounts of several micronutrients, commonly iron, calcium, magnesium, vitamin B_6, and vitamin C. This may result from diets that are too restrictive or lack variety, which can be characteristic of precontest competitive bodybuilders and other athletes trying to maintain low body fat. Although not specific to strength and power athletes, a longitudinal study of vitamin D status in collegiate athletes demonstrated that 64% were vitamin D deficient or insufficient in winter, 12% in the fall, and 20% in the spring (Halliday et al., 2011). Preliminary findings on American (gridiron) football players demonstrate that 30% are deficient and 50% insufficient in vitamin D, with African American players having poorer vitamin D status than Caucasians (Shindle et al., 2011). At this time, there is considerable controversy regarding the recommended cut points used to define vitamin D insufficiency and sufficiency, but it appears that poor vitamin D status is more common in athletes than previously believed (see Chapter 20). One final issue is the previously described displacement of carbohydrate-rich, nutrient-dense foods by high-protein foods. This may reduce intake of dietary fiber, and although a low-fiber diet may not be harmful to performance *per se*, low fiber intake is associated with increased morbidity.

Performance-Enhancing Substances

Optimum training programs and proper nutrition are essential for gaining an advantage over the competition. However, an athlete's performance is limited by their genetic potential, and with the tremendous pressure to succeed in high-level sports, athletes often resort to the use of performance-enhancing substances to gain an edge over the competition. Strength and power athletes have used performance-enhancing substances for centuries. Although performance-enhancing drugs are generally banned by major sport organizations, some performance-enhancing drugs, like caffeine, can be consumed. Also, some dietary supplements may be effective in improving strength and power

performance. Historically, athletes have used a variety of naturally occurring and synthetic performance-enhancing substances, often sacrificing safety for improved performance. Thus, athletes must be educated as to what constitutes a banned or legal substance, and in the case of legal substances, how to use them safely and effectively.

Banned Substances

The most prevalent drugs used by strength and power athletes are those that are intended to increase muscle mass and strength by increasing muscle protein synthesis and/or decreasing protein degradation, such as anabolic androgenic steroids. A recent World Anti-Doping Agency (WADA) report shows that 61% of all samples that tested positive for a banned substance in WADA-accredited laboratories were for anabolic androgenic compounds (WADA, 2010). Banned or not, the available data support that anabolic androgenic hormones increase fat-free mass and strength, with or without concurrent training (Bhasin et al., 1996). The effects of androgen administration are dose-dependent, with higher doses producing the most pronounced effects on strength and fat-free mass accrual. This may encourage abuse and increase risk of adverse effects, which can be severe.

Some evidence suggests that human growth hormone (GH) administration can increase lean body mass, but it appears to have no effect on strength or physical performance (reviewed in Liu et al., 2008). This suggests that the increased fat-free mass associated with GH administration is not due to increases in skeletal muscle contractile protein content, but is more likely due to fluid retention and/or increases in noncontractile proteins. Long-term abuse of GH can result in symptoms similar to patients with acromegaly, such as cardiomyopathy, hypertension, insulin resistance, and a higher risk for cancer development. Additionally, short-term side effects such as edema and fatigue have been observed in several studies. Despite poor evidence of efficacy and many dangers, it appears that athletes continue to use GH and dietary supplements purported to stimulate GH endogenous secretion, possibly because they believe they are undetectable.

At this time, there have been no scientific studies examining the efficacy of insulin administration for increasing strength or muscle mass. Reportedly, athletes inject insulin following a post-workout meal to increase delivery of carbohydrate and amino acids to the muscle. In theory, insulin use could be beneficial after training to replenish muscle glycogen stores and decrease muscle catabolism. Insulin, however, is a powerful metabolic hormone that can cause serious side effects if used inappropriately. The use of insulin without concurrent food intake or the use of too much insulin can cause rapid hypoglycemia, unconsciousness, or death in healthy individuals. In addition, long-term insulin abuse by athletes can result in a decrease endogenous insulin production or insulin resistance.

Synthetic β_2 agonists such as Clenbuterol are abused by strength and power athletes due to their ability to stimulate skeletal muscle β_2 adrenergic receptors. Animal data indicate that β_2 agonists increase muscle mass by both increasing protein synthesis and decreasing protein degradation via signaling through the β_2 adrenergic receptor. Several animal studies have also shown that the administration of β_2 agonists induces skeletal muscle fibers to shift from a slow- to fast-twitch phenotype (reviewed in Ryall & Lynch, 2008), which is a beneficial adaptation for strength and power athletes. Further, a recent meta-analysis reported that β_2 agonists may also be effective for increasing anaerobic power performance (Pluim et al., 2011). In addition to being banned substances, side effects of β_2 agonist ingestion can include minor symptoms associated with increased adrenergic stimulation such as headache, tachycardia, and tremor, and also major effects on cardiovascular function such as myocardial hypertrophy.

Testosterone precursors such as the steroid hormones androstenedione and androstenediol first became popular as a means to "naturally" increase circulating testosterone levels, without actually taking a banned anabolic androgenic steroid drug. These "prohormone" supplements are banned by WADA due to their potential to manipulate testosterone production, and are also illegal to sell in many countries. It appears that prohormone supplementation is ineffective in augmenting strength

or muscle mass with concurrent resistance training, and may have undesirable effects such as increased estrogen levels and decreased HDL cholesterol.

Banned central nervous system stimulants such as cocaine, amphetamines, and ephedrine have been used by strength and power athletes to increase energy, alertness, and focus prior to competition or training. In 2010, WADA-accredited laboratories found stimulants to be the second most common banned substance used by athletes (WADA, 2010). Additionally, drugs such as ephedrine have been used to reduce body mass/body fat by athletes who are attempting to lose weight, with modest success (reviewed in Shekelle et al., 2003). When combined with caffeine, ephedrine appears to enhance maximal and resistance exercise performance (reviewed in Shekelle et al., 2003). Stimulants have many cardiotoxic effects, including hypertension, irregular heartbeat, and fatal arrhythmia as well as negative psychiatric and thermoregulatory effects (Rawson & Clarkson, 2002). Psychological effects of stimulants as well as substances that exert indirect effects on the central nervous system (e.g., androgens, prohormones) include increased aggression as well as depression during withdrawal of use. The extent of aggressive behavior and withdrawal symptoms are likely dose dependent and also related to the psychological predisposition of the user. While most central nervous system stimulants are illegal (e.g., cocaine), require a prescription (e.g., amphetamines), and/or are banned substances (e.g., ephedrine), some stimulants are readily available and are permitted at certain quantities (e.g., caffeine).

Permitted Substances

Caffeine is a widely consumed organic alkaloid that is ingested by about 75% of athletes prior to competition or training to reduce fatigue and increase alertness. Although better known as a performance enhancer during endurance exercise, caffeine may improve strength, power, and resistance exercise performance particularly in trained athletes who do not regularly ingest caffeine (see also Chapter 25), but this is controversial. One group reported increased maximum strength and repetitions to failure at given percentage of maximum with caffeine ingestion (Duncan & Oxford, 2010), while another reported no effect (Astorino et al., 2008). Caffeine may improve strength and power performance through sympathetic nervous system stimulation, enhanced ion movement, improved excitation–contraction coupling, or increased motor unit recruitment. More research is needed to better understand the mechanism through which caffeine can exert an ergogenic effect on strength and power tasks. An effective dose as low as ≈1 mg/kg has been noted (Bellar et al., 2011), which is unlikely to have significant side effects. Higher doses of caffeine (≈10 mg/kg) may cause headache, tremor, tachycardia, anxiety, nervousness, insomnia, increased urination, and gastrointestinal discomfort, which could lead to performance decrements. Caffeine dependency and tolerance may blunt any ergogenic effect, so habitual caffeine users may consider slowly decreasing caffeine intake to zero over the course of the week prior to competition. This should reduce the side effects and any adverse performance consequences which can occur with abrupt cessation of caffeine use (Sökmen et al., 2008). Returning to normal levels of caffeine intake on the day of the athletic event will again provide ergogenic benefits (Sökmen et al., 2008). Since caffeine is a drug, there has been debate over the ethical issues regarding its use in sport. Today, caffeine is not a banned substance, but it remains on the World Anti-Doping Agency's (WADA) monitored substance list.

Ammonia inhalants have become increasingly popular among athletes, particularly powerlifters, and are used to acutely improve alertness, focus, strength, or power performance. These inhalants consist of ammonium carbonate enclosed in a capsule, which is opened and inhaled through the nose just prior to a competition or training. The ammonia acts as a respiratory irritant and triggers an inhalation reflex, which supposedly improves respiratory flow rates and improves alertness. There have been no scientific studies examining the effects of ammonia inhalants on strength or power, and large doses of ammonia inhalants can cause lung damage or anaphylaxis. Because they are a respiratory irritant, athletes with asthma should not use ammonia inhalants.

Dietary Supplements

For a nonstimulant dietary supplement to be relevant for a strength and power athlete, it must provide energy for or enhance recovery and adaptation from training or competition. A small number of dietary supplements have a body of research to support efficacy and are also relevant to strength and power athletes, including creatine monohydrate, β-alanine, and sodium bicarbonate.

Creatine Monohydrate Perhaps the most heavily researched dietary supplement, creatine monohydrate is widely used by strength and power athletes, with some survey research indicating prevalence of use up to 74% (see also Chapter 24). Although more recent data indicate lower rates of use (12%) (Dascombe et al., 2010), it is difficult to know if fewer strength and power athletes are using creatine supplements today, or if this discrepancy is a result of the difficulty involved in obtaining accurate reports on supplement use.

As described in Chapter 24, creatine monohydrate improves the performance of brief, intense exercise, particularly when there are repeated bouts (reviewed in Gualano et al., 2012; Rawson & Volek, 2003). Creatine acts through multiple mechanisms, including increased pre-exercise fuel availability (e.g., glycogen, phosphorylcreatine), faster phosphorylcreatine resynthesis during recovery from intense exercise, reduced muscle damage and inflammation, increased growth factor expression, increased satellite cell activity, and spontaneously increased training volume (reviewed in Rawson & Persky, 2007). Thus, creatine monohydrate supports ATP production during intense exercise and promotes skeletal muscle recovery and adaptations following training and competition. Even if creatine monohydrate does not enhance performance of the sport-specific performance of a strength and power athlete *per se*, the beneficial effects of creatine supplementation on chronic resistance and/or sport-specific training may allow athletes to make faster gains, which subsequently could improve performance during competition. In fact, creatine monohydrate appears to be most useful as an adjunct to progressive resistance exercise (reviewed in Rawson & Volek, 2003). Rawson and Volek (2003) estimated that creatine combined with resistance training increased maximum strength 8% and repetition endurance at a given percentage of maximum strength 14% more than resistance training alone.

On average, skeletal muscle creatine increases about 20% following either a brief, high-dose creatine monohydrate supplementation protocol (about 20 g/day for 5 days) or a longer-term, lower-dose protocol (3 g/day for about 30 days) (Harris et al., 1992; Hultman et al., 1996), with improvements in high-intensity exercise performance resulting from both the high-dose (numerous studies) and the lower-dose (Rawson et al., 2011) supplementation protocols. As described in Chapter 24, exercise and the co-ingestion of carbohydrate and protein can increase muscle creatine uptake (Harris et al., 1992; Pittas et al., 2010), so athletes should consider ingesting creatine monohydrate in the immediate post-workout period along with their post-workout carbohydrate–protein meal.

With the exception of increased body mass, there are no side effects associated with creatine monohydrate supplementation, and this has been extensively reviewed (Gualano et al., 2012; Persky & Rawson, 2007). However, the increased body mass may create difficulties for athletes who compete in sports with weight classes, as the weight gain associated with creatine monohydrate supplementation appears to track the lengthy muscle creatine washout period that occurs following cessation of the supplement (Hultman et al., 1996). Rawson et al. (2004) reported on an individual who had increased muscle creatine (+23%) and body mass (+2 kg) 30 days after a brief, high-dose creatine supplementation program (20 g/day for 5 days) was discontinued. It could be theorized that weight gain itself could be harmful to performance (e.g., when running or jumping) or alter biomechanics, although no data are available to prove that creatine supplementation is detrimental in any strength–power event. Alternative forms of creatine are heavily marketed toward strength and power athletes, but there are almost no data regarding safety or efficacy, the cost is substantially greater than for creatine monohydrate, and the product purity, largely unknown. The strength and power athlete who chooses to use creatine supplements should (1) not exceed

recommended doses, (2) be aware that creatine supplements may increase the difficulty of "making weight," and (3) use creatine monohydrate in favor of novel creatine supplements.

β-Alanine This nonessential amino acid combines with histidine to synthesize the dipeptide carnosine, which is abundant in skeletal muscle and acts as an intramuscular buffer to slow the drop in pH during intense exercise (see Chapter 26). Carnosine, supplies about 7% of skeletal muscle buffering capacity, but this can be doubled with β-alanine supplementation. There is increasing evidence that β-alanine supplementation of 3–6 g/day for about 4–8 weeks increases muscle carnosine by about 40–50% (Harris et al., 2006; reviewed in Sale et al., 2010). Subsequently, this increases the performance of high-intensity exercise lasting from 1 to 6 minutes (Sale et al., 2010). The only known side effect to β-alanine supplements is paresthesia, which appears to have been resolved with timed-release supplements. As with creatine monohydrate, if β-alanine supplementation does not improve sport performance for a given strength and power athlete, it may increase performance during resistance exercise, which may subsequently support sport-specific performance. Long-term side effects of β-alanine supplementation are unknown because of the brief period of time it has been studied, but β-alanine is an amino acid (albeit a β-amino acid rather that the α-amino acids that make up body proteins) and supplementation doses are not unlike normal dietary intake.

Sodium Bicarbonate ($NaHCO_3$) During high intensity exercise, H^+ accumulation can reduce force production and is a factor in fatigue, but supplementing with sodium bicarbonate increases the body's buffering capacity and should slow acidosis-related fatigue (see Chapter 26). Sodium bicarbonate is a strong buffer that creates an alkaline shift in acid–base balance prior to exercise and increases bicarbonate (HCO^-_3) concentrations, an important component of the extracellular buffering system. Not all studies of sodium bicarbonate supplementation show improved performance of intense exercise. For instance, sodium bicarbonate ingestion did not improve repeated Wingate cycling or countermovement jump performance in elite BMX cyclists (Zabala et al., 2011), but it increased intermittent sprint performance in team sport athletes (Bishop & Claudius, 2005). Overall, an athlete may experience about a 1.7% improvement in the performance of intense exercise, provided that the exercise task was intense enough and of sufficient duration to create acidosis and that performance was not decreased by side effects from the supplement (Carr et al., 2011a).

Acute doses of sodium bicarbonate from 0.3 to 0.5 g/kg (300–500 mg/kg) should be ingested 60–180 minutes before exercise, but supplementation for 6 days prior to competition may be a better strategy for strength and power athletes (McNaughton & Thompson, 2001). Many athletes (50%) experience gastrointestinal distress with sodium bicarbonate supplementation, but this may be alleviated by the co-ingestion of a carbohydrate and using encapsulated sodium bicarbonate (Carr et al., 2011b). Due to the high probability of experiencing gastrointestinal distress, athletes considering the use of sodium bicarbonate should experiment during training, not competition, to understand their own individual response to the supplement. The available data do not strongly support benefit of sodium bicarbonate supplementation on high-intensity exercise performance lasting less than 1 minute. Strength and power athletes should consider the risk to benefit ratio (gastrointestinal distress vs. improved performance) and whether the exercise task they are attempting to improve with sodium bicarbonate ingestion is of maximum intensity and the proper duration (1–7 minutes).

Positive Drug Tests and Product Purity

Several cases have been reported in which supplements have been found to contain stimulants, prohormones, and androgens that are prohibited in competitive sporting events (reviewed in Geyer et al., 2008, see Chapter 23). For example, a study performed at a WADA-accredited laboratory found that 14.8% of 578 nutritional supplements from around the world tested positive for prohormones that were not declared on the label (Geyer et al., 2004). Athletes, especially those competing at elite levels, should purchase supplements from reputable sources to prevent inadvertent positive drug tests caused by supplement contamination, but this does not

guarantee product purity. A related practical concern for athletes who do not use performance-enhancing substances is the issue of contaminated food. Liver in particular appears to harbor metabolites of drugs used to modify the body composition of the animal, and has resulted in cases of poisoning (Barbosa et al., 2005) and allegedly, positive drug tests. According to the WADA World Anti-Doping Code, "it is each athlete's personal duty to ensure that no prohibited substance enters his or her body. Athletes are responsible for any prohibited substance or its metabolites or markers found to be present in their samples."

Conclusion

On average, strength and power athletes ingest adequate energy, carbohydrate, and protein, although some may overconsume protein at the expense of valuable carbohydrate. The timing, digestion rate, and quality of postexercise protein ingestion appear to be important factors in protein balance. A small number of the largest strength and power athletes may be overeating dietary fat, which may have negative health consequences, although there are no known harmful effects on performance. Strength and power athletes who are attempting to lose body fat for aesthetic reasons, or to make weight, may not ingest enough energy and also may be deficient in several essential micronutrients. Also, vitamin D insufficiency, although not unique to strength and power athletes, is a growing concern. Strength and power athletes may ingest banned substances purposefully with drugs or accidentally through food or contaminated dietary supplements. In either case, if an athlete tests positive for a banned substance, it is viewed as their responsibility. A small number of dietary supplements may improve performance of strength–power exercise tasks, but supporting evidence of ergogenic effects in laboratory tests far outweighs the evidence for ergogenesis during sport-specific performance or competition. Nonetheless, some dietary supplements may be valuable to aid resistance training performance, if not sport-specific performance *per se*. Strength and power athletes, although a diverse and difficult to define group, require energy to compete and train, and nutritional support to recover from and adapt to training and competition. These common goals can be achieved with whole foods, proper postexercise nutrition, and sensible use of approved, performance-enhancing substances.

References

Astorino, T.A., Rohmann, R.L., & Firth, K. (2008) Effect of caffeine ingestion on one-repetition maximum muscular strength. *European Journal of Applied Physiology* 102(2), 127–132.

Barbosa, J., Cruz, C., Martins, J., et al. (2005) Food poisoning by clenbuterol in Portugal. *Food Additives and Contamination* 22(6), 563–566.

Bellar, D.M., Kamimori, G., Judge, L., et al. (2011) Effects of low-dose caffeine supplementation on early morning performance in the standing shot put throw. *European Journal of Sport Science* 1(12), 57–61.

Bhasin, S., Storer, T.W., Berman, N., et al. (1996) The effects of supraphysiologic doses of testosterone on muscle size and strength in normal men. *New England Journal of Medicine* 335(1), 1–7.

Bishop, D. & Claudius, B. (2005) Effects of induced metabolic alkalosis on prolonged intermittent-sprint performance. *Medicine & Science in Sports & Exercise* 37(5), 759–767.

Borchers, J.R., Clem, K.L., Habash, D.L., et al. (2009) Metabolic syndrome and insulin resistance in Division 1 collegiate football players. *Medicine & Science in Sports & Exercise* 41(12), 2105–2110.

Carr, A.J., Hopkins, W.G., & Gore, C.J. (2011a) Effects of acute alkalosis and acidosis on performance: a meta-analysis. *Sports Medicine* 41(10), 801–814.

Carr, A.J., Slater, G.J., Gore, C.J., et al. (2011b) Effect of sodium bicarbonate on [HCO3-], pH, and gastrointestinal symptoms. *International Journal of Sport Nutrition and Exercise Metabolism* 21(3), 189–194.

Cheetham, M.E., Boobis, L.H., Brooks, S., et al. (1986) Human muscle metabolism during sprint running. *Journal of Applied Physiology* 61(1), 54–60.

Chen, J.D., Wang, J.F., Li, K.J., et al. (1989) Nutritional problems and measures in elite and amateur athletes. *American Journal of Clinical Nutrition* 49(5 Suppl.), 1084–1089.

Dascombe, B.J., Karunaratna, M., Cartoon, J., et al. (2010) Nutritional supplementation habits and perceptions of elite athletes within a state-based sporting institute. *Journal of Science and Medicine in Sport* 13(2), 274–280.

Duncan, M.J. & Oxford, S.W. (2010) The effect of caffeine ingestion on mood state and bench press performance to failure. *Journal of Strength and Conditioning Research* 25(1), 178–185.

Gaitanos, G.C., Williams, C., Boobis, L.H., et al. (1993) Human muscle metabolism during intermittent maximal exercise. *Journal of Applied Physiology* 75(2), 712–719.

Geyer, H., Parr, M.K., Mareck, U., et al. (2004) Analysis of non-hormonal nutritional supplements for anabolic-androgenic steroids—results of an international study. *International Journal of Sports Medicine* 25(2), 124–129.

Geyer, H., Parr, M.K., Koehler, K., et al. (2008) Nutritional supplements cross-contaminated and faked with doping

substances. *Journal of Mass Spectrometry* 43(7), 892–902.

Gualano, B., Roschel, H., Lancha-Jr, A.H., et al. (2012) In sickness and in health: the widespread application of creatine supplementation. *Amino Acids* 43, 519–529.

Haff, G.G., Schroeder, C.A., Koch, A.J., et al. (2001) The effects of supplemental carbohydrate ingestion on intermittent isokinetic leg exercise. *Journal of Sports Medicine and Physical Fitness* 41(2), 216–222.

Halliday, T.M., Peterson, N.J., Thomas, J.J., et al. (2011) Vitamin D status relative to diet, lifestyle, injury, and illness in college athletes. *Medicine & Science in Sports & Exercise* 43(2), 335–343.

Harris, R.C., Söderlund, K., & Hultman, E. (1992) Elevation of creatine in resting and exercised muscle of normal subjects by creatine supplementation. *Clinical Science* 83(3), 367–374.

Harris, R.C., Tallon, M.J., Dunnett, M., et al. (2006) The absorption of orally supplied beta-alanine and its effect on muscle carnosine synthesis in human vastus lateralis. *Amino Acids* 30(3), 279–289.

Hartman, J.W., Tang, J.E., Wilkinson, S.B., et al. (2007) Consumption of fat-free fluid milk after resistance exercise promotes greater lean mass accretion than does consumption of soy or carbohydrate in young, novice, male weightlifters. *American Journal of Clinical Nutrition* 86(2), 373–381.

Hultman, E., Söderlund, K., Timmons, J.A., et al. (1996) Muscle creatine loading in men. *Journal of Applied Physiology* 81(1), 232–237.

Jones, L.C., Cleary, M.A., Lopez, R.M., et al. (2008) Active dehydration impairs upper and lower body anaerobic muscular power. *Journal of Strength and Conditioning Research* 22(2), 455–463.

Kraemer, W.J., Torine, J.C., Silvestre, R., et al. (2005) Body size and composition of National Football League players. *Journal of Strength and Conditioning Research* 19(3), 485–489.

Kraft, J.A., Green, J.M., Bishop, P.A., et al. (2010) Impact of dehydration on a full body resistance exercise protocol. *European Journal of Applied Physiology* 109(2), 259–267.

Kulik, J.R., Touchberry, C.D., Kawamori, N., et al. (2008) Supplemental carbohydrate ingestion does not improve performance of high-intensity resistance exercise. *Journal of Strength and Conditioning Research* 22(4), 1101–1107.

Liu, H., Bravata, D.M., Olkin, I., et al. (2008) Systematic review: the effects of growth hormone on athletic performance. *Annals of Internal Medicine* 148(10), 747–758.

McNaughton, L. & Thompson, D. (2001) Acute versus chronic sodium bicarbonate ingestion and anaerobic work and power output. *Journal of Sports Medicine and Physical Fitness* 41(4), 456–462.

Moore, D.R., Robinson, M.J., Fry, J.L., et al. (2009) Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. *American Journal of Clinical Nutrition* 89(1), 161–168.

Persky, A.M. & Rawson, E.S. (2007) Safety of creatine supplementation. *Sub-cellular Biochemistry* 46, 275–289.

Pittas, G., Hazell, M.D., Simpson, E.J., et al. (2010) Optimization of insulin-mediated creatine retention during creatine feeding in humans. *Journal of Sports Science* 28(1), 67–74.

Pluim, B.M., de Hon, O., Staal, J.B., et al. (2011) Beta-agonists and physical performance: a systematic review and meta-analysis of randomized controlled trials. *Sports Medicine* 41(1), 39–57.

Rawson, E. S. & Clarkson, P. M. (2002). Ephedrine as an ergogenic aid. In *Performance-Enhancing Substances in Sport and Exercise* (eds. M.S. Bahrke & C.E. Yesalis), pp. 289–298. Human Kinetics, Champaign, IL.

Rawson, E.S. & Persky, A.M. (2007) Mechanisms of muscular adaptations to creatine supplementation. *International SportMed Journal* 8(2), 43–53.

Rawson, E.S. & Volek, J.S. (2003) Effects of creatine supplementation and resistance training on muscle strength and weightlifting performance. *Journal of Strength and Conditioning Research* 17(4), 822–831.

Rawson, E.S., Persky, A.M., Price, T.B., et al. (2004) Effects of repeated creatine supplementation on muscle, plasma, and urine creatine levels. *Journal of Strength and Conditioning Research* 18(1), 162–167.

Rawson, E.S., Stec, M.J., Frederickson, S.J., et al. (2011) Low-dose creatine supplementation enhances fatigue resistance in the absence of weight gain. *Nutrition* 27(4), 451–455.

Ryall, J.G. & Lynch, G.S. (2008) The potential and the pitfalls of beta-adrenoceptor agonists for the management of skeletal muscle wasting. *Pharmacology and Therapeutics* 120(3), 219–232.

Sale, C., Saunders, B., & Harris, R.C. (2010) Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids* 39(2), 321–333.

Shekelle, P.G., Hardy, M.L., Morton, S.C., et al. (2003) Efficacy and safety of ephedra and ephedrine for weight loss and athletic performance: a meta-analysis. *Journal of the American Medical Association* 289(12), 1537–1545.

Shindle, M.K., Voos, J.E., Gulotta, L., et al. (2011) Vitamin D status in a professional American football team. *Proceedings from the AOSSM Annual Meeting*, ID 46-9849.

Slater, G. & Phillips, S.M. (2011) Nutrition guidelines for strength sports: sprinting, weightlifting, throwing events, and bodybuilding. *Journal of Sports Science* 29(Suppl. 1), S67–S77.

Sökmen, B., Armstrong, L.E., Kraemer, W.J., et al. (2008) Caffeine use in sports: considerations for the athlete. *Journal of Strength and Conditioning Research* 22(3), 978–986.

Tang, J.E., Moore, D.R., Kujbida, G.W., et al. (2009) Ingestion of whey hydrolysate, casein, or soy protein isolate: effects on mixed muscle protein synthesis at rest and following resistance exercise in young men. *Journal of Applied Physiology* 107(3), 987–992.

Tesch, P.A., Colliander, E.B., & Kaiser, P. (1986) Muscle metabolism during intense, heavy-resistance exercise. *European Journal of Applied Physiology and Occupational Physiology* 55(4), 362–366.

Tucker, A.M., Vogel, R.A., Lincoln, A.E., et al. (2009) Prevalence of cardiovascular disease risk factors among National Football League players. *Journal of the American Medical Association* 301(20), 2111–2119.

World Anti-Doping Agency [WADA] (2010) *2010 Adverse analytical findings and atypical findings reported by accredited laboratories*. Available at: http://www.wada-ama.org/Documents/Resources/Statistics/Laboratory_Statistics/WADA_WADA_2010_Laboratory_Statistics_Report.pdf (accessed May 15, 2013).

Wilkinson, S.B., Tarnopolsky, M.A., Macdonald, M.J., et al. (2007) Consumption of fluid skim milk promotes greater muscle protein accretion after resistance exercise than does consumption of an isonitrogenous and isoenergetic soy-protein beverage. *American Journal of Clinical Nutrition* 85(4), 1031–1040.

Zabala, M., Peinado, A.B., Calderon, F.J., et al. (2011) Bicarbonate ingestion has no ergogenic effect on consecutive all out sprint tests in BMX elite cyclists. *European Journal of Applied Physiology* 111(12), 3127–3134.

Chapter 46

Sprinting: Optimizing Dietary Intake

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Introduction

Sprint performance, or the ability to generate maximal velocities, is important to competitive success of athletes across a range of Olympic disciplines including athletics events and team sports. The latter are characterized by repeated high-intensity sprints. In contrast, the track sprinter is concerned only with generating maximum velocity and with limiting the loss of this as the sprint progresses. The sprints at the Summer Olympic Games include the 100, 200, and 400 m sprints, plus 4×100 m and 4×400 m relay, as well as the 100 m (female)/110 m (male) and 400 m hurdles. Successful athletes in these events are characterized by the ability to generate high velocity for relatively short periods of time.

Sprint performance is determined primarily by reaction time, acceleration, maximum running velocity, and the ability to sustain this in the presence of increasing fatigue (Ross et al., 2001). A sprint event can be broken down into five interlinked components, including the reaction response, block clearance, running acceleration, maximum velocity, and decreasing velocity (Watts et al., 2012). These elements and their contribution to a 100 m race are shown in Table 46.1. During the longer 400 m sprint, running acceleration peaks during the first 100–150 m, followed by a significantly longer decreasing velocity that is accentuated toward the finish of the race, presumably because of substantial acid–base disturbances (Saraslanidis et al., 2011).

Having an appreciation of training and competition demands offers insight into optimum nutrition support for sprinters. Sprinters typically undertake periodized training programs that aim to develop maximum power of the major muscle groups using a range of modalities including not only sprinting but also plyometric exercises, weighted or resisted running drills, plus power and Olympic lifts. Training typically involves brief maximum intensity sprint repetitions of varying length (both below and above competition distance), with either long or short recovery periods. This style of training enhances traits important to athletic development and is common among explosive athletics disciplines. Among elite athletes, this is typically prescribed across multiple daily sessions, most days of the week.

Sprint training adaptations can be separated into several distinct outcomes, including neural and metabolic adaptations (Dawson et al., 1998) as well as physique changes (Watts et al., 2012). While adaptations are dependent on the specific training intervention applied, sprint training appears to induce favorable enzymatic adaptations across all three energy systems, resulting in faster rates of

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Table 46.1 The five components of a sprint race, their percentage contribution to the race, and approximate times for male and female 100 m sprinters

Component	Percent contribution	Male (10.0 s)	Female (11.0 s)
Reaction response	1	0.1–0.3	0.1–0.3
Block clearance	5	0.3–0.4	0.3–0.4
Acceleration	64	5.5–7.0	5.0–6.0
Maximum velocity	18	1.5–3.0	1.5–2.5
Decreasing velocity	12	1.0–1.5	1.5–2.5

Source: Adapted from Watts et al. (2012).

phosphocreatine breakdown as well as greater glycolytic and mitochondrial enzyme activity.

Intense sprint exercise results in rapid increases in energy turnover from both aerobic and anaerobic metabolism. Having an appreciation of energy system contribution influences training prescription as well as directing nutrition guidelines for both training and competition. The relative energy system contribution varies between events, with the anaerobic energy system dominant across all distances. Anaerobic glycolysis is a dominant energy system, as is reflected in the high lactate production, especially during the 400 m (Duffield et al., 2005). This ability to rapidly supply adenosine triphosphate (ATP) via anaerobic sources correlates with performance in the sprint events. The relative aerobic contribution becomes more important as the distance increases, with ~40% of energy derived from the aerobic energy system in the 400 m events (Spencer & Gastin, 2001), and slightly higher contribution for 400 m hurdles.

The ability to generate explosive muscle power and strength is critical to success in sprint events. Given the desire to enhance power-generating capacity, it is often assumed that sprint athletes are primarily interested in promoting muscle hypertrophy. While athletes may periodically attempt to promote skeletal muscle hypertrophy, key nutritional issues are broader than those pertinent to hypertrophy, including the strategic timing of nutrient intake to maximize fueling and recovery objectives, plus the enhancement of power-to-weight ratio, achieved via skeletal muscle hypertrophy and/or maintaining low body fat levels.

The source of fatigue during sprint training is likely multifactorial (Green, 1997), including neuromuscular and peripheral metabolic factors including a decline in intramuscular pH, the latter somewhat dependent on the intensity and volume of training undertaken as well as the time point within a training session. Metabolic fatigue during the earlier part of a workout may be due at least partially to reductions in phosphagen energy system stores and mild acidosis while subsequent fatigue may result more from acidosis and impaired energy production from glycogenolysis.

Dietary Practices and Recommendations

The dietary intakes of sprint athletes are poorly represented in the literature. Table 46.2 summarizes the available literature on dietary intake of sprint athletes, which appears to remain stable throughout the year, presumably independent of training prescription. When contrasted against other track-and-field athletes, relative energy intake is lower among sprinters than in middle-distance and long-distance runners, with a similar trend for carbohydrate, protein, and fat intake (Sugiura et al., 1999). Despite this, micronutrient intakes are similar between runners. Less is known about the distribution of dietary intake throughout the day, including intake before, during, and after exercise, a time where nutrient intake can have a significant impact on not only substrate availability but also adaptation to the training stimulus.

Carbohydrate

Maintaining carbohydrate availability is recognized as a key nutritional strategy for athletes undertaking prolonged endurance exercise, but the ergogenic potential of carbohydrate availability for sprint athletes is less well understood. The metabolic mechanism for the benefit of carbohydrate status on sprint performance is unclear. There is evidence that maintenance of an extremely low-carbohydrate diet can impair performance of events as brief as one 30-second sprint, presumably because of low

Table 46.2 Reported daily dietary intake of energy and macronutrients among sprint athletes during training (unless otherwise stated) since 1980

Gender	Population	Body mass (kg)	Energy		Carbohydrate		Protein		Fat		Survey method	Reference
			MJ	kJ/kg	g	g/kg	g	g/kg	G	% E		
Male												
	National level ($n = 10$)	67	11.1 ± 1.5	167 ± 33	340 ± 57	5.1 ± 1.0	102 ± 20	1.5 ± 0.4	90 ± 16	30 ± 3	3-day diary	Sugiura et al. (1999)
	Adolescent ($n = 30$)	61	11.1 ± 1.5	182	362 ± 54	6.0 ± 0.9	92 ± 17	1.5 ± 0.3	91 ± 21	30 ± 5	7-day diary	Aerenhouts et al. (2008)
Female												
	National level ($n = 11$)	54	10.0 ± 2.2	191 ± 46	305 ± 79	5.8 ± 1.6	89 ± 25	1.7 ± 0.5	86 ± 17	33 ± 4	3-day diary	Sugiura et al. (1999)
	Adolescent ($n = 26$)	55	8.4 ± 1.6	153	273 ± 54	5.1 ± 1.1	78 ± 15	1.5 ± 0.3	69 ± 17	30 ± 5	7-day diary	Aerenhouts et al. (2008)

muscle glycogen stores and decreased rates of glycolysis (Langfort et al., 1997). Indeed, muscle glycogen stores can be reduced by almost half following just three 30-second maximal sprints. However, this alone does not appear to affect sprint exercise performance. Rather, fatigue may be caused by reduced creatine phosphate availability, increased hydrogen ion concentration, impairment in sarcoplasmic reticulum function, or some other fatigue-inducing agent (Hargreaves et al., 1998).

Within the training context, where multiple daily sessions are undertaken including repeat sprints and other modalities such as resistance training and plyometrics, carbohydrate availability may play a more important role, especially among trained individuals, with muscle glycogen stores reduced by 70–80% following repeated 60-second sprints (MacDougall et al., 1977). These reductions in substrate availability are likely sufficient to impair both repeat sprint performance (Rockwell et al., 2003) and other forms of training undertaken by sprinters. Furthermore, a single resistance training session can reduce muscle glycogen stores by as much as 24–40% (Koopman et al., 2006). Reductions in muscle glycogen stores have been associated with performance impairment in both isokinetic torque and isoinertial resistance training capacity. Thus, it is not inconceivable that impaired training or competition performance could occur in any session or event that relies on rapid and repeated glycogen breakdown. If the low carbohydrate status is achieved via a low-carbohydrate, high-protein diet, acid–base status may also be impaired, further adversely affecting performance (Maughan et al., 1997).

Athletes are encouraged to pay particular attention to dietary intake in the hours before exercise, under the assumption that pre-exercise nutritional strategies can influence exercise performance. While this is a widely accepted practice prior to endurance exercise to enhance work capacity (Hargreaves et al., 2004), evidence is also emerging for a beneficial role of acute carbohydrate ingestion prior to strength training. For example, Lambert et al. (1991) reported that supplemental carbohydrate ingestion prior to and during resistance exercise (1 g/kg before, 0.5 g/kg during) increased total work capacity, a response that has been replicated elsewhere. However, not all investigations show a benefit of acute carbohydrate ingestion; we propose that the ergogenic potential for carbohydrate ingestion is most likely to be observed when undertaking longer duration, high-volume training among sprint athletes. At present, a specific recommendation for an optimum rate or timing of carbohydrate ingestion for sprint athletes before and during any given training session cannot be determined.

Dietary survey literature relating to sprint athletes suggests they typically report daily carbohydrate intakes of 5–6 g/kg body mass, independent of gender (Table 46.2). While this may appear low relative to the intakes of endurance athletes, conclusive evidence of benefit from maintaining a habitual high

carbohydrate intake among sprint athletes remains to be confirmed. Given the lower relative energy expenditure of larger athletes and their requirements for other nutrients, plus the effect of adjusting carbohydrate on total energy intake, recommendations for carbohydrate intake at strategic times, including before, during, and after exercise, may be more applicable for the sprint athlete, ensuring carbohydrate availability is optimized at critical time points. Thus, we would consider a range of daily carbohydrate intakes between 5 and 6 g/kg body mass as reasonable for these athletes depending on their phase of training (Tipton et al., 2007).

Protein

Strength-power athletes like sprinters have advocated high-protein diets for many years. While debate continues on the need for additional protein among sprint-trained athletes, general guidelines now recommend that athletes undertaking strength-power training ingest approximately twice the current recommendations for protein of their sedentary counterparts or as much as 1.6–1.7 g protein/kg/day (Phillips, 2004). Given the relatively wide distribution of protein in the meal plan and increased energy intake of athletes, it should not be surprising to learn that the majority of sprint athletes achieve these increased protein intake targets (Table 46.2). Exceeding the upper range of protein intake guidelines offers no further benefit and simply promotes increased protein oxidation. Furthermore, there is evidence that an intense period of resistance training reduces protein turnover and improves net protein retention, thus reducing relative dietary protein requirements of experienced resistance-trained athletes.

Simply contrasting an athlete's current daily protein intake against guidelines does not address whether protein intake has been optimized to promote gains in muscle mass or optimize repair of damaged tissues. Rather, consideration should be given to other dietary factors, including total energy intake, the daily distribution of protein intake, especially as it relates to training, and the source of dietary protein. While there is very little information available on the eating patterns of sprint athletes, available literature suggests the majority of daily protein intake is ingested at main meals, with little consideration for between-meal intake, presumably inclusive of pre- and posttraining snacks (Burke et al., 2003). Thus, rather than focusing on total daily intake, sprint athletes are encouraged to consume rapidly digested protein meals/snacks in close proximity to their exercise bout, with ingestion during and soon after exercise appearing to be most favorable (Phillips & Van Loon, 2011). Less is known about the effect of protein distribution in the meal plan outside the acute period before and/or after exercise (<3 hours). There is some evidence to suggest protein breakdown may be less with a wider distribution of daily protein intake than with an acute daily bolus of protein (Arnal et al., 2000). However, given muscle protein synthesis becomes refractory to persistent aminoacidemia, Moore et al. (2009) suggest the ingestion of 20 g of high biological value protein no more than five to six times daily may result in maximal stimulation of muscle protein synthesis.

Hydration

As with all athletes, sprint athletes are encouraged to initiate training in a euhydrated state. However, the duration of sprint events is clearly such that no hydration intervention is warranted during the event itself. Furthermore, the reduction in body mass associated with hypohydration may reduce the work required to accelerate the body, compensating for any reduction in muscular strength/power (Maughan & Shirreffs, 2010). Indeed, there is some evidence to show an enhancement in vertical jump performance following dehydration equivalent to ~3% of body mass (Viitasalo et al., 1987). Furthermore, sprint performance over distances of 50–400 m remain stable despite an acute reduction in body mass equivalent to 2.0–2.5% (Watson et al., 2005). Despite this, there is evidence to suggest longer duration activities undertaken by sprint athletes such as resistance training are impaired by hypohydration (Kraft et al., 2010). On the weight of this evidence, track sprinting performance does not appear to be influenced by a state of hypohydration within the range of 2–3% (see Chapter 15).

However, sprint training characterized by repeat high-intensity efforts may be impaired by hypohydration. Furthermore, gastrointestinal tract tolerance of fluid ingestion during running is often compromised, although athletes can be trained to better tolerate fluid intake while running if there is a rationale for increased fluid intake during exercise. Sprint athletes are advised to start training in a state of euhydration, drink to their thirst and gastrointestinal tolerance, and limit body mass loss to no more than 2–3% during any one training session, complementing this with aggressive postexercise recovery strategies, inclusive of adequate fluid and electrolytes.

Recovery

Given that sprint athletes typically undertake multiple daily training sessions, recovery strategies proven to enhance restoration of muscle glycogen stores, such as postexercise carbohydrate ingestion, should be routinely implemented following training. General sports nutrition guidelines advocate the ingestion of carbohydrate at a rate of 1.0–1.2 g/kg body mass in the immediate postexercise period (Burke et al., 2004). However, this has no influence on muscle protein metabolism (Koopman et al., 2007). In contrast, postexercise dietary protein ingestion results in an exacerbated elevation in muscle protein synthesis with a concomitant minor suppression in muscle protein breakdown, resulting in a positive net protein balance (Phillips et al., 2009) (see Chapter 11). The ingestion of ~20 g of high biological value protein after resistance exercise appears to be sufficient to maximally stimulate muscle protein synthesis, with amounts in excess of this merely promoting protein oxidation (Moore et al., 2009). Thus, the combined ingestion of carbohydrate and protein acutely following sprint athlete training results in more favorable recovery outcomes, including restoration of muscle glycogen stores and muscle protein metabolism, than the ingestion of either nutrient alone. Postexercise protein ingestion also lowers carbohydrate intake requirements in the acute recovery period, with an energy-matched intake of 0.8 g/kg/h carbohydrate plus 0.4 g/kg/h protein resulting in similar muscle glycogen resynthesis over 5 hours compared to 1.2 g/kg/h carbohydrate alone following intermittent exercise (Van Loon et al., 2000), with a similar response evident following resistance exercise. Preliminary evidence also suggests that the postexercise co-ingestion of carbohydrate and protein may reduce the muscle damage often seen in strength-trained athletes (Cockburn et al., 2010); whether such a response has a functional benefit is unclear.

Supplementation

Supplement use is reported to be higher among athletes than their sedentary counterparts, with 85% of elite track-and-field athletes competing in International Amateur Athletics Federation (IAAF) championships acknowledging the use of at least one dietary supplement (Maughan et al., 2007), a rate higher than that observed in internationally competitive junior track-and-field athletes (Nieper, 2005). The incidence of supplement use among runners varies based on the event, with sprinters reported to have both higher (Tscholl et al., 2010) and lower (Maughan et al., 2007) rates of supplement use relative to distance runners, with poly-supplementation common. While multivitamin and mineral supplements remain popular, protein powders and specific amino acid supplements, caffeine, and creatine monohydrate are also frequently used by sprinters (Nieper, 2005; Tscholl et al., 2010). Sprinters are motivated to take supplements to enhance recovery, health, and performance (Maughan et al., 2007; Nieper, 2005). Sprint athletes continue to seek supplement information from readily accessible sources including magazines, fellow athletes, and coaches. Consequently, the accuracy of information provided may vary, leaving the athlete vulnerable to inappropriate and/or ineffective supplementation protocols.

After reviewing the metabolic demands of sprinting, Tipton and colleagues describe four supplements that could benefit the sprint athlete, whether in training or competition, including β-alanine, sodium bicarbonate, creatine, and caffeine (Tipton et al., 2007). The majority of the energy required during a single bout of brief, maximal exercise is provided via anaerobic pathways, specifically

glycogenolysis resulting in lactate formation and phosphocreatine degradation. Interventions able to influence energy availability via these pathways may favorably affect sprint exercise performance.

Given the dependence on anaerobic glycogenolysis and the associated acid–base disturbances, sprint performance may be enhanced if buffering capacity can be increased, especially the longer duration sprints characterized by substantial acid–base disturbances. Carr and associates estimate a performance enhancement of ~2% during a 1-minute sprint from a dose of 0.3 g/kg body mass of sodium bicarbonate, with even greater improvement when undertaken prior to repeat sprints, as may occur during sprint training (Carr et al., 2011). The impact of buffering agent ingestion on shorter duration sprint performance has not been explored but is likely smaller than that observed in the longer sprints.

More recently, there has been interest in the histidine-containing dipeptide carnosine, which contributes significantly to the physiochemical buffering in skeletal muscles. Indeed, muscle carnosine concentration correlates with power output during a 30-second sprint, especially the latter half of the sprint (Suzuki et al., 2002). Chronic supplementation with β-alanine, the rate-limiting precursor to carnosine synthesis, has been shown to augment muscle carnosine content. Although not entirely consistent, there is now a growing body of evidence to show that chronic β-alanine supplementation improves exercise capacity during exercise limited by the accumulation of hydrogen ions (Sale et al., 2010) (see Chapter 26). Thus, among sprint athletes, an ergogenic effect is most likely for the longer duration sprints, although this is not always evident, especially in highly trained sprinters (Derave et al., 2007). The manipulation of both blood (sodium bicarbonate or sodium citrate supplementation) and muscle buffering capacity (β-alanine supplementation) via dietary manipulation also has potential, although again this has not been explored among sprint athletes.

Finally, caffeine ingestion has been used successfully to enhance exercise performance across a range of sports, including endurance events, stop-and-go events, and sports involving sustained high-intensity activity lasting 1–60 minutes (see Chapter 25). However, the impact of caffeine ingestion on sprint performance is less well described and somewhat contradictory (Astorino & Roberson, 2010). Despite this, there is some evidence that caffeine (5 mg/kg body mass) can enhance both single and multiple sprint performance (Glaister et al., 2008), and its use should be considered in the supplementation armory of sprint-trained athletes.

Competition Nutrition Strategies

While sprint performance is important to competitive success in other Olympic events like athletic jumps and team sports, these are characterized by repeat high-intensity sprints. In contrast, the track sprinter is concerned with running as fast as possible for a single effort. In major competitions, a sprint athlete must advance through qualifying rounds, a semi final, and final, each typically separated by several hours, and in the case of the longer sprints, typically a 24-hour period. Given the brief nature of sprint events, the relative importance of competition nutrition strategies might be assumed to be negligible, but there is evidence to suggest that pre-competition nutrition, including the use of some ergogenic aids, influences performance outcomes in these events. Consideration should be given to pre-competition muscle glycogen stores, promoting a state of euhydration and acid–base status.

Competition demands of sprinters are typically characterized by high-intensity efforts lasting approximately 10–60 seconds, with significant recovery between races. Due to the scheduling of major competitions (Table 46.3), it is rare for sprinters to participate in more than two individual events, although athletes competing in multiple events, including relays, may have several races on a single day. With significant periods for recovery between races, muscle energy reserves are unlikely to be depleted, even in challenging environmental conditions of competitions like the Summer Olympic Games. Consequently, nutrition priorities remain with more general goals like optimizing gastrointestinal tract comfort and preventing weight gain during the competition taper.

Table 46.3 The sprint event schedule for males at the 2011 World Championships

100 m		
Day 1	21:45 h	Heats
Day 2	18:30 h	Semi finals
Day 2	20:45 h	Final
Day 9	19:00 h	4 × 100 m heats
Day 9	21:00 h	4 × 100 m final
200 m		
Day 7	11:10 h	Heats
Day 7	19:55 h	Semi finals
Day 8	21:20 h	Final
400 m		
Day 2	11:15 h	Heats
Day 3	20:00 h	Semi finals
Day 4	21:45 h	Final
Day 6	12:30 h	4 × 400 m heats
Day 7	21:15 h	4 × 400 m final
110 m hurdles		
Day 2	9:50 h	Heats
Day 3	19:00 h	Semi finals
Day 3	21:25 h	Final
400 m hurdles		
Day 3	11:30 h	Heats
Day 4	19:30 h	Semi finals
Day 6	21:30 h	Final

As depicted in the competition schedule specified in Table 46.3, major international track-and-field competitions generally see the initial heats of an event being held early in the day while finals are often run in the evening. Prerace nutrition from qualifying rounds to finals will therefore involve different meals. A key consideration for the prerace meal, regardless of the time of day, is to consume a comfortable, familiar meal. Where gastrointestinal symptoms have been reported in 20–50% of endurance athletes, the shorter duration of sprint events means that gastrointestinal disturbances are not as commonly reported, but inappropriate food selection may affect an athlete's energy availability and gut comfort. A state of low carbohydrate availability has been shown to impair anaerobic work capacity in events of as little as 30-second duration (Langfort et al., 1997). However, this effect is evident only following severe dietary carbohydrate restriction, sufficient to promote a state of ketosis. Such a state is unlikely among competitive athletes tapering prior to competition who follow a meal plan with even a moderate carbohydrate content. In light of this, sprint athletes are advised to choose a familiar meal ideally containing 1–2 g/kg body mass of carbohydrate approximately 1–4 hours prior to competition.

The use of prerace ergogenic aids, such as buffering agents or caffeine, requires careful consideration of the competition schedule. Athletes using these products should determine the optimum dosage and timing for enhanced performance across single and repeat performances as repeat dosing may be considered when races are close together, such as the 100 m semi final and final. Administering a standard dose prior to each race may result in adverse outcomes depending on the specific product and its half-life. Given this, it is essential that athletes trial supplement strategies in training or smaller competitions to determine optimal dosage and timing of administration. In the case of bicarbonate, this may result in an athlete choosing to complete a chronic supplementation strategy over a period of days, rather than an acute loading protocol immediately prior to competition (McNaughton & Thompson, 2001).

Novel pre-competition approaches to facilitate acute weight loss in an attempt to optimize power-to-weight have been implemented by some sprint athletes in recent times. Although there is no research supporting this practice in a sporting context, low-residue diets have been shown to facilitate weight loss, as a result of decreased fecal matter. Weight loss associated with the adoption of a low-residue diet is individual and influenced by factors such as gender and body size. The weight loss associated with adherence to a low-residue meal plan for a day may be within the range of 200–400 g and thus may be insufficient to significantly affect the power-to-weight ratio. Intentional dehydration is an alternative approach to promote greater weight

loss acutely prior to competition but existing evidence suggests sprint performance over distances of 50–400 m remain stable despite an acute reduction in body mass equivalent to 2.0–2.5% (Watson et al., 2005). Athletes wishing to trial these acute weight loss strategies before competition should seek professional advice and support in advance so that health and performance implications can be more closely assessed.

Physique Traits

Despite a long history of sprinting in the Olympic Games, relatively few studies describe the physique of sprinters. What is known is that successful sprinters have physique traits that predispose them to excellence. Some of these are responsive to training stimuli and nutritional interventions, including skeletal muscle fiber type and area (Dowson et al., 1998), fascicle area and length (Aagaard et al., 2001; Abe et al., 2001) plus adiposity (Dowson et al., 1998), while other architectural features such as stature, toe, foot, and lower leg length are not (Lee & Piazza, 2009). The available literature clearly reflects an emphasis on the importance for sprinters to maximize skeletal muscle mass to enhance power. However, this may not be appropriate for all sprinters with skeletal muscle hypertrophy possibly resulting in adverse adaptations, including a transition away from fast-twitch glycolytic fibers and slower contraction velocity characteristics (Alway et al., 1988) if inappropriately prescribed. Thus, unless the increase in power proportionally exceeds any associated weight gain, sprint performance is unlikely to be enhanced by an increase in skeletal muscle mass.

Sprinters do tend to be heavier and more muscular than other runners. Early data from Carter (1984) of athletes participating in the 1960, 1968, and 1976 Olympic Games reported sprinters had a somatotype (endomorphy, mesomorphy, ectomorphy) of 1.5-3.3 for males and 2.5-4.3 for females, with both genders characterized as ecto-mesomorphs. These ratings are consistent with more contemporary data (Abe et al., 2001) demonstrating sprinters have higher muscularity and low relative adiposity. Sprinters are not on average the tallest or most ectomorphic of the running disciplines and are reported (Uth, 2005) to have a reasonably wide range for stature (men: 1.68–1.91 m; women: 1.52–1.82 m). However, in the past decade, the stature of successful male sprinters has been biased toward the upper limit or even exceeding this range, with speed records suggested to continue to be dominated by heavier and taller athletes (Charles & Bejan, 2009).

A recent manuscript by Watts et al. (2012) provides a comprehensive description of the evolution of successful world-class 100 m sprinter mass and stature characteristics (Table 46.4). The available data span ten decades (1910–2009) for men and eight (1940–2010) for women. Records typically included top ten 100 m sprinters for both genders during these periods. Athlete speed (m/s), body mass index (BMI; kg/m^2), and reciprocal ponderal index (RPI; $cm/kg^{0.333}$) were calculated. The BMI provided a proxy for muscularity and the RPI a measure of linearity, with higher BMI and RPI representing greater muscularity and tallness/linearity, respectively. As expected, speed increased over the decades in both genders. In the male world-class sprinters, a high BMI was positively associated with success until the most recent decade where the trend was reversed. Most interesting was the finding that recently successful sprinters tended to have higher RPI, that is, taller, more linear sprinters are achieving greater success. This was consistently observed over the decades for women with RPI positively associated with success and BMI exhibiting a weaker, negative association for speed. Taken together, it would appear that the influence of muscle mass to sprint performance is less important, with taller, more linear sprinters achieving greater success. This might be explained by the influence of stride length on sprint speed.

Interestingly, the current 100 m world record for women of 10.49 seconds attained in 1988 (Florence Griffith-Joyner) was for a runner (stature 1.70 m; mass 59 kg) with a more extreme RPI (43.728 $cm/kg^{0.333}$). The next closest time (10.65 seconds) to this long-standing world record was for another "linear" sprinter (Marion Jones) with an identical RPI (stature 1.80 m; mass 70 kg). For men, the current world record time (set in 2009) for 100 m sprint (9.58 sec-

Table 46.4 Body size and shape characteristics of male and female world-class sprinters per decade

Decade	Gender (*n*)	Age (years)	Body mass (kg)	Stature (m)	BMI (kg/m^2)	RPI ($cm/kg^{0.333}$)
1940–1949	F(3)	28.8 ± 5.0	65.3 ± 9.9	1.73 ± 0.1	21.7 ± 1.4	43.2 ± 0.9
1950–1959	F(2)	28.5 ± 6.4	65.4 ± 2.1	1.74 ± 0.02	21.4 ± 1.2	43.3 ± 1.0
1960–1969	F(2)	25.8 ± 4.6	58.5 ± 2.1	1.75 ± 0.08	19.4 ± 1.2	45.1 ± 1.6
1970–1979	F(2)	23.4 ± 4.1	62.0 ± 1.4	1.73 ± 0.04	20.7 ± 0.5	43.8 ± 0.7
1980–1989	F(71)	25.0 ± 3.1	57.7 ± 3.9	1.68 ± 0.05	20.4 ± 1.1	43.6 ± 1.1
1990–1999	F(74)	26.4 ± 4.0	59.1 ± 5.2	1.69 ± 0.07	20.8 ± 1.5	43.4 ± 1.3
2000–2009	F(74)	26.2 ± 3.9	58.7 ± 6.6	1.66 ± 0.07	21.1 ± 1.4	42.9 ± 1.0
2010	F(1)	22.4 ± 2.0	56	1.70	19.4	44.5
1910–1919	M(9)	23.3 ± 3.0	69.2 ± 3.6	1.77 ± 0.04	22.1 ± 1.4	43.3 ± 1.1
1920–1929	M(23)	23.6 ± 2.9	69.5 ± 4.7	1.73 ± 0.04	23.3 ± 1.3	42.1 ± 0.9
1930–1939	M(25)	23.7 ± 2.8	74.6 ± 6.7	1.78 ± 0.03	23.4 ± 1.8	42.4 ± 1.1
1940–1949	M(22)	23.4 ± 3.3	72.4 ± 4.6	1.80 ± 0.03	22.5 ± 1.8	43.2 ± 1.3
1950–1959	M(30)	24.1 ± 3.2	71.9 ± 6.3	1.76 ± 0.09	23.3 ± 2.3	42.3 ± 2.0
1960–1969	M(23)	23.6 ± 3.0	75.9 ± 7.3	1.79 ± 0.07	23.7 ± 1.3	42.4 ± 1.1
1970–1979	M(21)	23.2 ± 2.9	76.9 ± 5.4	1.84 ± 0.07	22.8 ± 1.9	43.3 ± 1.5
1980–1989	M(34)	23.8 ± 2.7	73.7 ± 6.0	1.83 ± 0.05	21.9 ± 1.2	43.9 ± 0.8
1990–1999	M(54)	26.1 ± 3.5	75.2 ± 5.3	1.80 ± 0.05	23.1± 1.6	42.8 ± 1.2
2000–1909	M(99)	25.6 ± 3.4	77.9 ± 5.6	1.81 ± 0.06	23.8 ± 1.8	42.5 ± 1.4

Source: Adapted from Watts et al. (2012). Data are reported as mean ± standard deviation.
M, male; F, female; BMI, body mass index; RPI, reciprocal ponderal index.

onds) was for an athlete with a stature of 1.93 m (Usain Bolt). It is recognized, though, that the use of prohibited performance-enhancing pharmaceuticals by some athletes may confound this type of analysis.

Rather than absolute power output, acceleration in sprinting is also a function of power-to-weight. An increase muscle cross-sectional area is usually accompanied by greater muscle strength and power but the ability to generate force also requires improved neuromuscular recruitment. In a study comparing heavier, more muscular adult to adolescent sprinters, higher muscularity and mass were reported to explain slower sprint start dynamics in the adults (Aerenhouts et al., 2012). While optimizing muscle mass is important for the development of explosive power, especially at the sprint start, training should advance technical skills to facilitate effective transfer of strength benefits. Although most studies report a "normalized" muscle strength of participants by adjusting for total or lean body mass, this unfortunately fails to adequately account for regional mass differences (e.g., differences in upper to lower body mass), which are likely important to performance. Locating mass closer to the joint center helps optimize biomechanical efficiency, a concept supported by research showing sprinters with greater deposition of muscle in the upper portion of the quadriceps are faster (Kumagai et al., 2000).

Muscularity for sprinters needs to be optimized rather than maximized and currently there are insufficient comprehensive morphological data to provide detailed guidance. Small differences in adiposity on the limbs of sprinters have also been demonstrated to predict performance with relatively small reductions in medial calf skinfold associated with faster run times (Legaz & Eston, 2005). This suggests that subtle differences in the distribution of

mass influences performance, possibly the result of increased muscular effort and energy expenditure associated with heavier lower limbs when running. This raises the concept of optimizing nutritional support of some training sessions but not others, i.e., support those where hypertrophy may be beneficial but do not optimize nutritional support of other sessions where you want neural adaptations but not a hypertrophy response. Such an approach would demand strategic prescription of nutrition support in accordance to the training program, with significant dialogue between athlete, coaching personnel, and nutrition professional.

Conclusions

Nutrition plays a number of important roles for sprint athletes. Sprint athletes will benefit from a greater focus on training nutrition given the metabolic demands of training far exceed those of competition. An emphasis should be placed on the strategic timing of nutrient intake before, during, and after exercise to assist sprinters in optimizing resistance training work capacity, recovery, and body composition. While it is often assumed that sprint athletes are primarily interested in promoting muscle hypertrophy, optimization of body composition demands consideration of the effect of any changes in physique traits on power-to-weight and biomechanical efficiency. Nutritional supplements remain very popular among sprinters and there is evidence to support the use of a small number of products to assist sprinters in the training and/or competition environment. However, as with any dietary intervention for sprinters, these need to be trialed in training to assess tolerance and likely individual performance response.

References

Aagaard, P., Andersen, J.L., Dyhre-Poulsen, P., et al. (2001) A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. *Journal of Physiology* 534(Pt 2), 613–623.

Abe, T., Fukashiro, S., Harada, Y., et al. (2001) Relationship between sprint performance and muscle fascicle length in female sprinters. *Journal of Physiological Anthropology and Applied Human Science* 20(2), 141–147.

Aerenhouts, D., Hebbelinck, M., Poortmans, J.R., et al. (2008) Nutritional habits of Flemish adolescent sprint athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 18(5), 509–523.

Aerenhouts, D., Delecluse, C., Hagman, F., et al. (2012) Comparison of anthropometric characteristics and sprint start performance between elite adolescent and adult sprint athletes. *European Journal of Sport Science* 12(1), 9–15.

Alway, S.E., MacDougall, J.D., Sale, D.G., et al. (1988) Functional and structural adaptations in skeletal muscle of trained athletes. *Journal of Applied Physiology* 64(3), 1114–1120.

Arnal, M.A., Mosoni, L., Boirie, Y., et al. (2000) Protein feeding pattern does not affect protein retention in young women. *Journal of Nutrition* 130(7), 1700–1704.

Astorino, T.A. & Roberson, D.W. (2010) Efficacy of acute caffeine ingestion for short-term high-intensity exercise performance: a systematic review. *Journal of Strength and Conditioning Research* 24(1), 257–265.

Burke, L.M., Slater, G., Broad, E.M., et al. (2003) Eating patterns and meal frequency of elite Australian athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 13(4), 521–538.

Burke, L.M., Kiens, B., & Ivy, J.L. (2004) Carbohydrates and fat for training and recovery. *Journal of Sports Sciences* 22(1), 15–30.

Carr, A.J., Hopkins, W.G., & Gore, C.J. (2011) Effects of acute alkalosis and acidosis on performance: a meta-analysis. *Sports Medicine* 41(10), 801–814.

Carter, J. (1984) Somatotypes of Olympic athletes from 1948–1976. In *Physical Structure of Olympic Athletes: Part II. Kinanthropometry of Olympic Athletes* (ed. J. Carter), pp. 80–109. Karger, Basel, Switzerland.

Charles, J.D. & Bejan, A. (2009) The evolution of speed, size and shape in modern athletics. *Journal of Experimental Biology* 212(Pt 15), 2419–2425.

Cockburn, E., Stevenson, E., Hayes, P.R., et al. (2010) Effect of milk-based carbohydrate-protein supplement timing on the attenuation of exercise-induced muscle damage. *Applied Physiology, Nutrition, and Metabolism* 35(3), 270–277.

Dawson, B., Fitzsimons, M., Green, S., et al. (1998) Changes in performance, muscle metabolites, enzymes and fibre types after short sprint training. *European Journal of Applied Physiology and Occupational Physiology* 78(2), 163–169.

Derave, W., Ozdemir, M.S., Harris, R.C., et al. (2007) beta-Alanine supplementation augments muscle carnosine content and attenuates fatigue during repeated isokinetic contraction bouts in trained sprinters. *Journal of Applied Physiology* 103(5), 1736–1743.

Dowson, M.N., Nevill, M.E., Lakomy, H.K., et al. (1998) Modelling the relationship between isokinetic muscle strength and sprint running performance. *Journal of Sports Sciences* 16(3), 257–265.

Duffield, R., Dawson, B., & Goodman, C. (2005) Energy system contribution to 400-metre and 800-metre track running. *Journal of Sports Sciences* 23(3), 299–307.

Glaister, M., Howatson, G., Abraham, C.S., et al. (2008) Caffeine supplementation and multiple sprint running performance. *Medicine & Science in Sports & Exercise* 40(10), 1835–1840.

Green, H.J. (1997) Mechanisms of muscle fatigue in intense exercise. *Journal of Sports Sciences* 15(3), 247–256.

Hargreaves, M., McKenna, M.J., Jenkins, D.G., et al. (1998) Muscle metabolites and performance during high-intensity, intermittent exercise. *Journal of Applied Physiology* 84(5), 1687–1691.

Hargreaves, M., Hawley, J.A., & Jeukendrup, A. (2004) Pre-exercise carbohydrate and fat ingestion: effects on metabolism and performance. *Journal of Sports Sciences* 22(1), 31–38.

Koopman, R., Manders, R.J., Jonkers, R.A., et al. (2006) Intramyocellular lipid and glycogen content are reduced following resistance exercise in untrained healthy males. *European Journal of Applied Physiology* 96(5), 525–534.

Koopman, R., Beelen, M., Stellingwerff, T., et al. (2007) Coingestion of carbohydrate with protein does not further augment postexercise muscle protein synthesis. *American Journal of Physiology—Endocrinology and Metabolism* 293(3), E833–E842.

Kraft, J.A., Green, J.M., Bishop, P.A., et al. (2010) Impact of dehydration on a full body resistance exercise protocol. *European Journal of Applied Physiology* 109(2), 259–267.

Kumagai, K., Abe, T., Brechue, W.F., et al. (2000) Sprint performance is related to muscle fascicle length in male 100-m sprinters. *Journal of Applied Physiology* 88(3), 811–816.

Lambert, C.P., Flynn, M.G., Boone, J.B.J., et al. (1991) Effects of carbohydrate feeding on multiple-bout resistance exercise. *Journal of Strength and Conditioning Research* 5(4), 192–197.

Langfort, J., Zarzeczny, R., Pilis, W., et al. (1997) The effect of a low-carbohydrate diet on performance, hormonal and metabolic responses to a 30-s bout of supramaximal exercise. *European Journal of Applied Physiology and Occupational Physiology* 76(2), 128–133.

Lee, S.S. & Piazza, S.J. (2009) Built for speed: musculoskeletal structure and sprinting ability. *Journal of Experimental Biology* 212(Pt 22), 3700–3707.

Legaz, A. & Eston, R. (2005) Changes in performance, skinfold thicknesses, and fat patterning after three years of intense athletic conditioning in high level runners. *British Journal of Sports Medicine* 39(11), 851–856.

MacDougall, J.D., Ward, G.R., & Sutton, J.R. (1977) Muscle glycogen repletion after high-intensity intermittent exercise. *Journal of Applied Physiology* 42(2), 129–132.

Maughan, R.J. & Shirreffs, S.M. (2010) Development of hydration strategies to optimize performance for athletes in high-intensity sports and in sports with repeated intense efforts. *Scandinavian Journal of Medicine and Science in Sports*, 20(Suppl. 2), 59–69.

Maughan, R.J., Greenhaff, P.L., Leiper, J.B., et al. (1997) Diet composition and the performance of high-intensity exercise. *Journal of Sports Sciences* 15(3), 265–275.

Maughan, R.J., Depiesse, F., & Geyer, H. (2007) The use of dietary supplements by athletes. *Journal of Sports Sciences* 25(Suppl.1), S103–S113.

McNaughton, L. & Thompson, D. (2001) Acute versus chronic sodium bicarbonate ingestion and anaerobic work and power output. *Journal of Sports Medicine and Physical Fitness* 41(4), 456–462.

Moore, D.R., Robinson, M.J., Fry, J.L., et al. (2009) Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. *American Journal of Clinical Nutrition* 89(1), 161–168.

Nieper, A. (2005) Nutritional supplement practices in UK junior national track and field athletes. *British Journal of Sports Medicine* 39(9), 645–649.

Phillips, S.M. (2004) Protein requirements and supplementation in strength sports. *Nutrition* 20(7–8), 689–695.

Phillips, S.M. & Van Loon, L.J. (2011) Dietary protein for athletes: from requirements to optimal adaptation. *Journal of Sports Sciences* 29(Suppl. 1), S29–S38.

Phillips, S.M., Tang, J.E., & Moore, D.R. (2009) The role of milk- and soy-based protein in support of muscle protein synthesis and muscle protein accretion in young and elderly persons. *Journal of the American College of Nutrition* 28(4), 343–354.

Rockwell, M.S., Rankin, J.W., & Dixon, H. (2003) Effects of muscle glycogen on performance of repeated sprints and mechanisms of fatigue. *International Journal of Sport Nutrition and Exercise Metabolism* 13(1), 1–14.

Ross, A., Leveritt, M., & Riek, S. (2001) Neural influences on sprint running: training adaptations and acute responses. *Sports Medicine* 31(6), 409–425.

Sale, C., Saunders, B., & Harris, R.C. (2010) Effect of beta-alanine supplementation on muscle carnosine concentrations and exercise performance. *Amino Acids* 39(2), 321–333.

Saraslanidis, P.J., Panoutsakopoulos, V., Tsalis, G.A., et al. (2011) The effect of different first 200-m pacing strategies on blood lactate and biomechanical parameters of the 400-m sprint. *European Journal of Applied Physiology* 111(8), 1579–1590.

Spencer, M.R. & Gastin, P.B. (2001) Energy system contribution during 200- to 1500-m running in highly trained athletes. *Medicine & Science in Sports & Exercise* 33(1), 157–162.

Sugiura, K., Suzuki, I., & Kobayashi, K. (1999) Nutritional intake of elite Japanese track-and-field athletes. *International Journal of Sport Nutrition* 9(2), 202–212.

Suzuki, Y., Ito, O., Mukai, N., et al. (2002) High level of skeletal muscle carnosine contributes to the latter half of exercise performance during 30-s maximal cycle ergometer sprinting. *Japanese Journal of Physiology* 52(2), 199–205.

Tipton, K.D., Jeukendrup, A.E., Hespel, P., et al. (2007) Nutrition for the sprinter. *Journal of Sports Sciences*, 25(Suppl. 1), S5–S15.

Tscholl, P., Alonso, J.M., Dolle, G., et al. (2010) The use of drugs and nutritional supplements in top-level track and field athletes. *American Journal of Sports Medicine* 38(1), 133–140.

Uth, N. (2005) Anthropometric comparison of world-class sprinters and normal populations. *Journal of Sports Science and Medicine* 4(4), 608–616.

Van Loon, L.J., Saris, W.H., Kruijshoop, M., et al. (2000) Maximizing postexercise muscle glycogen synthesis: carbohydrate supplementation and the application of amino acid or protein hydrolysate mixtures. *American Journal of Clinical Nutrition* 72(1), 106–111.

Viitasalo, J.T., Kyrolainen, H., Bosco, C., et al. (1987) Effects of rapid weight reduction on force production and vertical jumping height. *International Journal of Sports Medicine* 8(4), 281–285.

Watson, G., Judelson, D.A., Armstrong, L.E., et al. (2005) Influence of diuretic-induced dehydration on competitive sprint and power performance. *Medicine & Science in Sports & Exercise* 37(7), 1168–1174.

Watts, A.S., Coleman, I., & Nevill, A. (2012) The changing shape characteristics associated with success in world-class sprinters. *Journal of Sports Sciences* 30(11), 1085–1095.

Chapter 47

Distance Running

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Introduction

As compared to the last 100+ years of distance running, the last decade has seen an explosion of performances, specifically the men's marathon which has entered an unprecedented era of performance. Just a decade ago, the marathon world record stood at 2:05:42. That time has now been bettered 37 times by 24 different athletes (23 of them being East African), with the world record currently standing at 2:03:38 (as of June 2013). This extraordinary performance progression has ignited the debate on the limits of human endurance, as captured by a recent scientific article titled, "The two-hour marathon: who and when?" (Joyner et al., 2011), and the numerous published counter commentaries (Stellingwerff & Jeukendrup, 2011).

Currently, the majority of evidence provided for the determinants of distance running performance has been physiologically based around aerobic capacity (VO_2 max), lactate threshold, and running economy, which are certainly crucial elements (Joyner & Coyle, 2008; Joyner et al., 2011). However, given that muscle glycogen is the dominant fuel at exercise intensities greater than 75% VO_2 max and can start to become limiting after ~90 minutes, this review will attempt to outline how nutrition-based interventions can also be fundamental determinants of distance running performance of longer than ~90 minutes (Figure 47.1). Further to this, recent studies have started to examine the interactions that nutrition and training may have on endurance training adaptations. For example, a periodic lack of carbohydrate (CHO) availability may further drive training adaptations (Hawley & Burke, 2010), the gastrointestinal tract may also adapt to handling increased fluid and CHO, and an individualized approach to race-day CHO type and fluid intake may contribute to distance running success (Jeukendrup, 2011; Pfeiffer et al., 2011; Stellingwerff, 2012).

Thus, this chapter will attempt to integrate the basic science described earlier in this book on CHO loading and daily requirements, CHO ingestion, and fluid requirements during exercise, along with several possible ergogenic nutritional supplements relevant to distance running performance. Although this chapter is focusing on practical, periodized, and individualized nutrition recommendations for the marathon runner, the majority of the information would also be applicable to race distances from 10,000 m to ultramarathons (50 km to 100 mile races).

Determinants of Endurance Running Performance

The physiological characteristics of endurance champions have been eloquently reviewed by Joyner and Coyle (2008). What this chapter hopes to portray is the intimate interplay between physiology and optimum nutrition, and Figure 47.1 provides the framework.

In this figure, the physiology-based determinants of endurance performance are outlined in light

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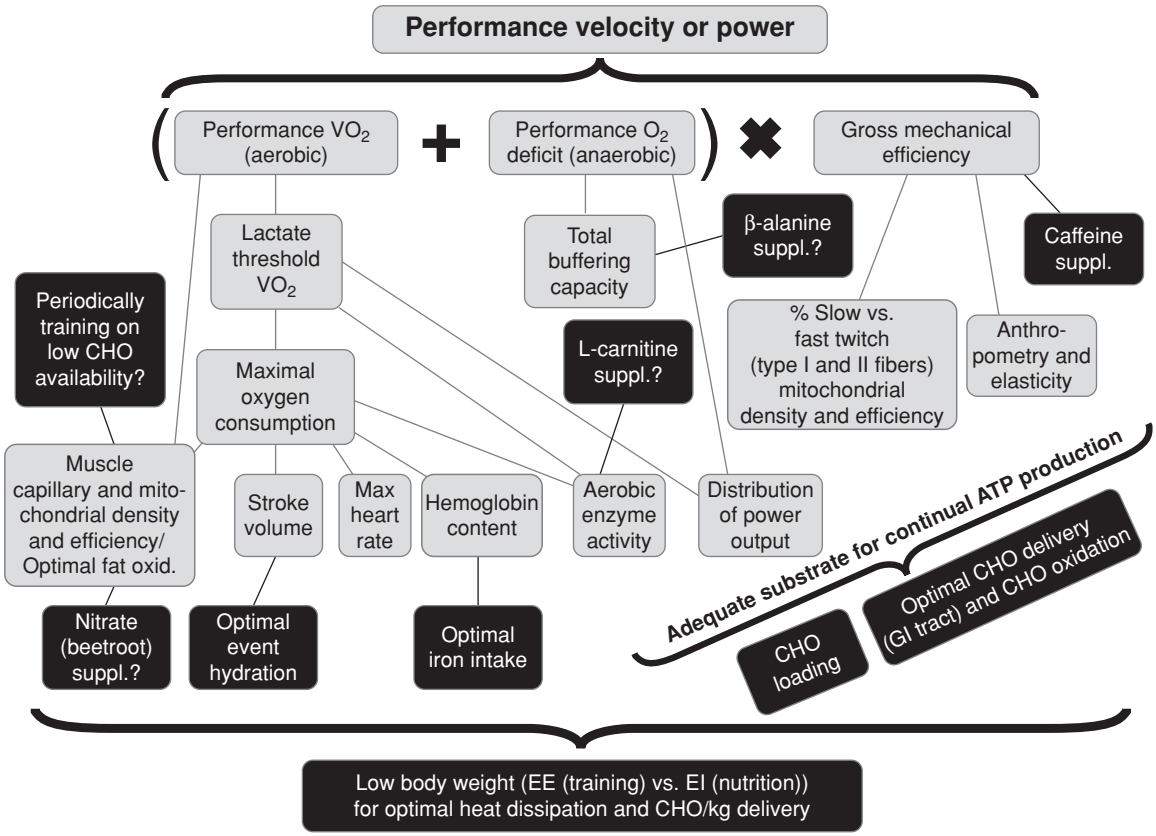

Figure 47.1 Schematic of the determinants (physiological and nutritional) of endurance performance. ATP, adenosine triphosphate/energy; CHO, carbohydrate; EE, energy expenditure; EI, energy intake; GI, gastrointestinal; oxid., oxidation; suppl., supplementation; ? indicates an emerging nutrition intervention that requires further scientific validation. Adapted from Joyner and Coyle (2008).

gray, while the suitably linked nutritionally based intervention to support or enhance the physiology is highlighted in black. Although this chapter will not reiterate each nutritional intervention in detail, as this has been covered in previous chapters, it will outline how to implement such interventions for distance runners.

One key facet of endurance events longer than ~90 minutes in duration is that the athlete needs a highly efficient CHO oxidative capacity, which is based on a highly functional gastrointestinal tract. This optimum delivery of CHO then continually fuels the mitochondria with energy, especially when muscle glycogen becomes limiting (Coyle et al., 1986). So, despite the fact that elite marathon runners are highly adapted to use fat as a fuel, they compete at such high exercise intensities (80–90% VO_2 max), that they must also have highly adapted abilities to utilize CHO during competition. No published data exist showing the relative CHO and fat utilization rates of a well-fueled elite marathon runner at world-class marathon race pace. However, non-elite marathon runners (~2 hours 45 minutes) during an overnight fasted treadmill marathon were found to have an average respiratory exchange ratio of 0.99 ± 0.01, which is ~96% dependence on CHO as a fuel (O'Brien et al., 1993). Furthermore, oxidizing CHO during exercise is energetically more efficient, as one produces 5 kcal of energy per liter of O_2 consumed compared to

4.7 kcal per liter of O_2 from fat. This has led some to hypothesize that elite marathon runners who consume exogenous CHO (i.e., sports drinks) may potentially complete the entire marathon using almost exclusively CHO as fuel (Spriet, 2007). Contrary to the widely held belief that marathon runners primarily use fat as a fuel, CHO-derived energy stores (e.g., CHO glycogen loading) and exogenous CHO supplementation are of paramount importance in optimizing performance in the endurance athlete (Figure 47.1).

Interestingly, as highlighted in Figure 47.2, there has been a steady decrease in body weight (BW) of the marathon world record holders over the last 50 years. There certainly appear to be several physiological advantages for an endurance athlete to have a low BW. Not only is this an advantage for optimum power-to-weight ratio, resulting in decreased VO_2 demand, but it also results in a better BW to surface area ratio to reduce thermoregulatory strain caused by the elevated metabolic heat production during prolonged endurance exercise, especially in hot and humid weather conditions (Marino et al., 2000).

Further to this, Jeukendrup has recently shown that there is no correlation between BW and exogenous carbohydrate oxidation among 63 subjects of varying mass (Jeukendrup, 2010), as some of the lightest subjects (~60 kg) had the same ability to absorb and oxidize exogenous CHO as some of the heaviest subjects (>90 kg). Thus, a low BW results in a physiological advantage of providing more exogenous CHO per kg BW during an endurance race. Accordingly, the lightest world record holder in the last 50 years at 56 kg is able to oxidize ~20% more CHO per kg BW compared with the heaviest world record holder at 70 kg, with a given exogenous CHO rate of 1 g/min (1.07 vs. 0.86 g CHO/min/kg BW). Finally, the overriding balance dictating an athlete's BW is the complex interaction between energy expenditure (training, basal metabolic rate) and energy intake (nutrition; Figure 47.1). An approach that addresses both training and nutrition will result in an optimally adapted phenotype, which when hydrated and fueled will produce the best endurance performance possible for an individual.

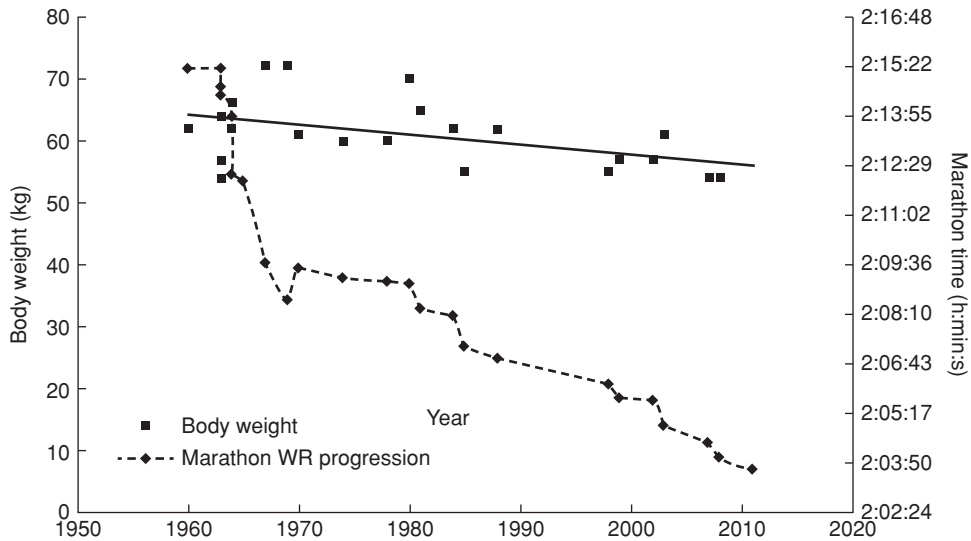

Figure 47.2 Comparison of the men's marathon world record progression and the respective body weight of each world record holder over the last 50 years.

General Daily Nutrition Recommendations

Modern training programs feature distinctly planned and periodized training approaches, with purposefully focused training blocks or periods so that an athlete can reach a desired physiologically readiness for optimum on-demand targeted performances. The current chapter will focus on the two most distinct training periods for endurance runners: "general preparation" phase and "competition" phase. For the reader further interested in nutritional periodization, refer to Stellingwerff et al. (2011, 2012). Regardless of the type of periodization approach, the exercise training stimuli during these different phases can differ drastically in terms of intensity, volume, and duration, and therefore, so do the types of fuels (CHO vs. fat) and the amount of energy that are used to generate the required ATP during training and competition. For more information on energy balance and daily CHO and protein requirements for athletes, see Chapters 5, 7, and 10.

General Preparation Phase

During the "general preparation" phase, elite athletes undertake large volumes of aerobic training at relatively low intensities (~50 to 75% VO_2 max), in which fat can be the dominant fuel. However, large amounts of CHO are also oxidized at exercise intensities approaching the onset of blood lactate accumulation. The primary purpose of these large volumes of aerobic conditioning is to improve muscle oxidative capacity through the proliferation of skeletal muscle mitochondrial and capillary density. Most elite marathon runners cover more than 200 km/week in, with some approaching upward of 260–280 km/week of running; there are documented cases of elite runners who have covered far greater distances for sustained training periods. This running volume, even for a typically small marathon runner (male 60 kg; Figure 47.2), results in an extra 1700–2300 kcal/day of required energy intake, beyond energy required for basal metabolic rate.

Table 47.1 provides an overview of reported dietary energy and macronutrient intakes from elite to international level male and female distance runners across five different countries. On average, male endurance athletes report consuming 7.7 g CHO/kg BW/day, which is within the recommended range (Burke et al., 2011), while females report a lower intake of 6.1 g CHO/kg BW/day. This gender difference in reported CHO intake is also represented in reported energy intake, even when corrected for BW (214 vs. 191 kJ/kg for males and females, respectively). Some contend that a greater emphasis needs to be made to help females meet their recommended CHO and energy intake needs. However, very well controlled studies are needed to confirm if this gender discrepancy is truly due to actual dietary CHO and energy underconsumption, or just due to dietary intake underreporting, or to a lowered training load in females as compared to males. Interestingly, several dietary intake studies have shown that the self-selected daily CHO intake of world class African runners is over 70% of total energy and over 10 g CHO/kg BW/day, which appears significantly greater than that of any other athletes (Table 47.1; Fudge et al., 2006; Onywera et al., 2004). Whether the large daily CHO intakes of East African runners helps to explain their massive training volumes resulting in marathon success, or whether it is representative of their normal cultural dietary intakes, remains to be confirmed. Nevertheless, to maintain immune function, recover glycogen, and reduce overreaching (Achten et al., 2004), a habitually high CHO diet (7–10 g CHO/kg BW/day) is recommended (Burke et al., 2011).

In 1967, Bergstrom et al. were the first to show that a high CHO diet leads to augmented glycogen stores translating into an increased time to exhaustion, as compared to a low CHO diet (Bergstrom et al., 1967). Conversely, low CHO diets (3–15% CHO) have uniformly shown impaired performance in high-intensity and endurance-based exercises (Burke et al., 2011). Consequently, recommendations have been made to endurance athletes to eat a diet chronically high in CHO, which will enable longer and harder training sessions to optimize the training adaptation. However, whether a high CHO diet (60–70% of total energy) provides increased performance benefits over a moderate CHO diet (50–55% of total energy) remains to be demonstrated

Table 47.1 Reported dietary energy and macronutrient intakes from elite to international level male and female distance runners across five different countries

				CHO			PRO			FAT	
Study	Subjects	Energy (MJ)	kJ/kg	g	g/kg	%E	g	g/kg	%E	g	%E
Males											
van Erp-Baart et al. (1989)	Dutch international level runners ($n = 56$)	13.4	193	417	6.1	50	116	1.7	14	122	34
Burke et al. (1991)	Australian national level marathoners ($n = 19$)	14.9	230	487	7.6	52	128	2.0	15	128	32
Ludbrook and Clark (1992)	Australian well-trained distance runners ($n = 12$)	14.6	211	482	7.0	54	112	1.6	13	120	30
Grandjean and Ruud (1994)	US Olympic team ($n = 11$)	13.1	189	420	6.1	53	124	1.8	16	106	29
Sugiura et al. (1999)	Japanese national team ($n = 8$)	14.3	229	382	7.1	52	139	2.3	15	131	32
Onywera et al. (2004)	Kenyan elite runners ($n = 10$)	12.5	212	607	10.4	76	75	1.3	10	46	13
Fudge et al. (2006)	Kenyan elite runners ($n = 10$)	13.2	236	549	9.8	67	123	2.2	15	61	17
	Mean	**13.7**	**214**	**478**	**7.7**	**58**	**117**	**1.8**	**14**	**102**	**27**
	SD	**0.9**	**18**	**80**	**1.7**	**10**	**20**	**0.4**	**2**	**34**	**8**
Females											
van Erp-Baart et al. (1989)	Dutch international level runners ($n = 18$)	8.8	168	301	5.8	50	82	1.6	15	68	29
Ludbrook and Clark (1992)	Australian well-trained distance runners ($n = 11$)	8.9	174	299	5.9	57	73	1.4	14	63	26
Grandjean and Ruud (1994)	US Olympic team ($n = 9$)	9.0	176	275	5.4	50	82	1.6	15	74	30
Sugiura et al. (1999)	Japanese national team ($n = 7$)	11.4	244	337	7.2	51	109	2.4	16	98	33
	Mean	**9.5**	**191**	**303**	**6.1**	**52**	**87**	**1.8**	**15**	**76**	**30**
	SD	**1.3**	**36**	**26**	**0.8**	**3**	**16**	**0.4**	**1**	**16**	**3**

Source: Data adapted from van Erp-Baart et al. (1989), Ludbrook and Clark (1992), Grandjean and Ruud (1994), Sugiura et al. (1999), Burke et al. (1991), Onywera et al. (2004), and Fudge et al. (2006).

CHO, carbohydrate; g, grams; kg, kilograms; kJ, kilojoule; MJ, megajoule; PRO, protein; %E, percent of total daily energy intake; SD, standard deviation.

(Burke et al., 2011). Furthermore, emerging scientific evidence suggests the possibility of conducting some training in either a low-energy or glycogen-depleted state to induce greater endurance training adaptations (Hawley & Burke, 2010). This concept is not necessarily new, as anecdotal reports from professional cyclists and East African distance runners indicate that some of these athletes purposely and periodically undertake training in a glycogen-depleted state, or a fasted/water-only state, in an attempt to "force" the muscle to adapt to the next level.

Several studies have demonstrated enhanced aerobic training adaptations with low CHO availability training (either low muscle glycogen or low exogenous CHO availability via overnight fasted training), which include elevations in resting muscle glycogen content (Nybo et al., 2009), increased maximum activity of several key β-oxidation pathway enzymes (De Bock et al., 2005; Nybo et al., 2009; Yeo et al., 2008), elevated skeletal muscle fatty acid protein transporters (De Bock et al., 2005), and higher rates of whole-body fat oxidation (Yeo et al., 2008). However, contrary to Hansen et al. (2005), a significantly greater posttraining performance enhancement with low-energy training, as compared to training with ample energy availability, has not consistently been shown (Nybo et al., 2009; Yeo et al., 2008).

Daily intake protein recommendations for endurance athletes have been set at 1.5–1.7 g/kg BW/day (Tarnopolsky, 1999). During endurance exercise, protein oxidation accounts for only 2–5% of total energy expenditure, but this proportion of amino acid oxidation can increase up to 10% when training at higher intensities, during longer exercise durations, or when muscle glycogen stores are depleted (Tarnopolsky, 1999). Interestingly, during the Tour de France, athletes self-select more than 2.5 g protein/kg BW/day (Saris et al., 1989). Considering the amount of amino acid oxidation occurring during prolonged intense endurance exercise, this high dietary protein intake might be needed to stay in nitrogen balance during these extreme conditions. In support of this, dietary studies in endurance athletes from Western countries have consistently shown that athletes generally consume more protein than any elevated dietary recommendation (Tarnopolsky, 1999), which is supported by the dietary intake data in elite runners highlighted in Table 47.1. Accordingly, more well-controlled dietary and nitrogen balance data are needed in elite endurance athletes who routinely undertake extreme training loads (e.g., >200 km/week of running) resulting in excessive amino acid oxidation to confirm if 1.5–1.7 g protein/kg BW/day will still result in nitrogen balance.

Competition Phase

Elite marathon runners tend to race marathons only once every 6–12 months. Consequently, the final preparation/competition phase (~4 to 6 weeks) is unique in that it usually features only a single peak race, not a series of high-quality races over a short period of time as middle distance athletes undertake. Elite marathon runners tend to taper for only 10–20 days in duration, which features approximately a 40% to 50% drop in training volume, with training quality maintained (Stellingwerff, 2012). Therefore, there is an associated decrease in energy expenditure, sometimes as much as 50%. Given the vital importance of glycogen to the endurance athlete's performance (Figure 47.1), a continual daily emphasis of ~7 to 10 g CHO/kg BW/day and 1.5–1.7 g protein/kg BW/day must be maintained. As outlined in Chapter 8, the most recent data show that as long as daily CHO intake is more than ~10 g CHO/kg BW/day, muscle glycogen loading can occur in as little as 24–48 hours (Bussau et al., 2002), due to the lowered training volume causing less glycogen turnover. Carbohydrate loading can result in ~2% to 3% increase in time-trial performance in events longer than 90 minutes (Hawley et al., 1997). However, due to the fact that glycogen synthesis also results in significant water storage and that well-trained athletes can store twofold more glycogen than untrained athletes (Greiwe et al., 1999), glycogen loading can result in a >2% increase in BW in some elite athletes. This increased glycogen and water storage may benefit endurance athletes in long flat races in warmer weather, especially in less weight-dependent

endurance sports (e.g., cycling). However, for some elite runners in shorter and hilly races, glycogen loading and the associated weight gain may actually be harmful to performance. Thus, even a nutrition intervention as recognized and established as carbohydrate loading should be practiced and optimized on an individual basis.

Also, as described above, a very light physique resulting in a high power-to-weight ratio is needed for marathon success (Figures 47.1 and 47.2). Accordingly, unlike the general preparation phase where some energy-dense foods/liquids are required to help maintain BW during high training loads, during the competition phase, fat intake should be reduced to ~1 to 1.5 g /kg BW/ day. Interestingly, the only dietary intake studies with elite runners reporting dietary fat intake at this level are the two studies in elite Kenyan runners (Table 47.1) (Fudge et al., 2006; Onywera et al., 2004). Finally, it has been shown that *ad libitum* energy intake does not immediately match the reduced energy expenditure that comes with decreased training volumes during the taper in the competition phase (Margaritis et al., 2003). Therefore, athletes need to make conscious decisions about limiting their total energy intake during this phase to maintain an ideal body composition, which needs to be closely monitored. Like training, body composition should be periodized over the training year and athletes should aim to be at competition performance BW and composition for only short periods of time throughout the year. Some athletes aspire to be at competition weight year-round by not taking in enough energy, but this approach can be both physically and emotionally difficult and can lead to risk of injury, sickness, and health issues.

This final preparation phase prior to a targeted event can also be ideal for practicing and optimizing fluid and CHO intake. The 2007 American College of Sports Medicine (ACSM) position stand on fluid intake illustrates the need for making individualized fluid and CHO intake recommendations according to individual sweat rates and individual gastrointestinal tolerances (Sawka et al., 2007). Table 47.2 outlines a worksheet recording tool developed to collect field data on a 2:10 male marathon runner during several key workouts in the final preparation phase. This worksheet recording tool allows for the assessment of subtle, but important, changes required to optimize fluid and CHO intake, while considering individual sweat rates and gastrointestinal tolerances.

Optimizing Nutrition for Recovery

Most runners compete only 0.5% of total time per year and spend only 7–10% of their total time training. This leaves some 90–95% of their yearly total time (~500,000 minutes) for "recovering," which includes eating, sleeping, and all other lifestyle factors that might enhance or detract from optimum adaptation. Furthermore, even with the best training stimulus in the world, without the proper substrates (energy, macronutrients, hydration), an athlete will never adapt to the desired level. The majority of the concepts for optimizing recovery are covered in Chapters 7, 8, 10, 11, and 16. However, given the importance of recovery, some of the key concepts will be overviewed, with emphasis on fluids for hydration and CHO and protein for glycogen and protein synthesis, respectively.

Glycogen Resynthesis

Stored muscle glycogen is the primary fuel source to produce energy for runners. Therefore, glycogen is of utmost importance with regard to optimizing its resynthesis. The highest rates of muscle glycogen synthesis occur during the first hour following exercise. When the time for glycogen recovery is very short (<4 hours), such as between training bouts, studies suggest utilizing immediate and frequent smaller high glycemic CHO doses for an overall intake rate of 1.2–1.5 g CHO/kg BW/h for the first several hours of recovery (Jentjens & Jeukendrup, 2003). During short-term recovery (<4 hours), high levels of fat and protein intake should also be avoided as they delay gastric emptying and can also potentially cause gastrointestinal upset for the subsequent exercise bout. Furthermore, the immediate postexercise consumption of CHO intake is very important, as delaying the intake of CHO

Table 47.2 Example of individualized sweat, fluid, and carbohydrate intake worksheet for a 2:10 marathon runner

Date	Temp (°C)	Humid. (%)	Prerun weight (kg)	Postrun weight (kg)	Ingest. fluids (l/h)	Total run time (h)	Sweat rate (l/h)	% Body weight loss	Ingest. CHO (g/h)	% CHO solution	Comments (feeling, effort, GI effects, etc.)
May 1st	14	94	60.5	59.5	0.000	1.05	0.952	1.7	0.0	0.0	16.7 km; fasted run—felt fine
May 3rd	15	46	61.7	59.3	0.900	2.00	1.650	3.9	27.0	3.0	33.3 km; 1 gel with 900 ml of water + 160 mg of caffeine
May 8th	15	82	60.5	59.3	0.000	1.33	0.902	2.0	0.0	0.0	20 km; fasted run—sunny and humid
May 10th	7	70	61.6	60.4	0.800	1.66	1.205	1.9	42.0	5.3	27 km; cool day and did not overdress—stomach was fine
May 12th	10	62	61.1	60.2	0.150	1.25	0.840	1.5	0.0	0.0	19 km; cool day—fasted run (last one before race)
May 15th	13	67	61.1	59.8	0.500	1.27	1.417	2.1	42.0	8.4	18 km; 1 gel + 250 ml at 7 km and 12.5 km—felt fine
May 17th	6	60	60.5	59.5	0.700	1.30	1.308	1.7	44.0	6.3	22 km; 1/3 gel + 100 ml every 3 km—slight side stitch
May 19th	18	75	61.4	59.2	0.500	2.35	1.149	3.6	40.0	8.0	39 km; 95 g CHO, with 6 g CHO in second hour, gut fine

CHO, carbohydrates; GI, gastrointestinal; Humid., humidity; Ingest., ingested; Temp., temperature.
Sweat rate calculated as ((prerun weight – postrun weight) + ingested fluids (liter))/total run time (hour).

until 2 hours after exercise has a significant negative effect on muscle glycogen resynthesis for up to 8 hours.

Protein Synthesis

Beyond current daily protein recommendations, strong emerging evidence exists to suggest that the timing and type of protein throughout the day has a significant effect on protein synthesis, optimization of postexercise recovery, and, ultimately, body composition. It appears that an essential element in optimizing protein synthesis is the timing, amount, and quality of dietary protein intake, with the most recent evidence showing that for optimizing muscle protein synthesis, it appears that whey protein is superior to either soy or casein (Tang et al., 2009).

A recent study has clearly shown that there appears to be a maximum effective dose of ~20 g of dietary protein for stimulating muscle anabolism after resistance exercise (Moore et al., 2009). Although no studies have yet clearly established whether more or less protein is needed to optimize acute protein synthesis for athletes of varying BWs, when examining the totality of acute studies, a BW-corrected protein dose of ~0.3 g/kg BW appears to be optimal. It should be clarified that this is the optimum acute dose and that most athletes would need four to six of these acute doses spread throughout the day, resulting in about 1.5 g PRO/kg BW/day, to maximize net protein synthesis. Finally, protein and CHO should be ingested in close proximity to the exercise bout (e.g., immediately after training) to maximize the anabolic response to training (Chapter 11).

Hydration Recovery

Fluid intake is the third requirement for optimum recovery (Chapter 16). During training or competition, athletes should generally aim to consume sufficient fluid to limit body mass loss to about 2% of pre-exercise mass, but athletes should track sweat rates and learn to individualize fluid intakes (Sawka et al., 2007). For optimum rehydration, it is recommended to consume 1.5× the net body mass loss, due to ongoing urinary losses of liquids during recovery.

Ergogenic Aids for Distance Runners

Fluid and Carbohydrate Intake for Optimum Performance

Beyond training and genetic status, perhaps the largest single determinant of performance during prolonged (>2 hours) endurance events is through the consumption of a CHO-based sports drink providing CHO energy, fluids, and electrolytes (Figure 47.1; Chapter 9). It is clear that most studies show an improvement in endurance performance or capacity when subjects consume CHO drinks as compared to water alone. However, some inconsistencies do exist in performance outcomes, most likely due to differences in subjects' fitness, the type of exercise (cycling vs. running), the amount and type of CHO supplemented, whether the subjects were fasted, or whether the study was properly powered to show a performance difference. A recent study has also shown a dose–response relationship, showing improved laboratory endurance performances in a dose-dependent manner, with 60 g CHO/h resulting in better performance than either 15 or 30 g CHO/h (Smith et al., 2010), suggesting more CHO is better for performance. It appears that the rate-limiting step to exogenous carbohydrate oxidation is at the level of the gastrointestinal tract, due to the intestinal CHO transport mechanisms, and specifically the SGLT1 transporter for glucose and the GLUT5 transporter for fructose (Jeukendrup, 2010). In line with this, an entire series of studies has clearly demonstrated that glucose and fructose CHO blends (multitransportable CHOs) are oxidized at rates ~20% to 50% higher than isoenergetic glucose-only CHO drinks, which results in significant performance benefits when exercise lasts more than 2 hours (Jeukendrup, 2010).

Table 47.3 highlights CHO, fluid, and caffeine intake data collected at a recent marathon in three different elite male runners (Figure 47.3). Each of these marathon runners undertook premarathon fuel and fluid practice testing (Table 47.2) and had settled upon an individual race intake profile that was optimum for their gastrointestinal and performance. It is important to note the variability of CHO and fluid intake amounts with the three marathon runners, despite running in the exact same race and weather conditions.

Optimizing Gastrointestinal Tolerance to Maximize Performance

Approximately 15–20% of all athletes experience adverse gastrointestinal effects during compe-

Table 47.3 Actual marathon carbohydrate, fluid, and caffeine intakes from three elite runners

Athlete	Total CHO intake (g)	CHO (g/h)	Total fluid intake (ml)	Fluid (ml/h)	% CHO solution	Total CAFF	CAFF/kg BW	Number of aid stations	CHO/aid station	Fluid/aid station
2:10:55 Marathoner	98	44.8	930	425	10.4	350	5.7	8	12.3	116
2:11:27 Marathoner	170	77.6	1525	696	11.1	300	4.9	8	21.2	190
2:12:56 Marathoner	137	61.8	1205	550	11.4	200	2.9	8	17.1	151
Mean	**135**	**61.4**	**1220**	**557**	**11.0**	**283**	**4.5**	**8**	**16.9**	**152**
SD	**36**	**16.4**	**298**	**136**	**0.5**	**76**	**1.4**	**0**	**4.5**	**37**

BW, body weight; CAFF, caffeine; CHO, carbohydrate; SD, standard deviation.
All data collected during the same race (Toronto Marathon; October 16th, 2011); 10–15°C, 20–35 km/h winds.

Figure 47.3 A race walker picking up their bottle from an in race aid station. © 2012 IOC/Christopher Furlong.

tition; these are more frequent in running events than in cycling, and are strongly correlated to a history of gastrointestinal problems (Pfeiffer et al., 2009, 2011). It is important to note that even minor gastrointestinal distress is associated with negative endurance performance outcomes (Thorburn et al., 2006). However, anecdotal reports support the idea that an individual's gastrointestinal tract can be trained and adapted to handle the intake of large amounts of CHO and fluid during exercise, although the tolerable amount appears to be very individual (Jeukendrup, 2010). Furthermore, it has been suggested that gastrointestinal tolerance to high fluid intakes can improve in as little as five training bouts (Lambert et al., 2008). As described above, utilizing a multitransportable CHO mixture can enhance both gastric emptying and CHO delivery and oxidation (Jeukendrup, 2010), which could theoretically also have a favorable effect on gastrointestinal comfort. Table 47.4 outlines several practical recommendations that can be made in athletes who consistently have gastrointestinal issues during endurance exercise. The recommendations are purposefully ordered from less important training situations to more important competition settings and from noninvasive to more invasive practices.

Table 47.4 Practical recommendations to minimize gastrointestinal discomfort during endurance exercise in problematic athletes

- Continually practice with fluid and CHO habituation in training sessions, and specifically sessions at goal race pace and in projected race weather conditions.
- Continually experiment with different fluid volumes and CHO intake rates (thus altered % CHO solutions) to find an optimal individual solution. Start with increasing fluid volumes and then try increasing CHO concentration.
- Use of glucose:fructose CHO blends rather than single CHO sources.
- Alter the acute rate of intake (e.g., spread a 250 ml drink over several minutes) and also experiment with higher intake rates early in a race when negative GI side effects tend to be lower.
- Practice in less important races to try and identify "outside" contributing factors (e.g., travel effects, stress/nerves).
- Experiment with different bottled water brands both chronically (throughout the day) and during exercise (to use with CHO gels & powders), to minimize effects of different electrolytes and other purification ingredients—this is especially important to consider when racing in different countries.
- Use of CHO mouth washing to maximize performance outcomes while minimizing GI discomfort—this can be especially beneficial in the late stages of races when GI discomfort tends to be the worse.
- Exploration and treatment of pre-existing gut intolerances—check for FODMAPS (intolerance to fructose and other oligo/mono/poly-alcohols), check for lactose intolerance, celiac disease, or other gluten intolerances. Specific outcomes may then require a uniquely made CHO drink that removes any offending CHOs. (Note: Some athletes may have subclinical or background GI issues that only really become evident under physical or mental stress).
- Prophylactic use of antidiarrheal medication and other medications altering gut motility.
- Utilization of psych counseling to reduce the nerves/stress and sympathetic hyperactivity associated with very important races. In extreme cases, use of antianxiety medications might be considered.
- Acceptance that an individually maximal CHO and fluid intake without problems is better than trying to be more aggressive and ending up with problems.

CHO, carbohydrate; FODMAPS, fermentable oligo-, di- and monosaccharides, and polyols; GI, gastrointestinal.

Caffeine

There is probably no ergogenic aid more researched than caffeine, and sound evidence shows that caffeine intake enhances endurance performance by a small, but worthwhile, amount. Current caffeine intake recommendations for athletes are ~3 to 6 mg caffeine/kg BW, consumed ~90 minutes prior to exercise (see Chapter 25), but worthwhile performance benefits have been found with caffeine doses as low as 1–3 mg/kg BW (Cox et al., 2002). Depending on the length of the race, some athletes will break up caffeine doses into ~1 to 2 mg/kg BW/doses and consume serially every 60–120 minutes of racing, to ensure peak plasma caffeine levels throughout. Table 47.3 shows the individual variability of caffeine dosing used between three elite marathon runners.

Conclusions

Given the duration and metabolic demands of marathon racing, there are many potential nutritional interventions that may provide a strategic performance advantage (Figure 47.1). This chapter took an individualized and periodized approach to much of the basic science recommendations presented earlier in this book, along with presenting information on several nutritional ergogenic aids relevant to distance running performance. Optimizing nutrition before, during, and after distance running is fundamental to competition success. In fact, the longer the exercise challenge the greater the potential for sensible nutritional and physiological interventions to have a positive performance impact.

References

Achten, J., Halson, S.L., Moseley, L., et al. (2004) Higher dietary carbohydrate content during intensified running training results in better maintenance of performance and mood state. *Journal of Applied Physiology* 96, 1331–1340.

Bergstrom, J., Hermansen, L., Hultman, E., et al. (1967) Diet, muscle glycogen and physical performance. *Acta Physiologica Scandinavica* 71, 140–150.

Burke, L.M., Gollan, R.A., & Read, R.S. (1991) Dietary intakes and food use of groups of elite Australian male athletes. *International Journal of Sport Nutrition* 1, 378–394.

Burke, L.M., Hawley, J.A., Wong, S.H., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Sciences* 29, 1–11.

Bussau, V.A., Fairchild, T.J., Rao, A., et al. (2002) Carbohydrate loading in human muscle: an improved 1 day protocol. *European Journal of Applied Physiology* 87, 290–295.

Cox, G.R., Desbrow, B., Montgomery, P.G., et al. (2002) Effect of different protocols of caffeine intake on metabolism and endurance performance. *Journal of Applied Physiology* 93, 990–999.

Coyle, E.F., Coggan, A.R., Hemmert, M.K., et al. (1986) Muscle glycogen utilization during prolonged strenuous exercise when fed carbohydrate. *Journal of Applied Physiology* 61, 165–172.

De Bock, K., Richter, E.A., Russell, A.P., et al. (2005) Exercise in the fasted state facilitates fibre type-specific intramyocellular lipid breakdown and stimulates glycogen resynthesis in humans. *Journal of Physiology* 564, 649–660.

Fudge, B.W., Westerterp, K.R., Kiplamai, F.K., et al. (2006) Evidence of negative energy balance using doubly labelled water in elite Kenyan endurance runners prior to competition. *British Journal of Nutrition* 95, 59–66.

Grandjean, A.C. & Ruud, J.S. (1994) Energy intake of athletes. In *Oxford Textbook of Sports Medicine* (eds. M. Harries C. Williams, W.D. Stanish, et al.). Oxford University Press, New York.

Greiwe, J.S., Hickner, R.C., Hansen, P.A., et al. (1999) Effects of endurance exercise training on muscle glycogen accumulation in humans. *Journal of Applied Physiology* 87, 222–226.

Hansen, A.K., Fischer, C.P., Plomgaard, P., et al. (2005) Skeletal muscle adaptation: training twice every second day vs. training once daily. *Journal of Applied Physiology* 98, 93–99.

Hawley, J.A. & Burke, L.M. (2010) Carbohydrate availability and training adaptation: effects on cell metabolism. *Exercise and Sport Sciences Reviews* 38, 152–160.

Hawley, J.A., Schabort, E.J., Noakes, T.D., et al. (1997) Carbohydrate-loading and exercise performance. An update. *Sports Medicine* 24, 73–81.

Jentjens, R. & Jeukendrup, A. (2003) Determinants of post-exercise glycogen synthesis during short-term recovery. *Sports Medicine* 33, 117–144.

Jeukendrup, A.E. (2010) Carbohydrate and exercise performance: the role of multiple transportable carbohydrates. *Current Opinion in Clinical Nutrition and Metabolic Care* 13, 452–457.

Jeukendrup, A.E. (2011) Nutrition for endurance sports: marathon, triathlon, and road cycling. *Journal of Sports Sciences* 29, S91–S99.

Joyner, M.J. & Coyle, E.F. (2008) Endurance exercise performance: the physiology of champions. *Journal of Physiology* 586, 35–44.

Joyner, M.J., Ruiz, J.R., & Lucia, A. (2011) The two-hour marathon: who and when? *Journal of Applied Physiology* 110, 275–277.

Lambert, G.P., Lang, J., Bull, A., et al. (2008) Fluid tolerance while running: effect of repeated trials. *International Journal of Sports Medicine* 29, 878–882.

Ludbrook, C. & Clark, D. (1992) Energy expenditure and nutrient intake in long-distance runners. *Nutrition Research* 12, 689–699.

Margaritis, I., Palazzetti, S., Rousseau, A.S., et al. (2003) Antioxidant supplementation and tapering exercise improve exercise-induced antioxidant response. *Journal of the American College of Nutrition* 22, 147–156.

Marino, F.E., Mbambo, Z., Kortekaas, E., et al. (2000) Advantages of smaller body mass during distance running in warm, humid environments. *Pflügers Archiv: European Journal of Physiology* 441, 359–367.

Moore, D.R., Robinson, M.J., Fry, J.L., et al. (2009) Ingested protein dose response of muscle and albumin protein synthesis after resistance exercise in young men. *American Journal of Clinical Nutrition* 89, 161–168.

Nybo, L., Pedersen, K., Christensen, B., et al. (2009) Impact of carbohydrate supplementation during endurance training on glycogen storage and performance. *Acta Physiologica (Oxford)* 197, 117–127.

O'Brien, M.J., Viguie, C.A., Mazzeo, R.S., et al. (1993) Carbohydrate dependence during marathon running. *Medicine & Science in Sports & Exercise* 25, 1009–1017.

Onywera, V.O., Kiplamai, F.K., Boit, M.K., et al. (2004) Food and macronutrient intake of elite Kenyan distance runners. *International Journal of Sport Nutrition and Exercise Metabolism* 14, 709–719.

Pfeiffer, B., Cotterill, A., Grathwohl, D., et al. (2009) The effect of carbohydrate gels on gastrointestinal tolerance during a 16-km run. *International Journal of Sport Nutrition and Exercise Metabolism* 19, 485–503.

Pfeiffer, B., Stellingwerff, T., Hodgson, A.B., et al. (2011) Nutritional intake and gastrointestinal problems during competitive endurance events. *Medicine & Science in Sports & Exercise* 44, 344–351.

Saris, W.H., Van Erp-Baart, M.A., Brouns, F., et al. (1989) Study on food intake and energy expenditure during extreme sustained exercise: the Tour de France. *International Journal of Sports Medicine* 10(Suppl. 1), S26–S31.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.

Smith, J.W., Zachwieja, J.J., Peronnet, F., et al. (2010) Fuel selection and cycling endurance performance with ingestion of [13C]glucose: evidence for a carbohydrate dose response. *Journal of Applied Physiology* 108, 1520–1529.

Spriet, L.L. (2007) Regulation of substrate use during the marathon. *Sports Medicine* 37, 332–336.

Stellingwerff, T. (2012) Case study: nutrition and training periodization in three elite marathon runners. *International journal of sport nutrition and exercise metabolism*, 22(5), 392--400.

Stellingwerff, T. & Jeukendrup, A.E. (2011) Don't forget the gut—it is an important athletic organ! *Journal of Applied Physiology* 110, 278; discussion 294.

Stellingwerff, T., Maughan, R.J., & Burke, L.M. (2011) Nutrition for power sports: middle-distance running, track cycling, rowing, canoeing/kayaking, and swimming. *Journal of Sports Sciences* 29(Suppl. 1), S79–S89.

Sugiura, K., Suzuki, I., & Kobayashi, K. (1999) Nutritional intake of elite Japanese track-and-field athletes. *International Journal of Sport Nutrition* 9, 202–212.

Tang, J.E., Moore, D.R., Kujbida, G.W., et al. (2009) Ingestion of whey hydrolysate, casein, or soy protein isolate: effects on mixed muscle protein synthesis at rest and following resistance exercise in young men. *Journal of Applied Physiology* 107, 987–992.

Tarnopolsky, M.A. (1999) Protein and physical performance. *Current Opinion In Clinical Nutrition and Metabolic Care* 2, 533–537.

Thorburn, M.S., Vistisen, B., Thorp, R.M., et al. (2006) Attenuated gastric distress but no benefit to performance with adaptation to octanoate-rich esterified oils in well-trained male cyclists. *Journal of Applied Physiology* 101, 1733–1743.

van Erp-Baart, A.M., Saris, W.H., Binkhorst, R.A., et al. (1989) Nationwide survey on nutritional habits in elite athletes. Part I. Energy, carbohydrate, protein, and fat intake. *International Journal of Sports Medicine* 10(Suppl. 1), S3–S10.

Yeo, W.K., Paton, C.D., Garnham, A.P., et al.(2008) Skeletal muscle adaptation and performance responses to once a day versus twice every second day endurance training regimens. *Journal of Applied Physiology* 105, 1462–1470.

Chapter 48

Cycling

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The Olympic Cycling Disciplines

International cycling involves eight disciplines (road, track, mountain bike, cyclocross, BMX, trial, indoor, para-cycling; see www.uci.ch), including a total of 30 different events. Determinants of performance are very different between the events, of course. In some events, maximum power output is pivotal to success (e.g., track sprint), while in others endurance is of primary importance (e.g., road races and mountain biking). Hence nutrition strategies also vary considerably between the events. This chapter will primarily focus on providing nutritional recommendations for the Olympic cycling events, as summarized in Table 48.1. However, because the "big tours" and particularly Tour de France are most popular in cycling, it also addresses some specific nutrition issues related to stage races. The chapter also focuses on practical issues, rather than on the theoretical backgrounds which are extensively covered in other chapters.

Preparing for Competition

Muscle Glycogen Loading

It has been known for more than 50 years that muscle glycogen availability is a primary determinant of endurance performance, at least in exercise exceeding about 60 minutes. With regard to the Olympic cycling events, this means that an explicit glycogen loading regimen during the 2–4 days prior to competition would make sense only for the individual road race and for cross-country mountain biking. For all other events (BMX, individual time trials on the road, all track events), a normal muscle glycogen content is sufficient to perform. Thus, no efforts need to be made to increase dietary carbohydrate load. As a rule, mountain bikers and road cyclists do a last intensive training session on the competition circuit on day 3 or 4 before the event, after which full tapering starts. Probably the most comfortable strategy to establish supercompensated muscle glycogen levels, while avoiding dietary stress and gastrointestinal (GI) problems, is to use a carbohydrate-rich diet supplying about 8–10 g carbohydrates/kg BW for 24 hours after this last training session. This procedure starts by using carbohydrate-rich drinks and snacks supplying carbohydrate at a rate of about 1.0–1.2 g/kg/h during the first 4 hours after the last training session, then consuming carbohydrate-rich meals at dinner and at the next day's breakfast and lunch. Thereafter, returning to a moderate carbohydrate diet (about 5 g/kg/day) along with tapered training and rest will maintain the supercompensated muscle glycogen levels until the day of competition (Burke et al., 2011; Goforth et al., 1997; Jeukendrup, 2011). In contrast to earlier opinions based on relatively sedentary subjects (Bergström et al., 1967), carbohydrate loading need not be preceded by 2–3 days of high-fat/protein diet in conjunction with high-intensity training to deplete glycogen stores. Such a strategy

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Table 48.1 Overview of Olympic cycling events (London 2012)

	Race duration	Competition schedule
BMX		
Supercross	~35 s	1 day, multiple heats
Road		
Individual road race	~3 h 30 min (♀)/ ~6 h (♂)	1 day, single race
Individual time trials	~30 min (♀)/ ~60 min (♂)	1 day, single race
Track		
Sprint	~10 to 12 s	1 day, multiple heats
Team sprint	~10 to 12 s	1 day, multiple heats
Keirin[a]	~2 min	1 day, multiple heats
Team pursuit	~3 to 4 min	2 days, multiple heats
Omnium	~20 s to ~30 min	2 days, multiple events
Mountain bike		
Cross-country	~2 h	1 day, single race

[a]Only in men's competition.

would increase the incidence of GI discomfort while stimulating perception of fatigue in the direct approach to the event, without beneficially affecting muscle glycogen content.

Pre-competition Meal

For events lasting more than 60 minutes, ingestion of a carbohydrate-rich meal after an overnight fast is likely to produce a slight increase in muscle glycogen content. In addition, such a meal is important to replenish the liver glycogen store and to provide an extra intestinal pool of absorbable glucose to support glucoregulation and blood glucose oxidation during the subsequent endurance exercise bout (Coyle et al., 1985 and see Chapters 7–9). This would make sense to improve performance only in events lasting more than about 60 minutes, i.e., the Olympic individual road races as well as the cross-country mountain bike competitions. For these events, the pre-competition meal, which is to be consumed between 4 and 3 hours before the start, must contain substantial amounts of carbohydrates (150–300 g). Riders should make a selection of appetizing carbohydrate-rich foods, preferentially low in fat and fiber content, and sure not to cause any GI discomfort in the often stressful approach to competition. For the men's road competition starting at 10:00 this implies that a carbohydrate-rich breakfast should be taken between 06:00 and 07:00. Because meal timing plays a pivotal role in setting diurnal rhythms (Armstrong, 2000), regularly consuming breakfast at about 06:00 hours in the weeks preceding the event is recommended to synchronize diurnal rhythm with the competition schedule. Athletes should also follow their individual experience and food preferences to make sure that they can meet the aforementioned carbohydrate fuel targets.

For events shorter than 60 minutes, which includes all track competitions as well as the time trials on the road, muscle glycogen loading prior to competition is redundant because normal muscle glycogen content is ample to perform, though low glycogen levels may impair performance. The pre-competition meal here serves primarily to fill up liver glycogen to facilitate maintenance of euglycemia throughout the upcoming competition day. Furthermore, the meal must bring the athlete to the start of competition with a comfortable feeling of satiety, yet with a virtually empty stomach. Gastric emptying and blood flow to the gut are significantly decreased during high-intensity exercise. Together with the psychological stress associated with top competition, this could stimulate the development of GI discomfort in the form of gastroesophageal reflux or diarrhea. Depending on the competition schedule, cyclists will thus need to consume either one (breakfast) or two (breakfast and lunch) light meals before the start of the competition. Meals should supply energy as carbohydrates (about 100–150 g), which may or may not be complemented with small amounts of proteins (20–30 g) in the form of lean dairy products, fish, or meat. Fiber-rich foods and fat should be used only in small amounts. Again it is important here that athletes follow a well-experienced and defined nutritional plan in the selection of foods for an appetizing light pre-exercise meal.

Carbohydrate Ingestion Following the Last Pre-exercise Meal

In general, an interval of about 3 hours should separate the end of the last pre-competition meal

from the start of the competition. Athletes should certainly consider giving the stomach a 90-minute "break" after the pre-competition meal, and thus drink only water in this period. However, confusion often exists as to the relevance of carbohydrate feeding during the final 60–90 minutes before the onset of the competition. For exercise events lasting less than about 1 hour, it is clear that acute pre-exercise carbohydrate ingestion will not help to stimulate performance. A small energy snack here may only serve to "tease" the stomach and thereby prevent the development of a feeling of starvation, which is important in maintaining mental readiness to compete. Conversely, for the exercises lasting more than 60 minutes, acute pre-exercise carbohydrate ingestion may improve performance (Sherman et al., 1989; Wright et al., 1991) by further expanding the carbohydrate pool available in the gut, and thereby facilitating blood glucose absorption and oxidation during exercise. Nonetheless, based on early studies with high oral glucose loads before exercise, it has been a prevailing opinion for many years that carbohydrate ingestion must be avoided because the development of reactive hypoglycemia during the early stage of exercise may impair performance. However, more recent studies that used more realistic nutritional approaches to prepare for endurance performance have clearly indicated that reactive hypoglycemia is an unlikely event in the majority of endurance athletes. Therefore, as long as this is part of a well-experienced and successful nutritional plan, it can be recommended that athletes ingest 30–90 g of carbohydrate over the final 90 minutes before a prolonged endurance competition (Jeukendrup, 2011; Jeukendrup & Killer, 2010). Effective strategies to avoid rebound hypoglycemia include using lower doses of low rather than high-glycemic-index carbohydrates, and timing the ingestion nearer the warm-up and start of the event. Finally, athletes must also realize that the time between the pre-exercise meal and the start of competition is often quite hectic due to transport to the site of competition and sometimes unexpected traffic delays, together with limited access to preferred foods and drinks. Therefore, athletes must be prepared to carry all foods and drinks they will need to adhere to the predesigned nutrition plan.

Pre-exercise Hydration

Based on extensive research, the theoretical threshold for body water loss that athletes could afford without consistent impairment of performance is often set at about 2% of body weight for endurance events, and about 3–4% for strength and power sports (Maughan & Shirreffs, 2008; Sawka et al., 2007; Shirreffs & Sawka, 2011, and see Chapter 15). However, some athletes may experience small performance decrements at even lower levels of dehydration, especially during competition in the heat. Thus, dehydration is not an "affordable risk" and cyclists must take care to report at the start of competition in a well-hydrated state. Normal overnight fluid loss by respiration, transpiration, plus urine production during sleep easily amounts to about 1% of body weight, and net dehydration may be greater if negative body water balance already existed prior to bedtime. In addition, early morning dehydration may be exaggerated by the diuretic effect of very strong coffee or tea consumption. Therefore, cyclists must drink sufficiently between the time they wake up and the start of the competition so as to assure an adequate pre-start hydration. Dark urine, or worse, failure to void urine before the start indicates dehydration. Conversely, pale urine before the start reflects euhydration (see Chapter 14). Finally, athletes having well-established caffeine habits must be aware that acute caffeine withdrawal may induce symptoms like headache that may negatively affect performance (Hetzler et al., 1994; James & Rogers, 2005; Rogers et al., 2010). Thus, adhering to the habitual coffee/caffeine intake in the direct approach to competition is a better option than sudden withdrawal.

During Exercise

Events Lasting Less Than 60 Minutes

Eating or drinking during short maximal exercise bouts lasting seconds to minutes (most track events + BMX) is totally pointless, of course, and can only impair performance. In addition, according to UCI rules (www.uci.ch), riders should not carry any object on them or on their bicycles that could drop onto the track, not even in the longer events like the 30 km

points race. Still, confusion sometimes exists as to the relevance of carbohydrate or fluid intake during 30- to 60-minute bouts of maximal exercise, like the Olympic time trials on the road. Available literature indicates that oral carbohydrate intake during exercise with the aim of stimulating exogenous carbohydrate oxidation, makes sense only in events lasting longer than about 60 minutes. Furthermore, the effect of dehydration on endurance exercise performance is negligible as long as sweating does not cause body weight to decrease by more than about 2% of body weight. For a 70 kg cyclist, this would require an average sweat rate of more than 2.8 liters per hour throughout a 30-minute time trial (about 1.7 liters per hour for a 50-minute time trial). Thus, starting the time trial in a well-hydrated state, by drinking 300–500 ml extra fluid during the last 15 minutes prior to the start, even in heavy sweaters would sufficiently compensate for the upcoming sweat loss. However, recent data indicate that rinsing the mouth with a carbohydrate solution may significantly stimulate time trial performance, even when the solution is not swallowed. Thus, carbohydrate mouth rinse during 30- to 60-minute cycling time trial simulations yielded similar performance improvement as actual carbohydrate ingestion (Carter et al., 2004). This effect is probably explained by "ergogenic" afferent feedback to the brain originating from supposed "carbohydrate receptors" in the oral cavity (Gant et al., 2010). Although the beneficial effect of mouth rinsing on performance seems to be suppressed by consumption of a carbohydrate-rich, pre-exercise meal (Beelen et al., 2009), it is probably reasonable to suggest that each individual rider participating in endurance competitions lasting between 30 and 60 minutes should find out whether rinsing the mouth at 5- to 15-minute intervals with a small aliquot of a so-called "isotonic" sports drink (~4% to 7% carbohydrate solution) may boost performance. For the shorter events undertaken on an empty stomach, i.e., pursuit races on the track or supercross in BMX, riders may also want to test whether a short pre-exercise mouth rinse provides a positive stimulus to performance.

Events Lasting More Than 60 Minutes

Over the last 30 years, many studies have been carried out to elucidate the optimum protocols for carbohydrate and fluid intake during prolonged endurance exercise, and there is no doubt that adequate carbohydrate and fluid intake is pivotal to performance in events longer than about 75–90 minutes (Burke et al., 2011; Jeukendrup, 2011). Here we address some of the more practical issues relating to carbohydrate intake during the Olympic events lasting more than 1 hour, i.e., MTB cross-country (2 hours) and the individual road races (more than 3 hours). In this regard, an important fact to consider is that the primary factor limiting the capacity for exogenous carbohydrate oxidation during exercise is the rate of carbohydrate absorption in the gut (g/h), which seems to be independent of body weight and gender. Thus, recommendations are similar for females and males and should not take account of the body weight of the athlete. Furthermore, the capacity for glucose absorption peaks at about 60 g/h, but when fructose sources are added as an additional route of carbohydrate absorption, capacity can be elevated to about 90 g/h (Jentjens et al., 2004a, 2004b).

Against this background, the recommended rate of carbohydrate ingestion during maximal exercise of about 2 hours duration, i.e., MTB cross-country, ranges between 30 and 60 g carbohydrate/h (Burke et al., 2011; Jeukendrup, 2011, and see Chapter 9). However, given that higher dosages yield greater performance benefits (Smith et al., 2010), riders should aim to ingest about 60 g/h, and distribute this amount equally between the start and about 5–10 minutes before the finish. Because sweat rate in MTB racing easily exceeds 1 liter per hour in most athletes, the most practical way to provide the full dose of carbohydrates is to supply an "isotonic" sports drink containing 50–70 g/l (one 500 ml bottle per 30 minutes), and drink extra water if needed to maintain euhydration. A sports drink containing only glucose/maltose/maltodextrin would perfectly do the job, and some riders may even want to avoid drinks containing fructose or sucrose because this might increase the risk of developing diarrhea during the race without adding to performance (Murray et al., 1989).

The duration of the Olympic road races is about 3 hours 30 minutes (females) to about 5 hours 30 minutes (males). The recommended rate of

carbohydrate intake in endurance exercise taking longer than 3 hours is 60–90 g/h (see Chapter 9), and the higher doses are likely to yield a greater performance benefit (Burke et al., 2011; Jeukendrup, 2011; Smith et al., 2010). Here we propose a feeding schedule (Figure 48.1) where riders organize to consistently achieve a minimum carbohydrate intake of about 60 g/h, from start to finish. In addition, riders must aim at using an extra portion of 10–30 g/h, within the limits of GI tolerance. The cyclist who neglected carbohydrate intake during an earlier episode of the race should not overcompensate (more than 90 g/h) in a later phase because carbohydrate bulking in the gut (rate of carbohydrate ingestion exceeds rate of carbohydrate absorption) will increase the risk of diarrhea. Furthermore, at intakes over 60 g/h, it is important to ingest a fraction of the carbohydrates (20–30%) in the form of fructose to allow for the higher rates of intestinal carbohydrate absorption needed (Jentjens et al., 2004a, 2004b). Theoretically riders could ingest the full dose (60–90 g) in the form of carbohydrate solutions (drinks or gels), and in triathlon this is common practice. However, cyclists rather choose a combination of fluid and solid carbohydrate sources so as to maintain appetite and satiety throughout the races. The range of carbohydrate supplements is huge (drinks, gels, sweets, bars, etc.), and in addition, cyclists also use more conventional foods like sandwiches, pastries, and even soft drinks

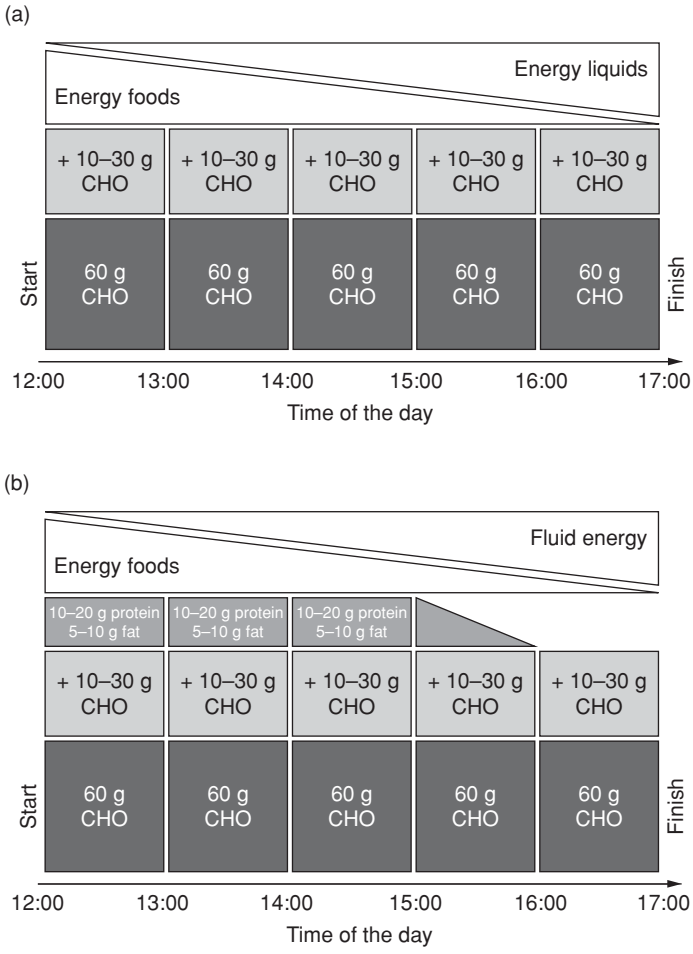

Figure 48.1 Time plan of carbohydrate intake throughout a 5-hour road race starting at noon in a one-day race (a) versus a stage race (b).

Table 48.2 Suggested carbohydrate content and energy values of some typical drinks and foods used during races in elite road cycling

	Carbohydrate (g)	Energy (kcal)
Energy liquids		
Energy sports drink (500 ml)	60–85	240–340
"Isotonic" sports drink (500 ml)	25–35	100–150
Energy gel (50 ml)	20–30	80–120
Soft drinks (33 cl can)	30–40	120–160
Energy foods (50 g portions)		
Muesli bar	~40	~185
Energy bar	~35	~190
Sandwich	~25	~135
Small sandwich with cheese or ham	~25	~200
Cakes/muffins	~25	~225
Banana, ripe	~10	~40

during competition. Therefore, it is essential that cyclists know the carbohydrate content of the drink and food portions they use (see Table 48.2) and translate this into a nutrition plan which minimizes GI discomfort and allows high rates of carbohydrate ingestion and adequate rehydration during exercise. Using this nutrition plan in training sessions lasting more than 3 hours will not only enhance training quality but at the same time train the gut for high rates of exogenous carbohydrate oxidation and better performance in endurance exercise (Cox et al., 2010).

The nutrition plan sets the limits for carbohydrate intake, but not water intake, during exercise. Therefore, besides the nutrition plan, depending on sweat rate, cyclists must drink extra water to prevent dehydration in excess of 2% of body weight. Body weight increase due to overdrinking must be avoided, of course. Water with a high salt content may help to prevent the development of muscle cramps and hyponatremia ("water intoxication"), especially in "salty sweaters" (Maughan & Shirreffs, 2008; Shirreffs & Sawka, 2011). Importantly, in the context of health promotion, commercial waters in general have very low sodium contents (less than 50 mg/l (2 mmol/l)). Therefore, it is recommended to add 500–1000 mg of sodium per liter, which corresponds to about 1250–2500 mg of kitchen salt. A teaspoon contains about 6 g of salt (2.5 g of sodium) which is enough to upgrade the sodium content of 3–5 liters of commercial water.

Another important question is the possible benefit of protein or amino acid intake in endurance exercise. From the perspective of performance enhancement, there is no reason to recommend protein or amino acid intake during aerobic exercise. However, moderate rates of protein intake will not negatively affect performance and may help to establish an appetizing nutritional plan to drive the required carbohydrate intake during exercise. Foods with high fiber and fat content must be carefully avoided because this may increase the incidence of GI problems without yielding any performance benefits.

Finally, it is important to emphasize that the above recommendations refer to one-day races, and must be at least partly revisited in the context of maintenance of energy balance and body weight in stage races (see section "Stage Races").

Recovery

Elite cyclists live in a permanent state of recovery from training or racing. They train daily, often twice per day, and race frequently, which implies that postexercise recovery mechanisms are recruited continuously. Recovery involves rehydration, restoration of muscle and liver glycogen stores, and last but not least, stimulation of muscle protein synthesis for muscle repair and remodeling which underpins training adaptation. Nutrition is pivotal to stimulate these processes, and neglecting postexercise nutrition inhibits the return of training in terms of performance improvement, in part because of decline of the training quality. Cyclists must be aware that the metabolic mechanisms involved in muscle recovery are highly activated immediately after exercise, and "recovery rate" rapidly drops as recovery time proceeds (see Figure 48.2). Thus, the initial few hours after an exercise bout must be considered "prime time"

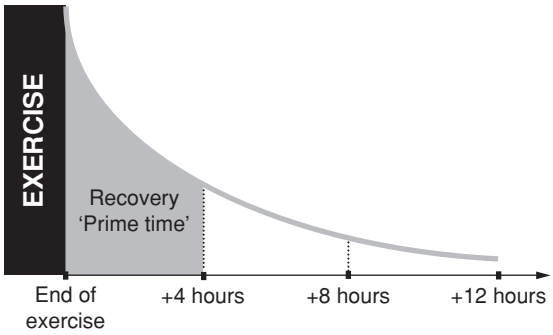

Figure 48.2 Rapid decline of muscle recovery rate after exercise.

for stimulation of recovery and training adaptation by nutritional interventions (Hawley et al., 2011; Mahoney et al., 2005). The shorter the time interval between training sessions or competitive events, the greater is the importance of developing an optimum nutrition plan for recovery in "prime time." It is impossible within the space of this chapter to provide detailed recommendation as to the optimum eating and drinking strategies to stimulate rehydration, glycogen repletion, as well as protein synthesis during recovery from training and competition in the different cycling disciplines. However, nutritional recovery procedures are explained in detail in Chapters 7 (muscle and liver glycogen resynthesis), 11 (muscle protein synthesis), and 16 (rehydration) earlier in this book.

It is also important to note that athletes probably should not aim at complete recovery of carbohydrate stores before every training session is started. Evidence is increasing that endurance training in a state of muscle glycogen depletion, due to omission of carbohydrate intake between training sessions, may facilitate training adaptations (see Chapter 13). By analogy, training with low liver glycogen but high circulating fatty acid concentration after an overnight fast, or training without exogenous carbohydrate supply during exercise, may augment training adaptation to the same or even greater degree compared to training with ample carbohydrate availability during exercise (Hansen et al., 2005; Hawley et al., 2011). However, such depletion strategies should be integrated in a well-balanced training schedule because consistent training in a carbohydrate depleted state in the end will exaggerate net protein breakdown (Wagenmakers et al., 1991), inhibit training adaptations, and suppress immunity and increase risk of infections (Gleeson & Walsh, 2012). In fact, maintaining adequate energy balance and carbohydrate availability in athletes is probably the best strategy to negate training-induced suppression of the immune system (Nieman, 2008).

Stage Races

Stage races are not on the Olympic calendar, but the Tour de France and other major stage races are probably the most popular cycling events worldwide. There are some important differences between nutrition for one-day races versus stage races (Table 48.3). In one-day races, as long as carbohydrate intake before and during exercise adequately supports energy production from carbohydrates throughout the race (Figure 48.1, panel a), maintenance of energy balance *per se* is not a matter of concern. Energy deficits (energy intake less than energy expenditure) developing during the race do not affect performance, and are easily compensated for by surplus energy intake on recovery days. In contrast, in stage races, a negative energy balance will impair the capacity to perform because consistent energy deficit can suppress even normal physiological functions at rest and thereby also inhibit recovery processes underpinning the next day's performance. Energy expenditure in the Tour de France stages on average amounts to about 6000 kcal per day, with peaks as high as 9000 kcal in strenuous mountain stages (Jentjens, 2004; Jeukendrup, 2004; Saris et al., 1989). This massive energy expenditure needs to be compensated for by equivalent energy intake during no more than 16 waking hours, of which 4–6 hours are spent on the bike and during which aggressive riding sometimes prevents the rider from eating. Moreover, the remaining resting time is often busy due to transport and changing accommodation, press meetings, start and finish procedures, etc., and appetite is suppressed by fatigue/exhaustion. Thus, maintenance of energy availability is a major challenge, and it

Table 48.3 Guidelines for a nutritional plan for carbohydrate and energy intake in one-day races versus stage races

	One-day race	Stage race
Breakfast	**Carbohydrate** 150–300 g depending on GI tolerance. **Protein** Not essential, but small amounts to serve appetite are OK. **Fat** Limit intake to allow for sufficient carbohydrate intake and to avoid GI problems. **Fiber** Limit intake to allow for sufficient carbohydrate intake and to avoid GI problems.	**Carbohydrate** 200–300 g depending on GI tolerance. **Protein** 25–50 g as an additional source of energy intake and within GI tolerance. **Fat** Limit intake to allow for sufficient carbohydrate intake and to avoid GI problems. **Fiber** Limit intake to allow for sufficient carbohydrate intake and to avoid GI problems.
Last hour before race	**Carbohydrate** 50–75 g of carbohydrate within limits of GI tolerance. Avoid intake of fat and fiber.	**Carbohydrate** 50–70 g of carbohydrate together with 5–10 g protein within limits of GI tolerance. Avoid intake of fat and fiber.
During the race	**Carbohydrate** 60–90 g/h depending on GI tolerance. **Protein** Not essential, but OK within GI tolerance. **Fat** Limit intake to avoid GI problems, but small amounts are OK. **Fiber** Limit intake to avoid GI problems, but small amounts are OK.	**Carbohydrate** 60–90 g/h depending on GI tolerance. **Protein** 10–20 g/h as an additional source of energy intake during lower intensity episodes of the race and within GI tolerance. **Fat** 5–10 g/h as an additional source of energy intake during lower intensity episodes of the race and within GI tolerance. **Fiber** Limit intake to avoid GI problems, but small amounts are OK.
Recovery 'prime time'	**Carbohydrate** 1.0–1.2 g/kg/h during initial 2 h. Thereafter resume habitual eating pattern for a hard training day. **Protein** 20–25 g high-quality protein containing about 10 g essential amino acids immediately after exercise. Thereafter resume normal eating pattern. **Fat** Limit intake during initial 2 h, but thereafter resume normal eating pattern. **Fiber** Limit intake during initial 2 h, but thereafter resume normal eating pattern, including fiber-rich foods.	**Carbohydrate** 1.0–1.2 g/kg/h for the first 4 h. **Protein** 20–25 g high-quality protein containing about 10 g essential amino acids immediately after exercise. Thereafter 5–10 g/h as an additional source of energy intake per hour and within GI tolerance. **Fat** Limit intake to avoid GI problems. **Fiber** Limit intake to avoid GI problems, but small amounts are OK.
Dinner	Start dinner with a light dish containing about 70–100 g carbohydrate (pasta, rice, etc.). Then shift to the other foods containing habitual amounts of the important macro- and micronutrients, including fiber.	Start dinner with a light dish containing about 70–100 g carbohydrate (pasta, rice, etc.). Then continue meal with foods containing extra energy in the form of protein and fat, within the limits of GI tolerance and assuring start of sleep with good GI comfort.

is probably correct to state that "GI performance" becomes a primary determinant of performance. Therefore, it is crucial that cyclists develop and train a nutrition plan providing (1) the required amounts of carbohydrates for muscle and liver glycogen repletion between stages, (2) surplus energy intake in the form of proteins and fats to maintain energy balance and body weight, and (3) ample fluid and sodium intake to restore fluid and sodium balance before the start of the next stage. In addition, such a plan must be successful in maintaining appetite throughout a 3-week period of mental stress and physical exhaustion.

Clearly, energy intake in the form of carbohydrates can never meet the energy requirements during a stage race. Carbohydrate intake recommended for timely restoration of glycogen stores between stages amounts to about 10–12 g/kg BW, which would make about 2800–3400 kcal in a 70 kg rider (1 g carbohydrate = 4 kcal). If the athlete is able to consistently maintain maximum rate of carbohydrate intake of about 90 g/h throughout a 5-hour stage, this would add about 1800 kcal. Thus, total daily energy intake in the form of carbohydrates at best amounts to no more than 4600–5200 kcal, which leaves an "energy gap" of about 1400–4000 kcal/day when balanced against expected energy expenditures. Positive energy balance in easy stages and on rest days can partly compensate for these energy deficits on "working days." Still, it is obvious that an important fraction of the energy requirement (1000–1500 kcal/day) will need to be covered by the intake of proteins (1 g protein = 4 kcal) and fat (1 g fat = 9 kcal). A high rate of protein intake (2 g/kg/day) could cover about half of this amount, leaving about 500–750 kcal to be ingested in the form of fat (50–80 g/day). In conclusion, in contrast to the plan for a one-day race which can focus only on carbohydrate availability, the nutritional plan for a stage race needs to supply a well-balanced mix of carbohydrates, proteins, and fats to assure adequate carbohydrate availability from the first to the last stage, as well as to maintaining energy availability and body weight throughout the competition (Table 48.3). Optimum utilization of "prime time" to stimulate recovery processes by adequate nutrition interventions is essential to maintaining the capacity to perform. Therefore, during prime time cyclists should try to ingest the required amounts of carbohydrates and proteins primarily in the form of liquids and light snacks, for they could present for dinner with a comfortable stomach allowing for additional carbohydrates and energy to be supplied. At the same time sufficient intake of water and sodium must power speedy rehydration to restore plasma volume, cardiac output, and peripheral blood flow (see Chapter 16).

Energy Balance and Weight Control

Weight Reduction for Efficient Uphill Cycling

Many cycling events such as road racing and mountain biking, include uphill cycling. Power requirements in uphill cycling are approximately proportional to speed and body mass. Therefore, within limits, anything that can be done to reduce body weight (BW) will improve hill-climbing performance. This knowledge often drives elite cyclists into mismanaged efforts to reducing fat mass to extremely low levels of less than 5% in males, or less than 12% in females. Improper weight reduction strategies can not only impair performance, but more importantly may compromise the athlete's health, certainly when disordered eating and eventually eating disorders are developing (Hagmar et al., 2008 and see Chapter 42). It is also well established that excessive energy deficits in athletes inhibit immune functions, which in turn increases the incidence of upper respiratory tract infections (Gleeson & Walsh, 2012; Hagmar et al., 2008). Furthermore, in females, excessive energy restriction in conjunction with intensive training also results in hormonal imbalances, which cause menstrual dysfunction as well as suppression of bone formation, with the possible risk of osteoporosis in a later stage (Loucks et al., 2011; Nattiv et al., 2007). Therefore, from the perspective of health and performance maintenance, it is essential that weight loss programs are designed by an experienced sports nutritionist with appropriate medical follow-up.

The effect of weight loss programs on health and performance depends largely on the effect of the diet on *energy availability*, which is defined

as dietary energy intake minus exercise energy expenditure (Loucks et al., 2011). For example, in a cyclist weighing 65 kg, with 6% body fat, and ingesting 5000 kcal/day, who has an average daily energy expenditure in training and competition of 4000 kcal/day, energy availability is limited to 1000 kcal/day or about 16 kcal/kg fat free mass per day. However, in young healthy adults, resting physiological functions are impaired at energy availabilities of less than 30 kcal/kg BW/day, quite apart from the loss of intended beneficial training adaptations due to nutrient deficiencies. Therefore, dietary regimens aiming for weight loss should not reduce energy availability to less than 30 kcal/kg BW/day (see Chapters 5 and 41). Any "intelligent" weight control program in elite cyclists should start with precluding excessive weight gain during off-season periods. Thus, riders can approach their target body weights early in the season by combining moderate dietary energy restriction with low-intensity endurance training to stimulate fat metabolism. Low energy availability is not compatible with high-intensity training and preparing for peak competition performances, because chronic carbohydrate deficiency impairs training quality and increases susceptibility to disease.

Maintaining Muscle Mass During Weight Loss

Cyclists typically aim at reducing inert mass (i.e., fat), without also losing muscle mass so as to obtain the highest possible fat-to-lean ratio and thus establish a high maximum oxygen uptake per kilogram body weight, which is closely correlated with uphill cycling performance (Swain, 1994). It is therefore important within weight loss diet not only to reduce total dietary energy intake, but at the same time to increase the fraction of energy intake via protein relative to carbohydrate and fat. Emerging evidence suggests that reducing the relative carbohydrate intake while increasing dietary protein intake to levels about twice as high as normally recommended, is pivotal to preserving muscle mass during periods of weight (fat) loss (Phillips & van Loon, 2011; Tipton, 2011). This means that during episodes of hypo-energetic diet with the express purpose of inducing weight loss, cyclists should aim to maintain a daily protein intake of at least 1.5 g/kg BW/day. Such high-protein diets are likely to enhance meal-induced stimulation of muscle protein synthesis, which is critical to maintaining muscle mass. Furthermore, the available literature data indicate that it probably is a good option to select high-quality dietary proteins, including a sufficient amount of essential amino acids (about 10 g, of which at least 2.5 g should be leucine) per meal (Phillips & van Loon, 2011 and see Chapter 11).

Building Muscle to Increasing Maximum Power Output

As indicated above, uphill cycling performance is strongly influenced by the capacity to develop a sustained high power output per kilogram body weight (Watt/kg). Conversely, in cycling on a flat route like on the track, the capacity to generate high absolute power outputs (Watt) comes into prominence, and thus obstinately seeking the "lowest possible body weight" is not on the agenda. Muscle strength is a primary determinant of maximum power output in cycling (Koninckx et al., 2010). Hence in track cyclists, and most particularly in sprinters, resistance training with the express purpose of inducing muscle hypertrophy becomes an important ingredient of the training program. It is well established that dietary protein intake plays an important role in stimulating muscle anabolism during resistance training (Tipton et al., 2007). Therefore, care must be taken to adequately tune the quality as well as the timing of protein intake in the context of resistance training sessions (see Chapter 11). Briefly, available evidence indicates that 20–25 g of high-quality protein, including about 10 g of essential amino acids, is an optimum quantity of protein to be consumed to maximally stimulate net muscle protein synthesis after resistance exercise (Phillips & van Loon, 2011). Milk proteins appear to be superior to other high-quality proteins like soy, probably because of the higher fractional content of leucine and other branched-chain amino acids in the former. Furthermore, the sooner after exercise the cyclist is able to consume the protein, the greater the stimulus to muscle protein accretion. However, the above rationale may require an alternative interpretation

for uphill specialists involved in a resistance training program either on the bike or in the gym: if a rider wants to avoid excess muscle mass accretion, it probably makes sense to abstain from protein supplement ingestion during the initial hour following specific resistance training sessions.

Conclusion

Nutrition plays an important role in cycling performance in both training and competition, but nutrition demands between the different cycling events. In any cycling discipline, the initial 4 hours following exercise are pivotal to facilitate recovery and stimulate training adaptations by deliberate intake of carbohydrates and high-quality proteins. Riders must develop and train a nutrition plan which delivers the amounts of carbohydrates, water, and sodium needed to support optimum performance on race days. However, in stage races, extra energy intake in the form of proteins and fats is essential to maintaining energy balance and performance capacity against the face of excessive daily energy expenditure. Weight loss to prepare for competition must be effected by a well-balanced dietary program involving increased fraction of energy intake via protein while maintaining energy availability higher than 30 kcal/kg BW/per day at all times.

References

Armstrong, L.E. (2000) Biorhytmic disturbances. In *Performing in Extreme Conditions*, 1st edn (eds. L.E. Armstrong), pp. 251–270. Human Kinetics, Champaign, IL.

Beelen, M., Berghuis, J., Bonaparte, B., et al. (2009) Carbohydrate mouth rinse in the fed state: lack of enhancement of time-trial performance. *International Journal of Sports Nutrition and Exercise Metabolism* 19, 400–409.

Bergström, J., Hermansen, L., Hultman, E., et al. (1967) Diet, muscle glycogen and physical performance. *Acta Physiologica Scandinavica* 71, 140–150.

Burke, L.M., Hawley, J.A., Wong, S.H., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Sciences* 29(Suppl. 1), S17–S27.

Carter, J.M., Jeukendrup, A.E., & Jones, D.A. (2004) The effect of carbohydrate mouth rinse on 1-h cycle time trial performance. *Medicine & Science in Sports & Exercise* 36, 2107–2111.

Cox, G.R., Clark, S.A., Cox, A.J., et al. (2010) Daily training with high carbohydrate availability increases exogenous carbohydrate oxidation during endurance cycling. *Journal of Applied Physiology* 109, 126–134.

Coyle, E.F., Coggan, A.R., Hemmert, M.K., et al. (1985) Substrate usage during prolonged exercise following a preexercise meal. *Journal of Applied Physiology* 59, 429–433.

Gant, N., Stinear, C.M., & Byblow, W.D. (2010) Carbohydrate in the mouth immediately facilitates motor output. *Brain Research* 1350, 151–158.

Gleeson, M. & Walsh, N.P. (2012) The BASES expert statement on exercise, immunity, and infection. *Journal of Sports Sciences* 30, 321–324.

Goforth Jr, H.W., Arnall, D.A., Bennett, B.L., et al. (1997) Persistence of supercompensated muscle glycogen in trained subjects after carbohydrate loading. *Journal of Applied Physiology* 82, 342–347.

Hagmar, M., Hirschberg, A.L., Berglund, L., et al. (2008) Special attention to the weight-control strategies employed by Olympic athletes striving for leanness is required. *Clinical Journal of Sports Medicine* 18, 5–9.

Hansen, A.K., Fischer, C.P., Plomgaard, P., et al. (2005) Skeletal muscle adaptation: training twice every second day versus training once daily. *Journal of Applied Physiology* 98, 93–99.

Hawley, J.A., Burke, L.M., Phillips, S.M., et al. (2011) Nutritional modulation of training-induced skeletal muscle adaptations. *Journal of Applied Physiology* 110, 834–845.

Hetzler, R.K., Warhaftig-Glynn, N., Thompson, D.L., et al. (1994) Effects of acute caffeine withdrawal on habituated male runners. *Journal of Applied Physiology* 76, 1043–1048.

James, J.E. & Rogers, P.J. (2005) Effects of caffeine on performance and mood: withdrawal reversal is the most plausible explanation. *Psychopharmacology* 182, 1–8.

Jentjens, R.L. (2004) Eating strategies for stage races. In *High-Performance Cycling*, 1st edn (ed. A.E. Jeukendrup), pp. 173–182. Human Kinetics, Champaign, IL.

Jentjens, R.L., Achten, J., & Jeukendrup, A.E. (2004a) High oxidation rates from combined carbohydrates ingested during exercise. *Medicine & Science in Sports & Exercise* 36, 1551–1558.

Jentjens, R.L., Moseley, L., Waring, R.H., et al. (2004b) Oxidation of combined ingestion of glucose and fructose during exercise. *Journal of Applied Physiology* 96, 1277–1284.

Jeukendrup, A.E. (2004) Energy needs for training and racing. In *High-Performance Cycling*, 1st edn (ed. A.E. Jeukendrup), pp. 141–153. Human Kinetics, Champaign, IL.

Jeukendrup, A.E. (2011) Nutrition for endurance sports: marathon, triathlon, and road cycling. *Journal of Sports Sciences* 29(Suppl. 1), S91–S99.

Jeukendrup, A.E. & Killer, S.C. (2010) The myths surrounding pre-exercise carbohydrate feeding. *Annals of Nutrition and Metabolism* 57(Suppl. 2), 18–25.

Koninckx, E., Van, L.M., & Hespel, P. (2010) Effect of isokinetic cycling versus weight training on maximal power output and endurance performance in cycling. *European Journal of Applied Physiology* 109, 699–708.

Loucks, A.B., Kiens, B., & Wright, H.H. (2011) Energy availability in athletes. *Journal of Sports Sciences* 29(Suppl. 1), S7–S15.

Mahoney, D.J., Parise, G., Melov, S., et al. (2005) Analysis of global mRNA expression in human skeletal muscle during recovery from endurance exercise. *FASEB Journal* 19, 1498–1500.

Maughan, R.J. & Shirreffs, S.M. (2008) Development of individual hydration strategies for athletes. *International Journal of Sports Nutrition and Exercise Metabolism* 18, 457–472.

Murray, R., Paul, G.L., Seifert, J.G., et al. (1989) The effects of glucose, fructose, and sucrose ingestion during exercise. *Medicine & Science in Sports & Exercise* 21, 275–282.

Nattiv, A., Loucks, A.B., Manore, M.M., et al. (2007) American College of Sports Medicine position stand: the female athlete triad. *Medicine & Science in Sports & Exercise* 39, 1867–1882.

Nieman, D.C. (2008) Immunonutrition support for athletes. *Nutrition Reviews* 66, 310–320.

Phillips, S.M. & van Loon, L.J. (2011) Dietary protein for athletes: from requirements to optimum adaptation. *Journal of Sports Sciences* 29(Suppl. 1), S29–S38.

Rogers, P.J., Hohoff, C., Heatherley, S.V., et al. (2010) Association of the anxiogenic and alerting effects of caffeine with ADORA2A and ADORA1 polymorphisms and habitual level of caffeine consumption. *Neuropsychopharmacology* 35, 1973–1983.

Saris, W.H.M., van Erp-Baart, A.M.J., Brouns, F., et al. (1989) Study on food intake and energy expenditure during extreme sustained exercise: the Tour de France. *International Journal of Sports Medicine* 10(Suppl. 1), S26–S31.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39, 377–390.

Sherman, W.M., Brodowicz, G., Wright, D.A., et al. (1989) Effects of 4 h preexercise carbohydrate feedings on cycling performance. *Medicine & Science in Sports & Exercise* 21, 598–604.

Shirreffs, S.M. & Sawka, M.N. (2011) Fluid and electrolyte needs for training, competition, and recovery. *Journal of Sports Sciences* 29(Suppl. 1), S39–S46.

Smith, J.W., Zachwieja, J.J., Peronnet, F., et al. (2010) Fuel selection and cycling endurance performance with ingestion of [13C]glucose: evidence for a carbohydrate dose response. *Journal of Applied Physiology* 108, 1520–1529.

Swain, D.P. (1994) The influence of body mass in endurance bicycling. *Medicine & Science in Sports & Exercise* 26, 58–63.

Tipton, K.D. (2011) Efficacy and consequences of very-high-protein diets for athletes and exercisers. *Proceedings of the Nutritional Society* 70, 205–214.

Tipton, K.D., Jeukendrup, A.E., & Hespel, P. (2007) Nutrition for the sprinter. *Journal of Sports Sciences* 25(Suppl. 1), S5–S15.

Wagenmakers, A.J.M., Beckers, E.J., Brouns, F., et al. (1991) Carbohydrate supplementation, glycogen depletion, and amino acid metabolism during exercise. *American Journal of Physiology—Endocrinology and Metabolism* 260, E883–E890.

Wright, D.A., Sherman, W.M., & Dernbach, A.R. (1991) Carbohydrate feedings before, during, or in combination improve cycling endurance performance. *Journal of Applied Physiology* 71, 1082–1088.

Chapter 49

Gymnastics

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Introduction

Elite competitive gymnasts are rarely older than the age of 25 and are typically between 16 and 19 years of age, a period that should be characterized by rapid growth and development. The traditional paradigm in gymnastics is to develop gymnasts who are small, and gymnasts themselves commonly view a small body as the ideal for their sport. Weight is, therefore, a prevailing theme regardless of the gymnastics discipline. In both men's and women's artistic gymnastics and in rhythmic gymnastics, it is commonly suggested that energy intake should be reduced to achieve lower weight, and that this is an appropriate and desired act if a gymnast is to achieve success (Deutz et al., 2000). This is, however, a counterproductive strategy that is likely to compromise both health and performance. Therefore, health assessments should include an evaluation of growth velocity, weight, body composition, bone density, eating behavior, menstrual status, and other developmentally important factors.

In 1960, the US Olympic gymnasts had an average height of 157.5 cm and an average weight of 50 kg. In 1992, the US Olympic gymnasts had an average height of 146 cm and an average weight of 37.5 kg. During this same time, the average age of these competitors dropped from 18.5 to 16 years. There is a premium placed on smallness, as illustrated by lower weights and heights of the most competitive gymnasts at the 2008 Beijing Olympic Games, than found in prior Olympic Games.

In 2000, the Fédération Internationale de Gymnastique addressed the issue of progressively smaller gymnasts by establishing 16 as the minimum age for competing at the Olympic Games. In the first year of this rule, a gymnast competing in the Sydney Olympics was later stripped of her medal because of a confirmed age falsification (Associated Press, 2010). There were also age controversies with gymnasts competing in the 2008 Olympics in Beijing. The main reason for falsifying age of gymnasts is that younger gymnasts are naturally smaller and may, therefore, be more capable of achieving difficult skills with a lower risk of injury.

Background

Elite level gymnastics has four disciplines:

- *Men's artistic gymnastics:* Competitions include six different events, including the floor exercise, side horse, horse vault, rings, parallel bars, and horizontal bar.
- *Women's artistic gymnastics:* Competitions include four different events, including the floor exercise, vault, uneven bars, and balance beam.
- *Women's rhythmic sportive gymnastics:* Competitions include four different routines, each performed as a floor exercise, with four of the five rhythmic apparatus (clubs, hoop, rope, ribbon, and balls). The four apparatus to be used are determined by

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Fédération Internationale de Gymnastique (FIG) every 2 years following the World Championships.

- *Women's rhythmic group gymnastics:* Competitions include two different routines performed by teams of six gymnasts. Each routine is performed with a combination of rhythmic apparatus (clubs, hoop, rope, ribbon, and balls). The apparatus combinations to be used are determined every 2 years by FIG following the World Championships.

Gymnastics training at the elite or aspirational levels typically takes place 5 or 6 days per week, for 3–5 hours each day, and for up to 30 hours of practice each week. Practice involves repeated bouts of highly intense, short-duration activity, and gymnasts rest after each practice bout to regenerate the phosphocreatine needed for repeated high strength bouts of activity (see Chapter 3). With the exception of the group competition in rhythmic gymnastics, which lasts 135–150 seconds, none of the competition events within each of these disciplines has a duration longer than 90 seconds. This exercise duration categorizes gymnastics as a high intensity, highly anaerobic sport, which is heavily dependent on phosphocreatine and glucose/glycogen as fuels for activity.

Energy and Nutrient Intakes

Elite gymnasts often demonstrate an inadequate intake of energy and several nutrients, primarily iron and calcium. A low intake of calcium, coupled with a low vitamin D exposure/intake may predispose gymnasts to stress fractures (Lovell, 2008). Inadequate iron intake is associated with anemia, which is a risk factor in the development of amenorrhea. Inadequate energy intake is implicated in a number of poor health and performance outcomes, including low bone density, an inability to sustain the muscle mass, and amenorrhea.

Energy Intake

Most competitive gymnasts are young and require sufficient energy to satisfy the physical demands of gymnastics plus the demands of growth and development, as well as the activities of daily living. As a result, adult energy requirements should not be applied to gymnasts as they require more energy per unit mass than more mature athletes doing the same level of work (Bar-Or, 2000). Of the published studies investigating energy intake in gymnasts, only former competitive gymnasts had average energy intakes that satisfied or exceeded the recommended level (Table 49.1). College team gymnasts from the United States were the oldest of the competitive gymnasts evaluated (mean age, 19.7 years) and had the lowest daily energy intakes of all the groups evaluated. The second lowest daily energy intake was seen in the US national team members. While underreporting of energy intake is a possibility (Jonnalagadda et al., 2000), this summary suggests that gymnasts involved in the highest levels of competition are most likely to have the greatest differential between the energy consumed and the energy required.

Energy intake inadequacy—or low energy availability (see Chapter 5)—has multiple implications, including impaired immunocompetence (Nova et al., 2001); feeling dizzy, weak, and short of breath during gymnastics practices (Ersoy, 1991); higher body fat percentages (Deutz et al., 2000); greater risk of multiple nutrient deficiencies; higher risk of low bone density (Deutz et al., 2000); and higher risk of amenorrhea (Loucks, 2006).

Energy Substrate Distribution The intake of energy substrates in gymnastics should be based on usage rate and the association of different energy substrates with other needed nutrients. Because gymnastics activity is primarily anaerobic, there is a heavy reliance on glycogen and phosphocreatine as fuels. Glycogen storage is best accomplished on diets that are relatively high in starchy carbohydrates, while creatine storage is best achieved through consumption of sufficient meat-based protein foods and adequate energy intake. Energy substrates for gymnasts should, therefore, be distributed as follows: 20–25% of total energy from fat, 15% (~1.2 to 1.7 g/kg) of total energy from protein, and 60–65% (~6 to 8 g/kg) of total energy from carbohydrate.

Table 49.1 Comparison of energy intakes versus requirements of different artistic gymnastic populations (female unless otherwise noted)

Subject age (years)	Population assessed (*n*)	Intake (kj)	Intake (kcal)	Percentage of pred. need	References
9.4 ± 0.8	US Jr Elite (29)	6934 ± 1525	1651 ± 363	76	Benardot et al. (1989)
11.4 ± 0.9	US Jr Elite (22)	7165 ± 1768	1706 ± 421	76	Benardot et al. (1989)
11.5 ± 0.5	Turkish Club (20)	6586	1568	—	Ersoy (1991)
12.3 ± 1.7	Italian Club (26)	6518 ± 2138	1552 ± 509	—	Reggiani et al. (1989)
12–15	US Jr Elite (21)	7194 ± 2418	1719 ± 578	83	Jonnalagadda et al. (1998)
14.8	US Level I, II (20)	7325	1744	—	Calabrese (1985)
14.8 ± 1.2	Swedish Elite (22)	8106 ± 1911	1930 ± 455	73	Lindholm et al. (1995)
15.2 ± 4.1	US High School (13)	8077 ± 2831	1923 ± 674	84	Moffatt (1984)
15.8 ± 0.9	US National Team (14)	6283 ± 1743	1496 ± 415	66	Benardot (1996)
16.02 ± 1.2	Spanish Gymnastics Fed(13)	5265 ± 557	1258 ± 133	—	Nova et al. (2001)
16–19	US National Team (8)	6574 ± 1904	1571 ± 455	72	Jonnalagadda et al. (1998)
19.7 ± 0.2	US College Team (26)	5800 ± 458	1381 ± 109	47	Kirchner et al. (1995)
	US College Team-Male (10)	8736	2080	—	Short and Short (1983)
36.3 ± 1.0	Former Competitive (18)	11,004 ± 1100	2620 ± 262	119	Kirchner et al. (1996)

It is unclear whether gymnasts would benefit by pursuing a glycogen loading technique to enhance total glycogen storage (Maughan & Poole, 1981). Supersaturation of the tissues with glycogen may cause excessive stiffness and a feeling of heaviness because of the increased water retention associated with stored glycogen (2.7 g H_2O for each gram of glycogen stored) (McArdle et al., 1986). This would be unacceptable in a sport where flexibility is needed for achieving the required skills. A reasonable approach would be one that encourages a high level of carbohydrate intake as a regular part of the diet rather than the initiation of a protocol that would lead to a supercompensation of carbohydrate in the tissues.

Of the energy gymnasts consume, on average larger proportion is derived from fats and lesser from carbohydrates (see Table 49.2). Of the 11 studies reviewed, only 3 had mean carbohydrate intakes greater than 60% of total energy, and 7 of the studies had fat intakes greater than 30% of total energy. It appears that most gymnasts would benefit by lowering fat intake and increasing the intake of carbohydrates. However, care must be taken that a shift toward carbohydrates and away from fat does not further exacerbate the already inadequate energy intake of gymnasts.

While there are limited data on male gymnasts, two surveys indicated that mean protein intake in male gymnasts is 2 g/kg//day, or more than 20% of total energy from protein (Brotherhood, 1984; Short & Short, 1983). By most measures, this level of protein intake is excessive and is not likely to be optimal for gymnasts (Butterfield et al., 1992).

Artistic gymnasts consume more carbohydrate (~57% of total energy) than rhythmic gymnasts (~48% of total energy); and relative to body weight artistic gymnasts consume more carbohydrate (~9.1 g/kg) than rhythmic gymnasts (~5.6 g/kg) (Soric et al., 2008). Despite the higher carbohydrate consumption, the artistic gymnasts had lower body fat percent than the rhythmic gymnasts (Deutz et al., 2000).

The issue of creatine intake (either as preformed creatine from dietary meat, or as a creatine monohydrate supplement) is an important one to consider,

Table 49.2 Energy substrate distribution in different gymnastic populations, organized by age of subjects

Subject age (years)	Total energy (kJ)	Total energy (kcal)	Energy from carb (%)	Energy from protein (%)	Energy from fat (%)	References
9.4 ± 0.8	6934 ± 1525	1657 ± 364	52	16	32	Benardot et al. (1989)
11.4 ± 0.9	7165 ± 1768	1712 ± 422	53	15	32	Benardot et al. (1989)
11.5 ± 0.5	6586	1574	57	15	28	Ersoy (1991)
12–15	7194 ± 2419	1719 ± 578	66	16	18	Jonnalagadda et al. (1998)
12.3 ± 1.7	6518 ± 2138	1557 ± 511	48	16	36	Reggianni et al. (1989)
14.8	7325	1750	50	12	38	Calabrese (1985)
14.8 ± 1.2	8106 ± 1911	1937	53	15	32	Lindholm et al. (1995)
15.2 ± 4.1	8077 ± 2831	1930 ± 676	46	16	38	Moffatt (1984)
15.8 ± 0.9	6283 ± 1743	1501 ± 416	65	19	16	Benardot (1996)
16–19	6574 ± 1904	1571 ± 455	70	16	14	Jonnalagadda et al. (1998)
19.7 ± 0.2	5800 ± 458	1385 ± 109	53	16	31	Kirchner et al. (1995)
—	8736	2087	44	16	40	Short and Short (1983)
36.3 ± 1.0	11004 ± 1100	2629 ± 263	49	14	37	Kirchner et al. (1996)

since athletes involved in high intensity anaerobic sports may benefit from a higher level of creatine intake (Maughan, 1995; see Chapter 24).

Nutrient Intakes

Gymnasts typically have intakes that are below established recommended levels in one or more nutrients, likely because total energy intake is also low. It is difficult to predict the true requirement for nutrients in this population because, although growing, they are small in stature with a higher proportion of metabolic mass than the average for people their age. In addition, there is no clear way to accurately predict how anaerobic activities might influence nutrient usage (and requirement) in this population (Table 49.3).

Vitamin A (Retinol) In three studies evaluating vitamin A intake in gymnasts, subjects consumed less than the recommended level of 1000 mg Retinol Equivalents (Ersoy, 1991; Moffatt, 1984; Reggiani et al., 1989;). In four other surveys, gymnasts were found to consume adequate levels of vitamin A (Benardot et al., 1989; Jonnalagadda et al., 1998; Lindholm et al., 1995; Short & Short, 1983;). There is no apparent pattern of vitamin A intake among younger, older, elite, and nonelite gymnasts. The recommended dietary allowance (RDA) is set at 2 standard deviations above the average requirement. When a value of 75% of the RDA is applied to the intake of vitamin A, all surveys indicate that the consumption of vitamin A in gymnasts is adequate.

Vitamin D (Cholecalciferol, Calcitriol, and Calciferol) Vitamin D is important in the development of healthy bones, muscle contraction, intestinal absorption of nutrients (including calcium), muscle protein anabolism, immune function, and anti-inflammatory action (Benardot, 2012). Inadequate sunlight exposure (likely in competitive gymnasts) would compromise vitamin D status and its functions. A study assessing vitamin D status in eighteen 10- to 17-year-old female gymnasts found that the majority (15) had below optimal vitamin D status (<75 nmol/l) and, of these, six had vitamin D levels below 50 nmol/l (Lovell, 2008).

Vitamin C (Ascorbic Acid) A study evaluating vitamin C consumption in 12- to 13-year-old competitive gymnasts found an average intake that was marginally below the recommended intake level

Table 49.3 Summary of selected nutrient intakes in surveys of artistic gymnasts

Subject group (*n*)	Vit. A (mg^{RE})	Vit. C (mg)	Vit. B1 (mg)	Vit.B2 (mg)	Niacin (mg^{NE})	Calcium (mg)	Iron (mg)	References
College elite male (10)	1100	97	1.10	1.20	16.0	1059	12.0	Short and Short (1983)
High-school female (13)	883	84	1.04	1.39	13.4	706	11.3	Moffatt (1984)
7- to 10-year-old competitive female (29)	1031	129	1.40	1.80	17.5	840	11.0	Benardot et al. (1989)
11- to 14-year-old competitive female (22)	1127	145	1.50	1.80	18.2	867	11.0	Benardot et al. (1989)
11- to 14-year-old elite female (8)	955	2019	1.7	2.5	21.0	1073	18.0	Jonnalagadda et al. (1998)
12- to 13-year-old competitive female (26)	771	56	0.60	0.70	8.7	539	6.2	Reggiani et al. (1989)
10- to 12-year-old competitive female (20)	834	64	0.74	1.45	8.5	397	8.4	Ersoy (1991)
15- to 18-year-old elite female (21)	1079	400	2.4	3.1	32	1094	25.0	Jonnalagadda et al. (1998)
Elite adolescent female (22)	1200	79	—	—	—	1215	14.0	Lindholm et al. (1995)
College elite female (26)	—	—	—	—	—	683	11.8	Kirchner et al. (1995)

Values are average intakes for assessed groups.

(Reggiani et al., 1989). Vitamin C intake in four other studies was only marginally better than the recommended intake of 60 mg/day (Ersoy, 1991; Lindholm et al., 1995; Moffatt, 1984; Short & Short, 1983). In two surveys of 7- to 10-year-old and 11- to 14-year-old gymnasts, the intake of vitamin C was approximately double the recommended level for age and gender (Benardot et al., 1989; Jonnalagadda et al., 1998).

Vitamin B1 (Thiamin) The intake of vitamin B1 was below the recommended level of 1.3–1.5 mg/day in three surveys of gymnasts (Ersoy, 1991; Moffatt, 1984; Reggiani et al., 1989; Short & Short, 1983). A marginally adequate intake of vitamin B1 was found in 7- to 10-year-old and 11- to 14-year-old competitive female gymnasts (Benardot et al., 1989). Since most of the gymnastic surveys indicate an underconsumption of energy, an appropriate strategy for improving vitamin B1 intake in gymnasts is an improvement in total energy consumption.

Vitamin B2 (Riboflavin) With the exception of two surveys (Benardot et al., 1989; Jonnalagadda et al., 1998), all other nutrient intake studies indicate that riboflavin intake is below the RDA of 1.5–1.8 mg/day. However, when evaluated as 0.6 mg per 4.2 MJ (1000 kcal) consumed (the basis of the RDA), the vitamin B2 intake of gymnasts meets or exceeds the required level in all of the surveys.

Niacin Using the niacin RDA for young and adolescent females of 15 mgNE, three groups of surveyed gymnasts had niacin intakes below the recommended level (Ersoy, 1991; Moffatt, 1984; Reggiani et al., 1989). These groups, including gymnasts in high school, elite gymnasts, and very young competitive gymnasts, had intakes of niacin that ranged between 89% and 57% of the recommended levels.

Calcium The results of several surveys on gymnasts indicate a level of calcium intake that is significantly lower than the recommended level of intake. Calcium intake of gymnasts appears to be low across all groups evaluated.

Iron The iron intake of gymnasts was found to be below the recommended level (15 mg/day in females between 11 and 24 years) in all of the

surveys reviewed. This has numerous implications for the gymnasts' resistance to disease, but also has implications for growth, strength, and the ability to concentrate (Loosli, 1993). The current recommendation of 15 mg iron/day for adolescent females (age 14–18 years) includes an allowance for menstrual losses and growth (Food and Nutrition Board, 2011). In fact, linear growth velocity and enlargement of blood volume during adolescence is the reason the male recommended intake is only slightly lower (11 mg/day) than that for females (Food and Nutrition Board, 2011). Limited published data on hemoglobin, hematocrit, and ferritin status of gymnasts makes it impossible to understand if current iron intakes match actual need. There are some data indicating, however, that a significant number of gymnasts have low serum iron and a high rate of anemia (Lindholm et al. 1995). It is estimated that the typical diet in industrialized nations provides ~6 mg of iron per 4.2 MJ (1000 kcal) of energy. Given the reported energy intakes of gymnasts, it is doubtful that gymnasts would consume more than 12 mg iron/day.

Nutritionally Related Problems Studied in Gymnasts

Female Athlete Triad

This triad of disorders represents eating disorders (anorexia nervosa, anorexia athletica, bulimia, and other restrictive eating behaviors), amenorrhea (both primary and secondary), and early development of osteoporosis (Smith, 1996; see Chapter 42). The prevalence of the female athlete triad in gymnastics remains unclear, but there is convincing evidence that it exists in gymnastics, and represents a serious and potentially life-threatening reality (Sundgot-Borgen, 1994).

Delayed menarche, menstrual irregularities, and low body fat are frequently seen in both rhythmic and artistic gymnasts (Georgopoulos et al., 2001) but, contrary to the commonly held belief that the low body fat is principally responsible for the menstrual dysfunction, it appears that low energy availability is the primary cause. According to Loucks (2003), dysfunction of reproductive hormones occurs below 20–30 kcal/kgLBM/day of energy availability (energy consumed minus exercise energy expended in training).

Amenorrhea is now considered the most serious clinical problem associated with abnormal bone development (Roupas & Georgopoulos, 2011). Trabecular bone is more sensitive to low circulating estrogen, while cortical bone may be stabilized or even increase in density with physical activity, even in the presence of inadequate estrogen. This has been clearly demonstrated in one study evaluating elite college gymnasts, which showed an increase in bone mineral density despite the presence of amenorrhea or oligomenorrhea (Nichols et al., 1994). Younger gymnasts, age 7–8 years, demonstrate a higher bone mineral density, likely due to the high impact and weight-bearing gymnastics activity (Zanker et al., 2003). This young age bone acquisition may serve to provide some protection to gymnasts through the adolescent years, when menarche is delayed (Zanker et al., 2004). Former collegiate female gymnasts who have not trained for 4+ years still have bone mineral densities that are higher than age-matched controls (Kudlac et al., 2004). While rhythmic gymnastics has less impact stress than artistic gymnastics, there is also evidence that rhythmic gymnasts have improvements in cortical bone density (Tournis et al., 2010).

Gymnastics Injuries

Gymnastics injuries occur, and often it is an injury that takes talented gymnasts out of the sport. In the study by Dixon and Fricker (1993), stress fractures of the lumbosacral spine accounted for 45% of all skeletal injuries in female gymnasts. The feet accounted for 32% of stress fractures and 28% of all bony injuries. In male gymnasts, stress fractures of the lumbosacral spine accounted for 33% of all stress fractures and 16% of all bony injuries. In the male gymnasts, there were approximately the same number of stress fractures and fractures (Dixon & Fricker, 1993). Pain from minor injury is also an important factor, as it serves to decrease

training and hampers performance. In artistic gymnasts, it was found that the most common sites of pain were the ankle and low back, both of which appear to be associated to training (Marini et al., 2008). Rhythmic gymnasts appear to be at higher risk of back pain (Piazza et al., 2009).

Several studies have demonstrated a relationship between injury frequency and nutritional factors. Muscle-glycogen depletion is associated with fatigue, muscle fiber damage, and joint weakness that could predispose an athlete to skeletal injury (Schlabach, 1994). An adequate calcium intake of 1500 mg/day may impart some degree of safety in helping to reduce fracture risk (Heaney, 1991), and if it is not possible to obtain sufficient calcium through food consumption, calcium supplementation has been found to be effective in increasing bone mineral density in children (Johnston et al., 1992).

Attainment of Ideal Weight and Body Composition

Gymnastics favors individuals with a high strength-to-weight ratio, and who are relatively small in stature (Cort, 2006; Table 49.4). This sport-specific 'norm' may predispose gymnasts to manipulate diet, often using unhealthy energy-restricting strategies, in an attempt to achieve the desired body characteristics that are associated with success in competition. There is evidence that female and male gymnasts may both use dietary restriction as a means of achieving an artificially small body size, but who mistakenly believe this places them in a more 'fit' state (Atkinson, 2011). It is notable that rhythmic gymnasts have higher body fat percentages than artistic gymnasts at comparable ages, despite the finding that rhythmic gymnasts had higher energy balance deficits than artistic gymnasts (Deutz et al., 2000).

Growth Retardation

Gymnasts are significantly smaller than non-gymnasts of the same age and miss the distinct growth spurt typically seen in adolescence (Lindholm et al., 1994). It has been reported that gymnasts who train more than 18 hours/week before and during puberty have marked stunting of growth (Theintz et al., 1993). If this intensive exercise schedule occurs before puberty, the gymnasts would permanently alter the growth rate and keep them from ever reaching full adult height. Gymnasts between the ages of 13 and 20 years are considerably shorter and lighter with narrower hips than age-matched non-gymnasts.

It is unclear whether the reduced growth in gymnasts is due to a diet-related inhibition of the hypothalamic–pituitary–gonadal axis from inadequate energy and nutrient intake, or from the combination of inadequate energy and nutrients coupled with a heavy training regimen (Lindholm et al., 1994). Female gymnasts have significantly delayed the age of menarche when compared to non-gymnasts, and are also shorter and lighter. A study of hormonal responses to gymnastics training in elite peripubertal male gymnasts found that there was a change in the anabolic to catabolic balance, as represented by IGF-1:Cortisol ratio, which indicated a catabolic state from energy intake inadequacy (Daly et al., 1998).

Not all gymnasts experience growth retardation. A study of rhythmic gymnasts found a significant delay in normal growth-spurt associated skeletal maturation and pubertal development. These gymnasts experienced a later growth spurt that preserved and, in some cases, exceeded linear growth expectations (Georgopoulos et al., 2001, 2002). By contrast, female artistic gymnasts were found to have heights that were below the predicted values that could result in shorter adult stature. A study of gymnasts competing at the European Championships found that artistic gymnasts achieved each pubertal stage at a later age than rhythmic gymnasts, and that the delay was closely associated with the amount of energy expended, which was higher in the artistic gymnast (Theodoropoulou et al., 2005). There is also evidence of different age at peak height velocity in advanced (higher training) versus intermediate (lower training) gymnasts. Height velocity was lower in the advanced level gymnasts (Daly et al., 2005).

Table 49.4 Heights, weights, and body-fat percentages of female gymnasts

Population, age in years (*n*)	Height (cm)	Weight (kg)	Body fat (%)	Method	References
Artistic gymnasts, female					
Junior elite, age 8.0 (10)	125.3 ± 1.6	24.1 ± 0.7	15.1 ± 0.9	DEXA	Zanker et al. (2003)
Junior elite, age 9.1 (100)	131.1 ± 6.6	27.3 ± 4.1	8.6 ± 2.0	Skinfolds	Benardot and Czerwinski (1991)
Junior elite, age 9.4 (51)	134.9	30.6	9.3	Skinfolds	Benardot et al. (1989)
Junior elite, age 11.3 (46)	141.0 ± 6.9	32.8 ± 4.9	9.2 ± 1.9	Skinfolds	Benardot and Czerwinski (1991)
Junior elite, age 11.5 (19)	142.0 ± 2.8	31.6 ± 1.5	21.5	Skinfolds	Ersoy (1991)
Junior club, age 12.3 (26)	145.8 ± 8.5	37.9 ± 6.9	15.0 ± 3.5	Bioelectrical impedance	Reggiani et al. (1989)
Junior elite, age 12.3 (22)	142.0 ± 1.3	33.2 ± 1.0	14.9 ± 0.7	Skinfolds	Theintz et al. (1993)
Junior elite, age 13.3 (20)	148.0 ± 9.6	39.9 ± 7.0	10.9 ± 3.2	Hydrostatic weighing	Bale et al. (1996)
Club level, age 14.8 (20)	152.0	43.5			Calabrese (1985)
Junior elite, age 14.8 (22)	158.0	46.8	13.2	Skinfolds	Lindholm et al. (1995)
High school, age 15.2 (13)	16.1 ± 3.8	50.4 ± 6.5	13.1 ± 5.1	Hydrostatic weighing	Moffatt (1984)
Elite, age 15.2 (31)	150.9 ± 8.2	46.5 ± 8.3	12.36 ± 3.9	DEXA	Deutz et al. (2000)
Elite, age 15.2 (31)	150.9 ± 8.2	46.5 ± 8.3	11.31 ± 2.5	Skinfolds	Deutz et al. (2000)
Elite, age 15.8 (22)	153.3 ± 5.9	46.9 ± 6.1	11.3 ± 3.7	DEXA	Benardot (1996)
Elite, age 16.2 (388)	—	—	21.8 ± 5.8	Infrared analysis	Georgopoulos et al. (2002)
Elite, age 17.3 ± 1.9 (142)	154.4 ± 6.6	47.2 ± 6.9	20.6 ± 5.3	Infrared analysis	Georgopoulos et al. (2002)
College, age 19.5 (21)	159.4 ± 4.3	55.0 ±6.5	15.6 ± 2.9	DEXA	Robinson et al. (1995)
College, age 19.7 (10)	158.7 ± 4.8	53.0 ± 6.1	16.8 ± 3.2	Hydrostatic weighing	Barlett et al. (1984)
College, age 19.7 (26)	158.0 ± 1.1	54.1 ± 1.2	17.0 ± 0.5	DEXA	Kirchner et al. (1995)
College, age 20.4 (10)	163.0 ± 6.1	60.0 ± 5.4	23.2 ± 1.7	DEXA	Kudlac et al. (2004)
Former elite, age 25.8 (18)	162.0 ± 5.8	59.0 ± 7.4	30.1 ± 6.0	DEXA	Zanker et al. (2004)
Former elite, age 36.3 (18)	161.6 ± 1.5	59.7 ± 1.8	23.9 ± 1.0	DEXA	Kirchner et al. (1996)
Rhythmic gymnasts, female					
Elite, age 14.5 (15)	160.0 ± 1.2	43.4 ± 1.3	14.3 ± 0.5	Skinfolds	Klentrou and Plyley (2003)
Elite, age 14.7 (30)	163.4 ± 1.8	45.9 ± 1.7	16.2 ± 0.4	Skinfolds	Klentrou and Plyley (2003)
Elite, age 15.9 (268)	—	—	15.5±4.6	Infrared analysis	Georgopoulos et al. (2002)
Elite, age 16.0 (104)	163 ± 5.6	45.3 ± 6.6	15.9 ± 4.9	Infrared analysis	Georgopoulos et al. (2001)
Elite, age 16.5 (11)	164.3 ± 3.3	51.0 ± 4.3	16.6 ± 3.1	DEXA	Deutz et al. (2000)
Elite, age 16.5 (11)	164.3 ± 3.3	51.0 ± 4.3	13.7 ± 1.6	Skinfolds	Deutz et al. (2000)
Elite, age 17.1 (129)	166.3 ± 4.6	47.3 ± 4.8	13.1 ± 4.9	Infrared analysis	Georgopoulos et al. (2002)

Summary Recommendations: General Guidelines

Gymnastics training coupled with proper energy/nutrient intake can have a positive impact on health and performance. However, a failure to supply adequate energy and nutrients may have a profound negative impact on health factors that directly or indirectly affect performance, including the development of poor bone mass, menstrual dysfunction, and eating disorders (Beals & Hill, 2006). Gymnasts should consume sufficient energy for activity plus the needs of growth, and should consume sufficient fluids to assure adequate hydration. Adequate energy availability is a key factor in reducing health risks in all athletes, including gymnasts (Loucks, 2003, 2006).

Complex carbohydrate foods should be a large part of the diet, but the consumption of fibrous vegetables should be avoided for several hours before training or competition because they are potentially gas-causing and may make the gymnast uncomfortable from distention. A slight lowering of fat intake coupled with an increase in carbohydrate intake may be a desirable for many gymnasts. This can most easily be achieved through limited consumption of fried foods, visible fats, and fatty dairy products. There should be a reliance on food rather than vitamin and mineral supplements for obtaining needed nutrients but, with proper medical supervision, the intake of certain mineral supplements (calcium and iron in particular) may be advisable. Vegetarianism may increase nutrient risk for protein, iron, zinc, and calcium, but proper dietary planning can overcome these risks.

Fluid should be consumed at fixed and regular intervals to maintain optimum hydration status. Avoidance of thirst is important, since the thirst sensation does not occur until there has been a significant lowering of total body water.

Pre-competition/Pretraining Eating

The two main goals for the pre-competition/pretraining eating (PCPTE) include the provision of energy to see the athlete through a significant portion of the PCPTE, and sufficient fluid to assure optimally hydrated muscles. Unfamiliar foods should be avoided at this time. In general, the PCPTE should focus on providing starch-based carbohydrates and fluids. Provision of a nutritionally balanced meal should not be a major concern at this time, especially if nutritious foods are commonly consumed during other times.

There should be adequate opportunity for gastric emptying before the initiation of exercise. Because fats cause a delay in gastric emptying, fat intake for the PCPTE should be kept as low as possible. If the meal consumed is large, it should be completed 3.5–4.0 hours prior to the start of training or competition. Small meals can be completed 2–3 hours before exercise. Light carbohydrate snacks (crackers, etc.) may be consumed within 1 hour of exercise, but solid foods should always be consumed with fluids.

Nervous gymnasts may not tolerate solid food before competition, yet they still require energy to fuel the activity. One possible solution is to consume large amounts of carbohydrate the day before the competition (as small frequent meals), and consume only small periodic snacks with fluids on the day of competition. Fluid consumption should be sufficient before the PCPTE to produce clear urine. The usual recommendation is the consumption of 5–7 ml of fluid/kg body weight at least 4 hours prior to physical activity (American Dietetic Association, 2009).

Eating During Competition/Practice

Gymnasts require some source of energy during training and competition. Two main strategies may be tried during training. One strategy is to sip on a sports beverage that contains carbohydrate energy throughout the practice. Consumption of ~115 to 235 ml of a beverage containing 6–8% carbohydrate every 15–20 minutes is the generally accepted recommendation (American Dietetic Association, 2009), but the amount should be adjusted according to the size of the gymnast and environmental heat and humidity. It is useful to consume a beverage that also contains sodium at a concentration of 200–400 mg/l. It is important to avoid drinking a great deal all at one time, since that may cause

bloating and difficulties with training. Instead, the gymnast must become accustomed to sipping on the beverage periodically. The goal is to ensure that blood glucose and blood volume are maintained.

During gymnastics competition, it is not reasonable to assume that the gymnast will be able to take a snack break. Therefore, gymnasts should periodically sip small amounts of sports beverage between events throughout the competition (115–235 ml every 15–20 minutes when possible). This consumption pattern should be well-practiced prior to competition.

Post-competition/Post-practice Eating

Muscles are receptive to replacing glycogen within the first hour following strenuous activity. Therefore, gymnasts should have carbohydrate snacks available to consume immediately following training and competition. Ideally, the gymnast should consume 840–1670 kJ (200–400 kcal) (one medium-sized bagel is 695 kJ or 165 kcal; 1 cup pasta is 900 kJ or 215 kcal) immediately following the activity, and then consume an additional 840–1260 kJ (200–300 kcal) of carbohydrate within the next several hours. As always, fluids should be consumed when solid foods are consumed. Rehydration can be achieved by drinking ~1.5 liters of fluid for every 1 kg (24 oz for every pound) of body weight lost during exercise (American Dietetic Association, 2009).

References

American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine (2009) Position of the American Dietetic Association, Dietitians of Canada, and the American College of Sports Medicine: nutrition and athletic performance. *Journal of the American Dietetic Association* 109, 509–527.

Associated Press (2010) IOC strips 2000 Games bronze medal from China. *USA Today* (April 28). Available at: http://usatoday30.usatoday.com/sports/olympics/2010-04-28-211516359_x.htm (accessed May 17, 2013).

Atkinson, M. (2011) Male athletes and the cult(ure) of thinness in sport. *Deviant Behavior* 32, 224–256.

Bale, P., Doust, J., & Dawson, D. (1996) Gymnasts, distance runners, anorexics body composition and menstrual status. *Journal of Sports Medicine and Physical Fitness* 36, 49–53.

Bar-Or, O. (2000) Nutrition for child and adolescent athletes. *Sports Science Exchange* #77 13(2).

Barlett, H.L., Mance, M.J., & Buskirk, E.R. (1984) Body composition and expiratory reserve volume in female gymnasts and runners. *Medicine & Science in Sports & Exercise* 16, 311–315.

Beals, K.A. & Hill, A.K. (2006) The prevalence of disordered eating, menstrual dysfunction and low bone mineral density among US collegiate athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 16, 1–23.

Benardot, D. (1996) Working with young athletes: views of a nutritionist on the sports medicine team. *International Journal of Sport Nutrition* 6, 110–120.

Benardot, D. (2012) Chapter 2: vitamins. In *Advanced Sports Nutrition*, 2nd edn. Human Kinetics, Champaign, IL.

Benardot, D. & Czerwinski, C. (1991) Selected body composition and growth measures of junior elite gymnasts. *Journal of the American Dietetic Association* 91(1), 29–33.

Benardot, D., Schwarz, M., & Heller, D.W. (1989) Nutrient intake in young, highly competitive gymnasts. *Journal of the American Dietetic Association* 89, 401–403.

Butterfield, G., Cady, C., & Moynihan, S. (1992) Effect of increasing protein intake on nitrogen balance in recreational weight lifters. *Medicine & Science in Sports & Exercise* 24, S71.

Brotherhood, J.R. (1984) Nutrition and sports performance. *Sports Medicine* 1, 350–389.

Calabrese, L.H. (1985) Nutritional and medical aspects of gymnastics. *Clinics in Sports Medicine* 4, 23–30.

Cort, M. (2006) Even-specific nutrition and relationship to weight control. *Sports Coach* 29(2), 1–4.

Daly, R.M., Rich, P.A., & Klein, R. (1998) Hormonal responses to physical training in high-level peripubertal male gymnasts. *European Journal of Applied Physiology* 79, 74–81.

Daly, R.M., Caine, D., Bass, S.L., et al. (2005) Growth of highly versus moderately trained competitive female artistic gymnasts. *Medicine & Science in Sports & Exercise* 37(6), 1053–1060.

Deutz, R.C., Benardot, D., Martin, D.E., et al. (2000) Relationship between energy deficits and body composition in elite female gymnasts and runners. *Medicine & Science in Sports & Exercise* 32(3), 659–668.

Dixon, M. & Fricker, P. (1993) Injuries to elite gymnasts over 10 years. *Medicine & Science in Sports & Exercise* 25, 1322–1329.

Ersoy, G. (1991) Dietary status and anthropometric assessment of child gymnasts. *Journal of Sports Medicine and Physical Fitness* 31, 577–580.

Food and Nutrition Board, Institute of Medicine (2011) *Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes*. National Academy of Sciences, Washington, DC.

Georgopoulos, N.A., Markou, K.B., Theodoropoulou, A., et al. (2001) Height velocity and skeletal maturation in elite female rhythmic gymnasts. *Journal of Clinical Endocrinology & Metabolism* 86(11), 5159–5164.

Georgopoulos, N.A., Markou, K.B., Theodoropoulou, A., et al. (2002) Growth

retardation in artistic compared with rhythmic elite female gymnasts. *Journal of Clinical Endocrinology & Metabolism* 87(7), 3169–3173.

Heaney, R.P. (1991) Effect of calcium on skeletal development, bone loss, and risk of fractures. *American Journal of Medicine* 91(Suppl. 5B), 23–28.

Jonnalagadda, S.S., Benardot, D., & Nelson, M. (1998) Energy and nutrient intakes of the United States national women's artistic gymnastics team. *International Journal of Sport Nutrition* 8, 331–344.

Jonnalagadda, S.S., Benardot, D., & Dill, M.N. (2000) Assessment of under-reporting of energy intake by elite female gymnasts. *International Journal of Sport Nutrition and Exercise Metabolism* 10, 315–325.

Johnston, C.C., Miller, J.Z., Slemenda, C.W., et al. (1992) Calcium supplementation and increases in bone mineral density in children. *New England Journal of Medicine* 327, 82–87.

Kirchner, E.M., Lewis, R.D., & O'Connor, P.J. (1995) Bone mineral density and dietary intake of female college gymnasts. *Medicine & Science in Sports & Exercise* 27, 543–549.

Kirchner, E.M., Lewis, R.D., & O'Connor, P.J. (1996) Effect of past gymnastics participation on adult bone mass. *Journal of Applied Physiology* 80, 226–232.

Klentrou, P. & Plyley, M. (2003) Onset of puberty, menstrual frequency, and body fat in elite rhythmic gymnasts compared with normal controls. *British Journal of Sports Medicine* 37, 490–494.

Kudlac, J., Nichols, D.L., Sanborn, C.F., et al. (2004) Impact of detraining on bone loss in former collegiate female gymnasts. *Calcified Tissue International* 75, 482–487.

Lindholm, C., Hagenfeldt, K., & Ringertz, B.M. (1994) Pubertal development in elite juvenile gymnasts: effects of physical training. *Acta Obstetrica et Gynaecologica Scandinavica* 73, 269–273.

Lindholm, C., Hagenfeldt, K., & Hagman, U. (1995) A nutrition study in juvenile elite gymnasts. *Acta Paediatrica* 84, 273–277.

Loosli, A.R. (1993) Reversing sports-related iron and zinc deficiencies. *Physician and Sportsmedicine* 21, 70–78.

Loucks, A.B. (2003) Energy availability, not body fatness, regulates reproductive function in women. *Exercise and Sport Sciences Reviews* 31(3), 144–148.

Loucks, A.B. (2006) The response of luteinizing hormone pulsatility to 5 days of low energy availability disappears by 14 years of gynecological age. *Journal of Clinical Endocrinology and Metabolism* 91, 3158–3164.

Lovell, G. (2008) Vitamin D status of females in an elite gymnastics program. *Clinical Journal of Sports Medicine* 18(2), 159–161.

McArdle, W.D., Katch, F.I., & Katch, V.L. (1986) *Exercise Physiology: Energy, Nutrition, and Human Performance*, 2nd edn. Lea & Febiger, Philadelphia, PA.

Marini, M., Sgambati, E., Barni, E., et al. (2008). Pain syndromes in competitive elite level female artistic gymnasts. Role of specific preventive-compensative activity. *Italian Journal of Anatomy and Embryology* 113(1), 47–54.

Maughan, R.J. (1995) Creatine supplementation and exercise performance. *International Journal of Sport Nutrition* 5, 94–101.

Maughan, R. & Poole, D. (1981) The effects of a glycogen-loading regimen on the capacity to perform anaerobic exercise. *European Journal of Applied Physiology* 46, 211–214.

Moffatt, R.J. (1984) Dietary status of elite female high school gymnasts: inadequacy of vitamin and mineral intake. *Journal of the American Dietetic Association* 84, 1361–1363.

Nichols, D.L., Sanborn, C.F., Bonnick, S.L., et al. (1994) The effects of gymnastics training on bone mineral density. *Medicine & Science in Sports & Exercise* 26, 1220–1225.

Nova, E., Montero, A., Lopez-Varela, S., et al. (2001) Are elite gymnasts really malnourished? Evaluation of diet, anthropometry and immunocompetence. *Nutrition Research* 21, 15–29.

Piazza, M., DiCagno, A., Cupisti, A., et al. (2009) Prevalence of low back pain in former rhythmic gymnasts. *Journal of Sports Medicine and Physical Fitness* 49(3), 297–300.

Reggiani, E., Arras, G.B., Trabacca, S., et al. (1989) Nutritional status and body composition of adolescent female gymnasts. *Journal of Sports Medicine* 29, 285–288.

Robinson, T.L., Snow-Harter, C., Taaffe, D.R., et al. (1995) Gymnasts exhibit higher bone mass than runners despite similar prevalence of amenorrhea and oligomenorrhea. *Journal of Bone and Mineral Research* 10, 26–35.

Roupas, N.D. & Georgopoulos, N.A. (2011) Menstrual function in sports. *Hormones* 10(2), 104–116.

Schlabach, G. (1994) Carbohydrate strategies for injury prevention. *Journal Athletic Training* 29, 245–254.

Short, S.H. & Short, W.R. (1983) Four-year study of university athletes' dietary intake. *Journal of the American Dietetic Association* 82, 632–645.

Smith, A.D. (1996) The female athlete triad: causes, diagnosis, and treatment. *Physician and Sportsmedicine* 24, 67–76.

Soric, M., Misigoj-Durakovic, M., & Pedisic, Z. (2008) Dietary intake and body composition of prepubescent female aesthetic athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 18(3), 343–354.

Sundgot-Borgen, J. (1994) Eating disorders in female athletes. *Sports Medicine* 17, 176–188.

Theodoropoulou, A., Markou, K.B., Vagenakis, G.A., et al. (2005) Delayed but normally progressed puberty is more pronounced in artistic compared with rhythmic elite gymnasts due to the intensity of training. *Journal of Clinical Endocrinology & Metabolism* 90(11), 6022–6027.

Theintz, G.E., Howald, H., Weiss, U., et al. (1993) Evidence for a reduction of growth potential in adolescent female gymnasts. *Journal of Pediatrics* 122, 306–313.

Tournis, S., Michopoulou, E., Fatouros, I.G., et al. (2010) Effect of rhythmic gymnastics on volumetric bone mineral density and bone geometry in premenarcheal female athletes and controls. *Journal of Clinical Endocrinology and Metabolism* 95, 2755–2762.

Zanker, C.L., Gannon, L., Cooke, C.B., et al. (2003) Differences in bone density, body composition, physical activity, and diet between child gymnasts and untrained children 7–8 years of age. *Journal of Bone and Mineral Research* 18(6), 1043–1050.

Zanker, C.L., Osborne, C., Cooke, C.B., et al. (2004) Bone density, body composition and menstrual history of sedentary female former gymnasts, aged 20–32 years. *Osteoporosis International* 15, 145–154.

Chapter 50

Swimming

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Introduction

Swimming is a sport enjoyed at levels from recreational to elite and from age groups to Masters. Swimming has been included in modern Olympics since its inception in 1896, and includes a large number of events on the competition program; remarkable swimmers have won seven (Mark Spitz, Munich 1972) and eight (Michael Phelps, Beijing 2008) gold medals at a single Olympic Games. While the first Olympic events were undertaken in open water, the vast majority of swimming events are now conducted in pools. Open water swimming is a separate endurance sport and was recently incorporated into the Olympic program. Swimming and open water swimming are two of the five aquatic disciplines under the governance of the Federation Internationale de Natation (FINA).

Swimming involves the completion of large volumes of training from an early age to develop (1) biomechanical technique for the different strokes of freestyle, backstroke, butterfly, and breaststroke; (2) racing skills such as starts and turns; and (3) the physiological capability to sustain large power outputs over a duration from 20 seconds to 16 minutes (pool racing) through to several hours (open water racing). The traditional calendar was based on long cycles of training interspersed with several competition blocks involving a relatively pronounced taper and short competition period. The modern program has more opportunities for racing, and a reduced duration of the periods of high training volume. Figure 50.1 illustrates the typical periodization of the swimmer's year with the fluctuations in training volume and intensity of various phases. Significant differences in these characteristics are evident within the 50–100 m "sprint" swimmers, 200–400 m "middle distance" swimmers, and 800–1500 m "distance swimmers." Racing is undertaken in both single day and multiday meets in which a swimmer often competes in several events and may include heats, semi finals, and finals. These characteristics and the culture of swimming produce a number of nutritional challenges.

Energy Requirements

A swimmer's energy requirements are largely determined by the energy cost of his or her training/racing program, body mass (BM), and needs for growth or to alter body composition. Ideally, a swimmer's energy intake should be matched to their requirements. Characteristics of swimming, such as the highly periodized training program and the age range from adolescent to adult, mean that the training volumes and energy requirements vary between swimmers and fluctuate from day to day, over the periodized calendar and over their swimming career. The energy costs of training vary markedly from day to day according to the training intensity/focus of pool workouts and the addition of "dry land" training (resistance training, flexibility work, cross-training). Dietary surveys show variable results for self-reported energy intakes with the

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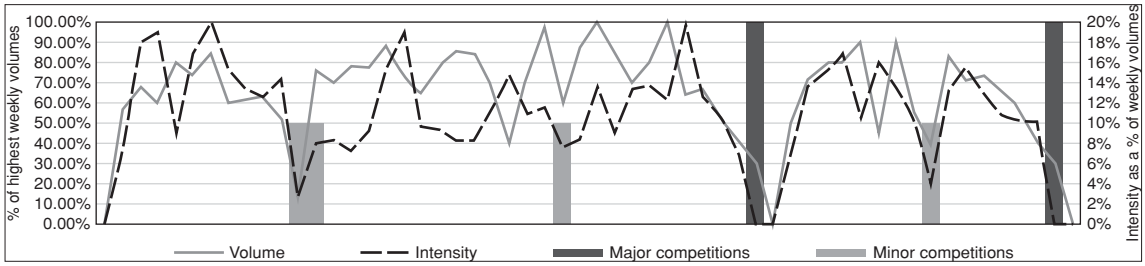

Figure 50.1 Representation of the periodization of training and nutrition in the annual swimming calendar.

typical range of mean values being 15–20 MJ/day and 8–11 MJ/day for males and females, respectively (Burke, 2007). The energy intake of swimmers created great media interest during the 2008 Beijing Olympic Games, when US swimmer Michael Phelps was reported to consume ~12,000 kcal (~50 MJ) per day. This was later corrected to a claim of 32–40 MJ/day (8000–10,000 kcal) (Phelps & Abrahamson, 2008). Even if these figures are estimates of his highest days of intake rather than a true average, they illustrate the potential for high energy needs among athletes with heavy training loads.

There is a potential mismatch between energy intake and expenditure if swimmers struggle to increase energy intake during periods of high expenditure and/or to reduce their intake during periods of inactivity (e.g., injury or off-season) or reduced load (e.g., taper). The available literature on swimmers supports this observation. Although some studies of swimmers have reported energy intakes that matched expected or measured energy expenditures (Almeras et al., 1997; Ousley-Pahnke et al., 2001; Vallieres et al., 1989), Trappe et al. (1997) reported that female swimmers who were undertaking large training volumes (17.5 km/day) incurred an apparent energy deficit of ~10 MJ/day. A longitudinal study of female college swimmers over 13 months showed that their reported energy intakes remained reasonably stable over different training periods, despite differences in the estimated energy expenditure (Almeras et al., 1997). There was a significant energy deficit when training volume was peaking in preparation for competition, while the off-season was associated with a significant increase in body fat (~4 kg); equivalent to the energy cost of the absent training load.

There may be a sex difference in energy requirements and mismatches in energy balance. Male swimmers appear more capable of changing their intake to match fluctuations in training loads. Female swimmers are more likely to report "energy efficiency," energy intakes that are less than their predicted expenditure while remaining weight stable, or less than their male counterparts who undertake similar training volumes. BM differences between male and female swimmers do not totally account for the differences in reported energy intake; for example, male and female swimmers undertaking the same training program reported mean energy intakes of 18.2 MJ/day and 9.6 MJ/day, respectively (Van Handel et al., 1984). This difference was still apparent when corrected for BM (220 kJ/kg vs. 150 kJ/kg), although in other studies differences in energy considerations between sexes are minimized when corrected for lean tissue (Jones & Leitch, 1993).

The apparently suboptimal energy intakes reported by female swimmers may be explained in several other ways. Female swimmers may be more efficient at training, with a lower energy cost of swimming for similar distances and pace due to absolute size, higher body fat, reduced resistance, and better positioning in the water (Zamparo et al., 2011). A final likely explanation for apparently low energy intakes involves the methodology of measurement of energy balance. Underreporting energy intakes is common among female endurance athletes and athletes involved in sports where body image is a large focus.

A more important issue for swimmers is to manipulate energy intake over periods of increased and decreased requirements to maintain suitable levels of energy availability. This is of particular concern since adequate energy availability, defined as the amount of energy intake available to the body once the energy cost of exercise has been taken into account, plays a critical role in health and performance (see Chapter 5). Low energy availability could arise either because of the failure of the swimmer to increase energy intake sufficiently during periods of high volume training or because of an intentional reduction in energy intake to achieve or maintain a loss of body mass/body fat. The pressure and challenges of achieving an ideal body composition will be covered below. The skills to match energy intake to energy requirements may be further challenged by contemporary training methods that promote even greater fluctuations in the volume and intensity of daily, weekly, and monthly training plans than in previous times (see Figure 50.1). For example, some swimmers may find daily energy requirements fluctuating from as little as 8 MJ on a rest day to as much as 20 MJ on a day of 2–3 training sessions. Periodizing nutritional intake to match daily variations in requirements will require skills and food literacy to manipulate food intake appropriately.

Achievement of Ideal Body Composition

In all sports, there is a range of specific physique traits favoring good performance, which elite athletes achieve as a result of their genetic endowment and the conditioning effects of training and diet. Swimming performance is based on the ability to generate forward propulsion while minimizing drag through the water, and is aided by a fine balance between muscle mass and appropriate morphology (e.g., surface area and body fat). Sprinters and higher-level swimmers are generally taller and heavier than longer distance swimmers and less successful swimmers, with female long-distance swimmers having significantly higher skinfolds than all other swimmers (Carter & Ackland, 1994). Swimmers typically have higher fat mass than their peers in distance running, cycling, or other high training volume sports (Jang et al., 1987). A certain level of body fat may be useful for the swimmer, enhancing buoyancy and body position in the water, or providing rounded body surfaces that have more favorable drag characteristics than angular protrusions.

Typically, swimming training is associated with increases in lean tissue and reductions in fat mass (Almeras et al., 1997; Anderson et al., 2006; Peterson et al., 2006). This is typically due to the influence of training rather than diet, but often occurs against the background of the physique changes associated with puberty and achieving adulthood. The importance of these physique changes for performance has been investigated in several studies with unclear results. One study found that a reduction in skinfold fat in female swimmers from taper to taper over a 5-year period was positively correlated with performance improvements (Anderson et al., 2006). Siders et al. (1993) found that faster swimming performance was associated with greater standing height, higher fat-free mass, and lower body fat in female swimmers at the beginning of a competition season, but only to fat-free mass and height by the end of the season. Of course, cross-sectional and longitudinal studies of performance and physique in swimming are limited by their inability to identify cause and effect relationships. In the case of observations of the extreme leanness of certain elite swimmers or improvements in performance associated with physique changes, it is hard to distinguish between the effects of body fat levels on performance *per se*, and the influence of the high volume training or dietary commitment that were involved in the achievement of such a physique. It is most likely that the ideal physique is individual to each swimmer, and that each swimmer has a range of desirable physical characteristics within which they train and perform well.

Nevertheless, some themes related to body fatness and swimming deserve mention. The first concerns the supposedly unusual effects of swimming on fat loss goals. It is frequently suggested that swimming is less effective than other types of exercise in promoting a loss of body fat; evidence for this idea is found in the higher body fat levels of swimmers than of other athletes, and the failure of exercise

programs based on swimming to produce substantial weight loss in general community members. Many swimmers and their coaches believe that the key to successful fat loss is to add "dry land" training to the program. There is some evidence to support the concept that swimming creates a different environment for producing an energy deficit. The first element may be that swimmers are more susceptible to overeating after exercise, or less likely to experience the reduction in appetite that often accompanies other forms of high-intensity exercise. Voluntary food consumption may be differentially affected by water immersion *per se* or the thermoregulatory response to swimming in cool water compared with other exercise forms. For example, White et al. (2005) reported that voluntary energy intake increased ~40% after exercise in cold water compared with thermoneutral water or a control environment. Additionally, *ad libitum* food intake increased when a running session was followed by immersion in cold or thermoneutral water (Halse et al., 2011). This suggests that swimmers who attempt to manipulate body composition through energy restriction may benefit from interventions that specifically address hunger and appetite control.

The second observation relates to the longstanding battle faced by many female swimmers regarding body composition. Gain of body fat, whether as an inevitable consequence of adolescence or during off-season, is a source of concern for many female swimmers and their coaches. Clearly, an excessive amount or rate of increase of body fat is detrimental to performance, due to the sudden change in the fluid dynamics and biomechanics of swimming. There is research and anecdotal evidence of weight control phobias among female swimmers, accompanied by disordered eating or pathogenic, weight-loss behaviors (Benson, 1991). A recognized risk factor for the development of disordered eating among athletes is the necessity to wear figure-revealing or skimpy sports clothing (Otis et al., 1997). Clearly, a complex array of factors contributes to the pressure felt by many female swimmers regarding weight and body fat levels. They are a population who should be targeted for sound advice regarding the setting and achievement of long-term goals for ideal physique (see Chapters 28, 41, and 42).

The final comment addresses apparent trends in the culture surrounding body composition in swimming over the past two decades. Over the first part of this period, we observed an increase in the value placed on lower body fat levels, with many successful swimmers exhibiting noticeably leaner and more muscular physiques than their predecessors. However, this situation appeared to reverse with the evolution of the controversial swimsuits which, as a result of full body cover and special textiles, provided buoyancy and compression. These tightly fitting suits allowed swimmers to carry greater mass and higher body fat levels through the water with minimal effects on drag, resulting in a more relaxed attitude by swimmers (and coaches) regarding body fat goals. With these swimsuits now banned, we expect to see a renewed interest in the manipulation of body composition and the avoidance of excessive "loss of shape" in the off-season.

Daily Carbohydrate Requirements

Carbohydrate requirements are elevated by increases in the volume and intensity of training. Interval training in swimming is characterized by high rates of carbohydrate oxidation and substantial depletion of glycogen content in the deltoid muscles of well-trained swimmers (Costill et al., 1988b). The special characteristics of the fuel utilization patterns of the arm (small muscle groups, upper body) are also of interest. Unfortunately, there is insufficient study of the specific fuel requirements of typical swimming workouts to make definitive recommendations for carbohydrate intake. The general guidelines for carbohydrate intake in the everyday training diet (see Chapter 7) recommend high carbohydrate availability for sessions focused on high-intensity activity and high-level performance, especially during a period of high volume training. High carbohydrate availability can be achieved through total daily intake of carbohydrate, varying from 3 to 12 g/kg BM according to the training load, and particularly by the intake of carbohydrate before, during, or after the training session. The benefits of this approach include better maintenance of muscle workloads as well as reduced central or neuromuscular fatigue—an interaction that is of

importance to sports involving highly coordinated technique. There is some evidence to support the benefits of following this principle.

Reilly and Woodbridge (1999) compared the acute effect of small changes in everyday carbohydrate intake on metabolism and performance by well-trained swimmers of typical test sets, including time trials over 100, 200, and 400 yd. A 10% decrease in carbohydrate over 3 days leading into the trials was associated with a reduction in 400 yd swimming performance, whereas a 10% increase in carbohydrate intake improved performance of 100 and 400 yd time trials. A secondary outcome was a shift in the relationship between swimming velocity and blood lactate response. This relationship is traditionally monitored over a training cycle as a sign of the training response, with a lower lactate level for a given swimming speed being considered to indicate a positive training effect. Similarly, the acute intake of a sports drink before a session also changes the lactate–swim velocity curve (Millard-Stafford et al., 2010). This does not undermine the general relationship between fast swimming performances in competition and high levels of lactate production, but shows the limitations of using lactate monitoring without dietary control as an indicator of training performance and prescription.

There is mild support for the benefits of consuming carbohydrate intake immediately before and during a prolonged swimming workout. Triathletes who consumed carbohydrate before a morning time-trial swim achieved a 2.5% improvement in swimming time (range = 24 seconds to 5 minutes), although differences failed to reach statistical significance (Smith et al., 2002). While this study does not mimic the interval-based sessions undertaken by swimmers, it supports the potential benefit of consuming carbohydrate around early morning sessions. Carbohydrate intake (1 g/kg BM) during an interval-training session (~6 km or 2 hours) by collegiate swimmers also failed to produce an overall improvement in training performance, as monitored by mean times achieved during a 10×100 yd test set at the end of the session (O'Sullivan et al., 1994). However, compared with the placebo trial, carbohydrate ingestion prevented a decline in blood glucose concentrations in two swimmers and was associated with better performances in these individuals.

Apart from direct enhancement of performance during the session, there may be other advantages to providing nutritional support during a workout. Prolonged and high-intensity exercise is associated with perturbations to the immune system and the strategies that enhance carbohydrate status may play an important role in maintaining effective immune function in athletes (see Chapter 39). Finally, a study of elite swimmers found that postexercise plasma concentrations of enzymes, often used as markers of muscle damage, were lower when sports drink was consumed during sessions than when water was drunk (Cade et al., 1991). This was attributed to a reduction in exercise-induced muscle catabolism secondary to the sustained availability of muscle carbohydrate.

Theoretically, chronic practices of good fuel support for training sessions should lead to superior outcomes from a block of training. This is an area of debate, however, due to the lack of clear evidence from training studies and the evolving interest in the "train low" model, whereby cellular signaling responses have been shown to increase when exercise is undertaken in an environment of a lack of muscle substrate (Chapter 7). The literature includes an intervention by Costill et al. (1988a) in which the training volume of a squad of collegiate swimmers was suddenly doubled over a 10-day period. While some swimmers ($n = 8$) appeared to tolerate the intervention, a subgroup ($n = 4$) failed to adapt, complaining of muscle fatigue, irritability, and an inability to finish some workouts. The discriminating features of this group were their failure to voluntarily increase total energy and carbohydrate intake (self-reported intake of 5.3 g/kg BM) and reduced muscle glycogen levels at the end of the training period. Meanwhile, the other swimmers automatically matched their new fuel demands with a greater energy and carbohydrate intake (8.2 g/kg BM), maintaining muscle glycogen stores. Performance measurements (maximum power on the swim bench, 2×25 yd freestyle swim with 2–3 minutes recovery interval and swimming efficiency at submaximal pace) did not differ between groups or over the intervention, except for

a reduction in swimming efficiency in the subgroup with the lower carbohydrate intakes (Costill et al., 1988a). Since muscle glycogen content is unlikely to limit performance in a sprint race, it is not surprising that "race" performance was unchanged after the 10 days. However, a longer duration of poor training might eventually diminish the adaptations achieved and lead to an impairment of race performance.

A more conventional crossover intervention investigated male college swimmers over a 9 day-block of twice per day training, while consuming an energy-matched diet with carbohydrate intake of either 6.5 or 12.1 g/kg BM/day (Lamb et al., 1990). This study did not measure muscle glycogen stores, but found no differences between dietary treatments in mean swimming times over a range of distances. There are several ways to interpret these results: either the moderate carbohydrate diet was sufficient to provide high carbohydrate availability for the training program of the swimmers, that the swimmers were able to adapt to less than optimal refueling or that the performance measurements were inadequate to detect differences that might ultimately be important. Unfortunately, the design of most training/nutrition studies is either flawed or unable to provide the information that would allow discrimination between these potential explanations.

The concept of the "train low" model, in which some training sessions are deliberately undertaken with low glycogen concentrations or after an overnight fast to enhance the cellular signaling response, is covered in Chapters 7 and 13. Here it was noted that although there is evidence of greater increases in muscle enzymes and other proteins with "train low" protocols, to date this has not been shown to lead to enhance performance outcomes in well-trained individuals. Although further research on the concept of deliberately training with low carbohydrate availability is warranted, it should be noted that (1) the current "train low" protocols have involved exposure of some rather than all training sessions to this environment, and (2) this has been achieved by manipulating the timing of carbohydrate intake around training sessions, or the time-tabling of two sessions in close succession rather than by implementation of a low or restricted carbohydrate intake.

Protein Requirements

Protein has reemerged as an area of high interest in sports nutrition. Dietary surveys of swimmers typically report intakes of >1.2 to 1.6 g/kg/day (Burke, 2007), which is above the suggested increase in total daily protein requirements associated with sport. However, the current research focuses on the optimum timing, type, and amount of protein to promote adaptation and recovery from the specific stimulus of each exercise bout. Swimming is a challenging sport since the periodized training program includes endurance exercise, intermittent high-intensity sprints, and resistance training. Each stimulates the synthesis of specific proteins, although undertaking mixed training stimuli in close proximity—as often occurs in the swimmer's training program (e.g., a pool session undertaken before or after a resistance workout)—may interfere with the net training stimulus (Chapter 11). Nevertheless, elite female swimmers who undertook a combination of an interval-training session and resistance workout showed an effective increase in muscle protein synthesis (Tipton et al., 1996). Therefore, swimmers should follow current guidelines to consume 20–25 g of high-quality protein, soon after a workout (or race) to maximize the early protein synthetic response to the exercise stimulus.

Further studies are needed to discern how to spread protein intake over the rest of the day to continue to take advantage of the period of enhanced protein synthesis. Until new data are available, it makes sense to consume worthwhile amounts of protein at each meal and snack. Western eating patterns do not typically achieve such a spread, with much of dietary protein intake being concentrated in the evening meal. Snacks, fortified drinks (e.g., fruit smoothies and milk shakes), and light meals that combine carbohydrate needs with such protein sources should form the basis of everyday eating in the earlier part of the day. Specialized sports foods,

such as liquid meal supplements and simple whey powders, may also be valuable. Their extra expense can be justified when they provide a practical solution to an eating challenge; however, most of the claims of special, muscle-enhancing properties are unjustified.

Micronutrient Requirements

The core characteristics of a diet high in micronutrients are a moderate- to high-energy intake and a wide variety of nutrient-rich foods. Swimmers are reported to consume high-energy intakes, and are not typically considered to be at risk of low micronutrient intakes. Reports of low micronutrient status are limited to female swimmers and concern iron (Farajain et al., 2004; Kabasakalis et al., 2007; Vallieres et al., 1989; Van Handel et al., 1984) and calcium (Farajain et al., 2004; Paschoal & Amancio, 2004). Low intakes were typically linked to restrained eating practices and limited food variety. In one study of female swimmers (Peterson et al., 2006), the improvement in the quality of dietary choices over a season was associated with increased vitamin and mineral intakes and an improved iron status. By ensuring appropriate food choices and ensuring adequate energy availability, good micronutrient status can be achieved.

Vitamin D insufficiency may be an issue for swimmers who train indoors or in environments where exposure to UVB radiation is minimal. One-third of a group of Israeli swimmers who trained indoors were found to have 25(OH) vitamin D levels below 30 ng/ml, a level considered to be insufficient for optimal health and performance (Constantini et al., 2010). Similarly, swimmers were among a group of indoor-training, American athletes who were found to have significantly lower vitamin D levels than athletes who trained outdoors (Halliday et al., 2011). Therefore, swimmers are encouraged to monitor their vitamin D status and seek appropriate interventions to enhance stores if they are found to be insufficient (Chapter 20). This may require dietary supplementation as dietary sources of vitamin D are generally insufficient to correct deficiencies (Chapter 20).

Competition Nutrition

Nutrition for racing encompasses two major themes: addressing the nutrition-related factors that underpin fatigue in the swimmers' specific event or competition schedule, as well as managing the practical challenges associated with the competition lifestyle. The practical challenges include achieving nutritional requirements in a foreign environment and while traveling to get there (Chapters 34 and 36).

As outlined earlier, swimmers typically undertake a pronounced training taper prior to major events, with a substantial reduction in the volume of training, and often the number of sessions undertaken. The energy expenditure of racing (which includes the warm-up and warm-down) is also considerably less than habitual training commitments. It can be difficult to adjust energy intake to the reduced energy requirements, especially in an environment in which most factors promote eating more than usual (e.g., dining hall, buffet, and room service eating). Many swimmers also increase their social eating activities to fill the vacuum left by the absence of training commitments. It is not unusual for a swimmer to gain substantial amounts of body fat during the taper and racing phase; therefore, each swimmer should be aware of their changing nutrient and energy needs as well as the special risks associated with the competition environment.

Race Nutrition for Pool Swimmers: The Multiday Meet

Although there are different competition formats, the typical swimming competition involves a multiday meet with some competitors undertaking one or more races in a session. The competition program for the major meets such as World Championships or Olympic Games has been extended to 8 days, with morning heats, evening semi finals, and finals. Although the extended schedule might appear to reduce the nutritional demands of racing, it has presented many swimmers with the opportunity to tackle more events across multiple strokes and distances. As an extreme example, in winning eight gold medals at the Beijing Olympic Games, Michael

Phelps raced 20 times, including three races on each of the 5 days.

Adequate carbohydrate and fluid levels are important for optimum performance but are not likely to be limiting for a single event if adequate prerace preparation and postrace recovery between races have taken place. However, the repetition of swimming multiple races in a session can challenge recovery strategies, especially when sessions finish late at night due to commitments to the media or doping control. In addition, the accumulated energy cost of warming up, racing, and swimming down can become significant. This calls for a planned approach to race eating, especially when the athlete is competing in events away from his or her home base.

The daily routine should start with a carbohydrate-rich, prerace meal, chosen from familiar and comfortably digested foods that have been trialled in previous competitions. Typically, swimmers consume a prerace breakfast 1–3 hours before commencing warm-ups. During and after warm-ups, and in recovery between events on the same program, carbohydrate-containing drinks (e.g., sport drinks) and light foods (sport bars, fruits) are consumed to maintain body carbohydrate supplies and prevent hunger. Carbohydrate–protein rich choices (e.g., liquid meal supplements) consumed after the race can promote repair and adaptation, and in the case where carbohydrate intake is suboptimal, promote glycogen resynthesis (see Chapter 7). The traditional pattern of swimmers who compete in an evening session is to consume a carbohydrate-rich lunch after the morning heats, before resting in the afternoon. A light snack is typically consumed before returning to the evening finals session, and the session is followed by a dinner to meet recovery goals of refueling, repairing muscle damage, and rehydrating. Quick access to food after sessions should be arranged, both to provide rapid access to key recovery nutrients and to maximize the time available for rest and sleep. Swimmers should pay particular attention to the use of supplements or foods containing caffeine (see Supplements and Sports Foods section below). Although little evidence exists to link one poor night of sleep to reduced performance on the subsequent day, the repeated use of caffeine over a multiple day competition can lead to accumulated sleep loss, which reduces performance toward the end of the meet, or to a cyclical misuse of sleeping aids followed by more caffeine intake.

Race Nutrition for Open Water Swimming

Open water swimming was first included on the FINA World Championship program in 1991, and a 10-km open water event was added to the 2008 Beijing Olympic schedule. While training nutrition requirements are similar to those of distance pool swimmers, open water swimmers may need to develop and practice strategies to consume fluid and food during their event. Competition nutrition strategies become more important with the increasing duration of the open water event. Since there are no studies of nutritional practices for open water swimming, current recommendations are based on the evidence-based guidelines for other sporting events of similar duration.

For the 5-km open water event, completed in ~1 hour, swimmers should focus on maximizing carbohydrate and fluid stores prior to racing, especially in warm waters. Swimmers undertaking the 10 km event maximize prerace glycogen stores (see Chapters 7 and 8) and supplement with carbohydrate intake during the race at rates of up to 60–90 g of carbohydrate per hour from multiple carbohydrate sources (see Chapter 9). Pre- and during event caffeine intake may also enhance performance (see Chapter 25). Official feeding pontoons are positioned during open water races to offer opportunities for refueling and rehydration strategies. However, some swimmers also carry their own supplies of carbohydrate sources (e.g., carbohydrate gels) to allow them to feed at intervals that are independent of these feed stations and thus gain a tactical advantage. Hydration and thermal factors may become important in some open water races, with those conducted in warm waters (e.g., 29–31°C) causing a concern for safety and performance. Fluid intake during such races is important, and strategies such as precooling via the consumption of ice slurries prior to racing may be beneficial (see Chapter 35).

Supplements and Sports Foods

Swimmers are regularly reported to have high rates of supplement use. Data collected at doping control at the Sydney 2000 Olympic Games showed that swimming was included among the four sports reporting the greatest prevalence of supplement use (Corrigan & Kazlauskas, 2003). Similarly, swimming was in the top three sports for the use of vitamins and nutritional supplements at both the Atlanta and Sydney Olympics among Canadian athletes (Huang et al., 2006). Baylis et al. (2001) looked specifically at the supplement practices of a group of high-level swimmers and reported that 99% of swimmers used some form of dietary supplement, with 94% using at least one nonfood-based dietary supplement. Polysupplementation (use of numerous products often containing a range of overlapping ingredients) is also common among swimmers, with one swimmer reporting the use of 27 different supplements.

The pros and cons of supplement and sports food use are discussed in Chapter 23, while a summary of the specific products that may have justified use in swimmers is summarized in Table 50.1. These include sport foods that address nutritional goals of swimming in a practical form as well as products providing ingredients claimed to directly enhance performance. Even when there is an evidence base for the use of such products, the swimmer is advised to be aware of the appropriate situations and protocols of use, and to take into account the "cost" of products in terms of financial outlay and the small but real risk of an inadvertent doping outcome.

Summary

Swimming provides considerable nutritional challenges in both the training and competition phases. Energy and carbohydrate needs can vary greatly between swimmers and across the various components of a week, macrocycle, annual program, or swimming career. To achieve the best outcomes, swimmers need to learn the skills

Table 50.1 Sport foods and supplements that are of likely benefit to swimmers

	Product	Comment
Use in achieving documented nutrition goals	Sport drinks	• Useful to refuel and rehydrate during prolonged workouts and to rehydrate after the session. • Useful to provide fluid and fuel replacement during open water swimming in warm water when fluid requirements are increased.
	Sport gels/sports confectionery	• Convenient and compact carbohydrate source that can be carried for intake during open water swimming or used during prolonged pool workouts.
	Sport bars	• Convenient, portable, and easy-to-consume source of carbohydrate, protein, and micronutrients for prerace meal or postexercise recovery. • Convenient and portable form of energy and nutrients that can help meet high energy needs, especially to support resistance training program or growth. • Convenient and compact source of energy and nutrients for the traveling swimmer.
	Liquid meal supplements	• Convenient, portable, and easy-to-consume source of carbohydrate, protein, and micronutrients for post-workout or postrace recovery. • Low-bulk and practical form of energy and nutrients that can help meet high energy needs, especially to support resistance training program or growth. • Well-tolerated, pre-event meal that can be consumed to provide a source of carbohydrate quite close to the start of a race or workout; liquid supplements seem to be better tolerated than solid food by some swimmers with high risk of gastrointestinal problems. • Convenient and compact source of energy and nutrients for the traveling swimmer.

(continued)

Table 50.1 *(continued)*

	Product	Comment
Use in achieving documented nutrition goals *(continued)*	Whey protein powder	• Convenient form of high-quality protein that can be used for post-training or postrace recovery. Can be used in situations where carbohydrate for refueling is not a priority, where a food form of carbohydrate is consumed at the same time, or to fortify a drink or meal that is otherwise suboptimal in protein content.
	Multivitamin and mineral supplements	• Supplemental source of micronutrients for traveling when food supply is not reliable. • Supplemental source of micronutrients during prolonged periods of energy restriction (especially female swimmers).
Strong potential for ergogenic benefit	Caffeine	• There is evidence that prerace caffeine supplementation may enhance swimming performance (Collomp et al., 1992; Macintosh & Wright, 1995), but further sport-specific studies are needed to investigate the range of swimming events and the range of protocols (e.g., the dose and timing of prerace caffeine intake) that are effective for swimming events. There is some evidence from endurance sports protocols that small to moderate doses of caffeine (1–3 mg/kg) are as effective as larger doses (5–6 mg/kg) in achieving benefits and may reduce risk of side effects, such as tremor and anxiety, that can affect technique (see Chapter 25). Because swimming events are performed as a series of heats and finals, an additional benefit of finding the lowest caffeine dose at which performance enhancement occurs is to reduce the effect of pre-event caffeine on postrace recovery and sleep patterns. Caffeine may be consumed in cola and energy drinks or as an ingredient in some sport products (e.g., some gels).
	Bicarbonate loading and other buffering strategies	• The acute use of bicarbonate to increase extracellular buffering capacity (e.g., 300 mg/kg BM bicarbonate, 1–2 hour prerace) might enhance the performance of swimming events lasting 2–8 minutes (200–800 m swimming events) via increased tolerance to the production of H^+ ions via anaerobic glycolysis (see Chapter 26). Further field studies are needed with high-level swimmers since the few available studies show both benefits (Pruscino et al., 2008; Siegler & Gleadall-Siddall, 2010) and the lack of effect (Joyce et al., 2012). Swimmers who intend to load for a series of events over a day or consecutive days (e.g., heats and finals) should experiment with a lower dose for subsequent races in view of residual increase in buffering capacity from earlier doses. Chronic supplementation with B-alanine to increase muscle carnosine, an intracellular buffer, may provide an alternative or additive strategy to support training adaptations and race performance (see Chapter 26).
	Creatine	• Studies show that creatine loading enhances the performance of exercise involving repeated high-intensity work bouts with short recovery intervals (see Chapter 24). The most likely benefits for swimmers come from using creatine in the training phase to enhance training adaptations to interval and resistance training. However, while there are studies that report an enhancement of the performance of such sessions in highly trained swimmers (Peyrebrune et al., 1998; Theodorou et al., 1999), it is often hard to find definitive proof that this leads to superior "race" performance after a training block (Peyrebrune et al., 2005; Selsby et al., 2003). A full summary of the literature on creatine supplementation in swimming is provided in Burke, 2007.

Source: Adapted from Burke (2007).

to vary their food intake accordingly, especially to provide nutritional support before, during, and after workouts. The achievement of desired physique can be difficult for some swimmers, particularly females. Competition nutrition requires a special eating plan to promote recovery between races; swimmers who race in multiple events face special logistical challenges. Although pool swimming does not provide a need or opportunity for intake during the event, open water swimming is an event in which fluid and carbohydrate intake can enhance performance. Some supplements and sports foods can be used by swimmers to achieve their nutritional goals and optimal performance. Many of the recommendations for swimmers are based on evidence gained from research on other sports with similar characteristics. More swimming-centered studies are needed to provide specific support for these guidelines or to evolve new practices.

References

Almeras, N., Lemieux, S., Bouchard, C., et al. (1997) Fat gain in female swimmers. *Physiology and Behavior* 61, 811–817.

Anderson, M.E., Hopkins, W.G., Roberts, A.D., et al. (2006) Monitoring seasonal and long-term changes in test performance in elite swimmers. *European Journal of Sport Science* 6(3), 145–154.

Baylis, A., Cameron-Smith, D., & Burke, L.M. (2001) Inadvertent doping though supplement use by athletes: assessment and management of the risk in Australia. *International Journal of Sport Nutrition and Exercise Metabolism* 1, 365–383.

Benson, R. (1991) Weight control among elite women swimmers. In *Eating Disorders among Athletes: Theory, Issues, and Research* (ed. D.R. Black), pp. 97–109. Association for the Advancement of Health Education, National Association for Girls and Women in Sport, Associations of the American Alliance for Health, Physical Education, Recreation and Dance, Reston, VA.

Burke, L.M. (2007) Swimming and rowing. In *Practical Sports Nutrition*, pp. 141–167. Human Kinetics Publishers, Champaign, IL.

Cade, J.R., Reese, R.H., Privette, R.M., et al. (1991) Dietary intervention and training in swimmers. *European Journal of Applied Physiology and Occupational Physiology* 63, 210–215.

Carter, J.E.L. & Ackland, T.R. (1994) *Kinanthropometry in Aquatic Sports: A Study of World Class Athletes*. Human Kinetics Publishers, Champaign, IL.

Collomp, K., Ahmaidi, S., Chatard, J.C., et al. (1992) Benefits of caffeine ingestion on sprint performance in trained and untrained swimmers. *European Journal of Applied Physiology* 64, 377–380.

Constantini, N.W., Arieli, R., Chodick, G., et al. (2010) High prevalence of vitamin D insufficiency in athletes and dancers. *Clinical Journal of Sport Medicine* 20(5), 368–371.

Corrigan, B. & Kazlauskas, R. (2003) Medication use in athletes selected for doping control at the Sydney Olympics (2000). *Clinical Journal of Sports Medicine* 13, 33–40.

Costill, D.L., Flynn, M.G., Kirwan, J.P., et al. (1988a) Effects of repeated days of intensified training on muscle glycogen and swimming performance. *Medicine & Science in Sports & Exercise* 20, 249–254.

Costill, D.L., Hinrichs, D., Fink W.J., et al. (1988b) Muscle glycogen depletion during swimming interval training. *Journal of Swimming Research* 4, 15–18.

Farajian, P., Kavouras, S.A., Yannakoulia, M., et al. (2004) Dietary intake and nutritional practices of elite Greek aquatic athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 14, 574–585.

Halliday, T.M., Peterson, N.J., Thomas, J.J., et al. (2011) Vitamin D status relative to diet, lifestyle, injury, and illness in college athletes. *Medicine & Science in Sports & Exercise* 43, 335–343.

Halse, R.E., Wallman, K.E., & Guelfi, K.J. (2011) Postexercise water immersion increases short-term food intake in trained men. *Medicine & Science in Sports & Exercise* 43, 632–638.

Huang, S.H., Johnson, K., & Pipe, A.L. (2006) The use of dietary supplements and medications by Canadian athletes at the Atlanta and Sydney Olympic Games. *Clinical Journal of Sport Medicine* 16, 27–33.

Jang, K.T., Flynn, M.G., Costill, D.L., et al. (1987) Energy balance in competitive swimmers and runners. *Journal of Swimming Research* 3, 19–23.

Jones, P.J. & Leitch, C.A. (1993) Validation of doubly labeled water for measurement of caloric expenditure in collegiate swimmers. *Journal of Applied Physiology* 74(6), 2909–2914.

Joyce, S., Minahan, C., Anderson, M., et al. (2012) Acute and chronic loading of sodium bicarbonate in highly trained swimmers. *European Journal of Applied Physiology* 112, 461–469.

Kabasakalis, A., Kalitsis, K., Tsalis, G., et al. (2007) Imbalanced nutrition of top-level swimmers. *International Journal of Sports Medicine* 28, 780–786.

Lamb, D.R., Rinehardt, K.F., Bartels, R.L., et al. (1990) Dietary carbohydrate and intensity of interval swim training. *American Journal of Clinical Nutrition* 52(6), 1058–1063.

MacIntosh, B.R. & Wright B.M. (1995) Caffeine ingestion and performance of a 1,500-metre swim. *Canadian Journal of Applied Physiology* 20, 168–177.

Millard-Stafford, M.L., Brown, M.B., & Snow, T.K. (2010) Acute carbohydrate ingestion affects lactate response in highly trained swimmers. *International Journal of Sports Physiology and Performance* 5, 42–54.

O'Sullivan, S., Sharp, R.L., & King, D.S. (1994) Carbohydrate ingestion during competitive swimming training. *Journal of Swimming Research* 10, 35–40.

Otis, C.L., Drinkwater, B., Johnson, M., et al. (1997) American College of Sports Medicine position stand. The female athlete triad. *Medicine & Science in Sports & Exercise* 29, i–ix.

Ousley-Pahnke, L., Black, D.R., & Gretebeck, R.J. (2001) Dietary intake and energy expenditure of female collegiate swimmers during decreased training prior to competition. *Journal of the American Dietetic Association* 101, 351–354.

Paschoal, V.C.P. & Amancio, O.M.S. (2004) Nutritional status of Brazilian elite

swimmers. *International Journal of Sport Nutrition and Exercise Metabolism* 14, 81–94.
Petersen, H.L., Peterson, C.T., Reddy, M.B., et al. (2006) Body composition, dietary intake, and iron status of female collegiate swimmers and divers. *International Journal of Sport Nutrition and Exercise Metabolism* 16, 281–295.
Peyrebrune, M.C., Nevill, M.E., Donaldson, F.J., et al. (1998) The effects of oral creatine supplementation on performance in single and repeated sprint swimming. *Journal of Sports Sciences* 16, 271–279.
Peyrebrune, M.C., Stokes, K., Hall, G.M., et al. (2005) Effect of creatine supplementation on training for competition in elite swimmers. *Medicine & Science in Sports & Exercise* 37, 2140–2147.
Phelps, M. & Abrahamson, A. (2008) *No Limits: The Will to Succeed*, pp. 161. Simon & Shuster, Sydney, Australia.
Pruscino, C.L., Ross, M.L., Gregory, J.R., et al. (2008) Effects of sodium bicarbonate, caffeine and their combination on repeated 200-m freestyle performance. *International Journal of Sport Nutrition and Exercise Metabolism* 18, 116–130.
Reilly, T. & Woodbridge, V. (1999) Effects of moderate dietary manipulations on swim performance and on blood lactate-swimming velocity curves. *International Journal of Sports Medicine* 20(2), 93–97.
Selsby, J.T., Beckett, K.D., Kern, M., et al. (2003) Swim performance following creatine supplementation in Division III athletes. *Journal of Strength and Conditioning Research* 17, 421–424.
Siders, W.A., Lukaski, H.C., & Bolonchuk, W.W. (1993) Relationships among swimming performance, body composition and somatotype in competitive collegiate swimmers. *Journal of Sports Medicine and Physical Fitness* 33(2), 166–171.
Siegler, J.C. & Gleadall-Siddall, D.O. (2010) Sodium bicarbonate ingestion and repeated swim sprint performance. *Journal of Strength and Conditioning Research* 24, 3105–3011.
Smith, G.J., Rhodes, E.C., & Langill, R.H. (2002) The effect of pre-exercise glucose ingestion on performance during prolonged swimming. *International Journal of Sport Nutrition and Exercise Metabolism* 12, 136–144.
Theodorou, A.S., Cooke, C.B., King, R.F.G.J., et al. (1999) The effect of longer-term creatine supplementation on elite swimming performance after an acute creatine loading. *Journal of Sports Sciences* 17, 853–859.
Tipton, K.D., Ferrando, A.A., Williams B.D., et al. (1996) Muscle protein metabolism in female swimmers after a combination of resistance and endurance exercise. *Journal of Applied Physiology* 81, 2034–2038.
Trappe, T.A., Gastaldelli, A., Jozsi, A.C., et al. (1997) Energy expenditure of swimmers during high volume training. *Medicine & Science in Sports & Exercise* 29, 950–954.
Vallieres, F., Tremblay, A., & St-Jean, L. (1989) Study of the energy balance and the nutritional status of highly trained female swimmers. *Nutrition Research* 9, 699–708.
Van Handel, P.J., Cells, K.A., Bradley, P.W., et al. (1984) Nutritional status of elite swimmers. *Journal of Swimming Research* 1, 27–31.
White, L.J., Dressendorfer, R.H., Holland, E., et al. (2005) Increased caloric intake soon after exercise in cold water. *International Journal of Sport Nutrition and Exercise Metabolism* 15, 38–47.
Zamparo, P., Capelli, C., & Pendergast, D. (2011) Energetics of swimming: a historical perspective. *European Journal of Applied Physiology* 111, 367–378.

Chapter 51

Winter Sports

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Introduction

Winter sports (see Table 51.1) are performed under various environmental conditions (see Table 51.2). Most winter sports are performed outdoors in the cold and at altitude. The endurance sports (e.g., Nordic skiing, biathlon, long-track speed skating) make use of altitude training camps, while others are naturally and intermittently exposed to altitude (e.g., Alpine skiing, freestyle, snowboard). Exposure to cold also varies in winter sport athletes. It is not uncommon for winter sport athletes to train and compete in temperatures well below 0°C. Both cold and altitude increase the need for energy, carbohydrate, and fluid (Askew, 1995; Butterfield, 1999; Vallerand et al., 1995). In addition, altitude can result in reduced appetite and put an athlete at risk for energy deficiency which may lead to muscle loss (Kayser, 1992). Athletes exposed to altitude may also need to adjust iron intake due to the enhanced red blood cell turnover and increased need for iron.

Several winter sports are indoor sports, and while temperatures vary, there has been a trend for warmer indoor conditions (range 5–10°C). Indoor environments may, therefore, lead to higher sweat rates especially if athletes wear heavy equipment (Palmer & Spriet, 2008). In addition, indoor environments may burden respiratory systems not only due to dry air but also due to air pollution. Those working in nutrition with winter sport athletes should evaluate each winter sport and environmental condition separately and focus on the most important nutritional issues.

The next section will provide a brief introduction to the different winter sports, emphasizing the major nutritional issues in each. A full review of each winter sport is beyond the scope of this chapter and the reader is referred to several reviews (Cox et al., 1995; Ferguson, 2010; Foster & De Koning, 1999; Platzer et al., 2009; Rusko, 2003; Sands et al., 2005). The chapter will conclude with a combined discussion on nutritional issues common among all winter sports.

Nordic Skiing (Cross-country Skiing, Biathlon, Nordic Combined, and Ski Jumping)

Each of the Nordic events is unique (see Table 51.2). Cross-country skiing, biathlon, and Nordic combined have a strong endurance component, and thus, rely similarly on a high endurance capacity as other endurance athletes (Rusko, 2003). One exception to the prolonged cross-country skiing events is the sprint. While still characterized by the need for good endurance, the sprint requires greater strength and anaerobic power, and this is visible in the physique of the athletes (Stöggl et al., 2010). Both cross-country skiing and biathlon have sprint events. Biathlon combines cross-country skiing with shooting: distances range from 7.5 to 12.5 km with 2–4 sessions of shooting, involving five shots

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Table 51.1 Winter sports, their international organizations and Olympic events

Sport	International federation	Olympic events
Cross-country skiing	FIS	(W) 1.5-km Sprint, 1.5-km Team Sprint, 10-km Individual Start, 30-km Mass Start; 4 × 5-km Relay (M) 1.5-km Sprint, 1.5-km Team Sprint, 15-km Individual Start, 50-km Mass Start; 4 × 10-km Relay
Biathlon	IBU	(W) 7.5 km + 2× Shooting, 15 km + 2× Shooting, 10 km Pursuit + 4× Shooting
Nordic combined	FIS	4 × 6-km Relay + 2× Shooting, 12.5-km Mass Start + 4× Shooting (M) 10 km + 2× Shooting, 20 km + 2× Shooting, 12.5-km Pursuit + 4× Shooting, 4 × 6-km Relay + 2× Shooting, 15-km Mass Start + 4× Shooting (M) 10 km + Individual K-90, 10 km + Individual K-120, 5-km Team Relay + K-120
Ski jumping	FIS	(M, W) K-90, K-120, Team
Alpine skiing	FIS	(M, W) Giant Slalom, Slalom, Super Giant Slalom, Downhill, Super Combined
Freestyle skiing	FIS	(M, W) Aerials, Moguls, Ski Cross
Snowboarding	FIS	(M, W) Parallel Giant Slalom, Halfpipe, Snowboard Cross
Long-track speed skating	ISU	(W) 500 m, 1000 m, 1500 m, 3000 m, 5000 m, Team Pursuit (M) 500 m, 1000 m, 1500 m, 5000 m, 10,000 m, Team Pursuit
Short-track speed skating	ISU	(W) 500 m, 1000 m, 1500 m, 3000 m Relay (M) 500 m, 1000 m, 1500 m, 5000 m Relay
Ice hockey	FIH	(W) 8 Teams; (M) 12 Teams
Figure skating, singles	ISU	(M, W) Compulsory Short and Free Skate Programs
Figure skating, ice dance	ISU	(M, W) Compulsory, Original, Free Dance Programs
Figure skating, pairs	ISU	(M, W) Compulsory Short and Free Skate Programs
Bobsled	FIBT	(W) 2-Person; (M) 2-Man and 4-Man (4 heats over 2 days)
Skeleton	FIBT	(M, W) Individual Events (4 heats over 2 days)
Luge	FIL	(M, W) Singles Events (4 heats over 2 days); Doubles Event (2 heats over 2 days)
Curling	WCF	(W) Tournament; (M) Tournament 10 teams each

W, women; M, men; FIS, International Federation of Skiing; IBU, International Biathlon Union; ISU, International Skating Union; FIH, International Hockey Federation; FIBT, International Bobsleigh and Tobogganing Federation; FIL, International Luge Federation; WCF, World Curling Federation.

each round. Biathlon skiing distances are typically shorter (except for the sprint) than cross-country skiing events, but shooting slows down the time and missed targets require an athlete to ski a penalty distance. Physiologically, opposing demands challenge the biathlete: engaging in hard physical exercise, while performing precision shooting activity (Lakie, 2010).

Ski jumping involves jumping off two different take-off ramps (normal and large hill). While ski jumping is categorized as a strength and power sport, these athletes are not as muscular (Rankinen et al., 1998). In fact, the physique of a ski jumper resembles more that of a cross-country skier, and this is mainly because body mass affects aerodynamics and lighter athletes may achieve longer jumps (Müller et al., 2006). Nordic combined athletes are challenged by both, cross-country skiing and ski jumping, and each sport's unique physical demands. All Nordic sports are weight-sensitive sports, increasing the risk for underfueling and eating disorders (Meyer & Parker-Simmons, 2009).

Table 51.2 Winter sport environments

Environment	Factors	Sports
Glaciers Intermittent exposure Live high/train high	Altitude (2600–3800 m) Cold (–25°C to +5°C)	Cross-country skiing Biathlon Nordic combined Alpine skiing Freestyle skiing Snowboarding
Northern latitudes	Altitude (500–2000 m) Cold (–25°C to +5°C)	All
Live high train low	Altitude (live at 2000–2500 m; Train at 1500 m) Cold (–25°C to +5°C)	Cross-country skiing Biathlon Nordic combined Speed skating
Indoor ice rink	Altitude (variable) Cold (0°C to –10°C) Lack of daylight Air quality	Figure skating Ice hockey Speed skating Curling
Summer venue	Altitude (variable) Suits (wet/dry)	Freestyle aerials Snowboarding Ski jumping Nordic combined

Nutrition Issues

Cross-country Skiing and Biathlon Cross-country skiers' energy expenditure is very high, ranging from 950 to 1200 kcal for a typical 15-km race, with higher energy demands for the 50-km race (3100–3600 kcal), and slightly more than 2000 kcal for a 30-km race (Ekblom & Bergh, 2000). Energy expenditure rates are also high during intense training in men (~6070 to 8300 kcal/day) and women (~3600 to 4800 kcal/day) using the doubly labeled water technique (Sjödin et al., 1994). Therefore, the major nutritional concern of cross-country skiers is to meet these high energy demands by adjusting energy intake to high-intensity training loads and long-duration events. While biathletes have similarly high training loads as cross-country skiers, their competitive events are shorter. Nevertheless, biathletes combine the difficult task of endurance performance and shooting precision. Thus, adequate energy and carbohydrate availability needs to be emphasized in these athletes not only to maintain carbohydrate oxidation rates in muscle but also to ensure stable blood glucose levels (Meyer & Parker-Simmons, 2009).

That carbohydrates are important for cross-country skiers is illustrated by the 30–40% reduction in muscle glycogen levels after a typical 15-km race and 100% depletion after a 50-km race (Rusko, 2003). Carbohydrate recommendations for intense training should be set according to current guidelines (Burke et al., 2011) and range from 6 to 10 g/kg/day or higher for intense training. This requires frequent eating, adequate pretraining and event fueling, and timely recovery nutrition (Meyer & Parker-Simmons, 2009). To maximize glycogen stores for racing, athletes should prepare with a 24- to 72-hour carbohydrate loading regimen to optimize endurance performance in races that may be limited by carbohydrate availability (Burke et al., 2011; see Chapters 7 and 8). In addition, training or competition exceeding 1–2 hours should be adequately fueled by carbohydrate supplementation using warm sport drinks in insulated containers (Meyer & Parker-Simmons, 2009). An ingestion

rate of 30–60 g/h, and possibly up to 90 g/h during long events is recommended (Jeukendrup, 2011; see Chapter 9). Athletes trying to lose body mass or maintain a very lean body composition should not attempt to restrict calories during training at altitude, as side effects might compromise physiological adaptation (Barnholt et al., 2006) and lead to glycogen depletion, fatigue, muscle loss, and suppressed immune function. Matching energy intake to energy expenditure can minimize body mass/muscle loss at altitude (Butterfield, 1999), and carbohydrate supplementation can assist in maintaining favorable performance effects during altitude exposure (Fulco et al., 2005). A new addition to Nordic skiing is the sprint event. This event requires more strength and power (Stöggl et al., 2010) and may benefit from a different nutritional approach.

Fluid needs of Nordic skiers vary by intensity and environmental condition. In cross-country skiers, sweat losses are estimated to range between 2% and 3% body weight loss after a 15–30 km skiing effort (Ekblom & Bergh, 2000). While fluid intake during a 15-km race may not be needed, fluid replacement in the form of a sport drink has been shown to maintain fluid balance and plasma volume, with a concomitant reduction in urine volume during prolonged skiing (Seifert et al., 1998).

Ski Jumping and Nordic Combined Ski jumping and Nordic Combined are challenging sports because low body mass is a major performance-determining factor (Müller et al., 2006). Fortunately, the newly updated rule, allowing ski lengths at 145% of an athlete's height, requires a body mass index (BMI) of 21 and 20.5 kg/m^2 (with boots and suit) for men and women, respectively. If an athlete's BMI is below this cut-off, they are forced to use shorter skis which may decrease performance. This is due to the importance of body mass on aerodynamic drag (Müller et al., 2006). This new rule will allow redevelopment of the physical characteristics of ski jumpers. Nevertheless, weight management will continue to be a focus area of coaches and athletes. Thus, nutritional work needs to continue to decrease risk in both genders (e.g., low energy availability, nutrient deficiencies, dehydration, and compromised bone mass) (Meyer et al., 2011; Rankinen et al., 1998). Sports dietitians use a low-risk modification of the precompetition diet (e.g., low fiber, low sodium) (Meyer & Parker-Simmons, 2009).

In Nordic Combined, the risk for underfueling and overtraining is substantial and should be of primary focus. During endurance training, fueling tactics as outlined under cross-country skiing will apply, especially focusing on recovery nutrition (Meyer & Parker-Simmons, 2009), while body mass manipulations should not compromise nutritional preparation for competition.

Sweat rates are variable in ski jumpers and Nordic Combined athletes. Ski jumpers train and compete using ramps in the summer. Ramps allow for winter sport-specific training in the summer environment. Rather than on snow, ski jumpers land on artificial turf that is watered to keep the surface smooth when landing. Heavy equipment and warm temperatures lead to greater sweat rates in the summer (Meyer & Parker-Simmons, 2009). Thus, frequent fluid breaks and the use of water and sport drinks should be an integrated summer training/competition strategy.

Alpine Skiing

Alpine skiing events (see Table 51.1) can be divided into speed and technical events. Alpine skiing is a strength/power sport, technically challenging, requiring high-force isometric and eccentric contractions (Ferguson, 2010). Alpine skiers must also exhibit a high level of fitness in other areas: agility and speed, endurance, and a well-developed glycolytic system (Tesch, 1995). Due to their reliance on summer snow, Alpine skiers are intermittently exposed to high altitude (training on glaciers) and need a well-developed oxygen transport system to enhance recovery. Alpine skiers' physiques have changed over the years: now athletes are leaner and more muscular (unpublished observation, N. Meyer). Alpine skiers typically do not qualify as weight-sensitive sports, but the recent emphasis on body mass gain to improve performance could lead to body image issues (Sundgot-Borgen & Torstveit, 2010).

Nutrition Issues

No studies have measured energy expenditure in alpine skiers and estimates are limited by

methodology, missing the additional effect of altitude and cold, as both can increase energy expenditure (Butterfield, 1999). General estimates range from 45 to 55 kcal/kg/day, but these numbers are expected to be higher if the athlete is at altitude, engages in intense training, and/or experiences cold-induced shivering thermogenesis (Meyer et al., 2011). Such extreme situations occur mainly during late autumn when athletes perform time trials in thin suits before snow is available at lower elevations (Meyer & Parker-Simmons, 2009). Both altitude and cold (e.g., shivering thermogenesis) increase the reliance on glucose and glycogen for fuel (Butterfield, 1999; Vallerand et al., 1995).

A day of skiing can reduce muscle glycogen levels by 50% and glycogen repletion during repetitive days of skiing requires a high carbohydrate intake (Tesch, 1995). Low muscle glycogen, in the absence of proper feeding, could lead to early fatigue and increased risk of injuries (Brouns et al., 1986). The high-intensity nature of skiing and blood occlusion with concomitant oxygen desaturation (Szmedra et al., 2001), especially during the giant slalom, are probably the most important factors leading to high lactate levels (Ferguson, 2010). Thus, alpine skiers need to prepare for repetitive days of skiing using a high carbohydrate intake along with properly timed fueling tactics during and after training. To meet daily carbohydrate requirements, alpine skiers should ingest between 6 and 10 g/kg/day (Burke et al., 2011), and use a sport drink or hot, sweetened tea and carbohydrate-rich snacks on the hill. Due to the long travel time to glacier environments, it is most critical to initiate recovery nutrition after the last run. This requires good planning and packing of food and fluid. Alpine skiers should add 15–25 g of protein to a recovery snack as soon as possible after skiing or heavy weight lifting sessions (Phillips & Van Loon, 2011).

Cold and altitude increase fluid loss in alpine skiers. Skiers lose 1% of their body mass if no fluids are ingested over a 2-hour on-snow training session at moderate altitude (Seifert et al., 2000). Glacier training increases the risk of dehydration, especially in female skiers who may consciously restrict fluid intake due to limited accessibility of restroom facilities and decreased drive to drink (Meyer & Parker-Simmons, 2009).

Freestyle Skiing and Snowboarding

Table 51.2 shows the freestyle skiing and snowboard events. Freestyle moguls, freestyle and snowboard cross, and parallel giant slalom (PGS) are most similar to alpine skiing in physiological demand, altitude, and cold exposure. In PGS, runs are shorter than alpine skiing events; however, the top athletes compete in multiple heats, making competition as challenging as training (Meyer et al., 2011). Freestyle events are mostly skill based, using quick energy systems for fuel. Freestyle aerialists are often former gymnasts, bringing enormous acrobatic skill level to the sport. While most of the freestyle disciplines are judging sports, the culture of the sport is different from that of other aesthetic sports. Nevertheless, being lean and muscular is also a focus in these sports. Few data are available on these sports and most are not published in scientific journals.

Nutrition Issues

Mogul Skiing, Freestyle and Snowboard Cross, and Parallel Giant Slalom Major nutrition issues of mogul, cross, and PGS athletes are similar to those of alpine skiers. Mogul skiing is a demanding and injury-prone sport. Most athletes complain of overuse injuries, and thus, fueling strategies should include high carbohydrate intakes when training intensely (6–10 g/kg/day), use of sport drinks during on-snow training, and quick recovery nutrition (with protein) to initiate repair of soft tissue (Meyer & Parker-Simmons, 2009). Freestyle and snowboard cross, along with PGS athletes, compete using multiple heats, which increases energy and carbohydrate requirements on competition day. A mild form of carbohydrate loading and using carbohydrate-rich foods and drinks (e.g., sport drinks, gels, bars) are needed to maintain glucose availability if athletes advance to the next heat (Meyer et al., 2011).

Freestyle Aerials Freestyle aerialists have a unique training pattern in the summer, using water ramps. This training requires more energy than on-snow, due to the climbing of stairs with boots and wet suit. Thus, energy, carbohydrate, and fluid needs are likely higher in the summer (Meyer & Parker-Simmons, 2009). Carbohydrate intake under

such conditions should be at least 6 g/kg/day. In the winter environment, cold-induced shivering may increase energy expenditure although this only occurs when unstable weather interferes with training or competition. Athletes should use warm fluids and keep moving to maintain core temperature. Freestyle aerialists are often former gymnasts and are at risk of restrictive eating. Assessment of the athlete's history related to previous sports and eating disorders can identify at-risk athletes.

Freestyle Snowboarders Halfpipe athletes are acrobats! These athletes do not shy away from extra speed and big air. While energy expenditure is probably lower than that of PGS athletes, energy expenditure will rise if athletes hike back up to the start carrying their snowboard. Especially in the summer months, these athletes may exceed any other winter sport athletes' sweat rates, if they train on snow but use hiking to get back to the start. Snowboarders also use water ramps but have a less demanding return to the start than aerialists. These sports may be at risk of several nutritional shortcomings (poor diet, poor nutrition knowledge) due to a lack of sport science resources, education, and the unique culture of these sports (Meyer & Parker-Simmons, 2009).

Skating Sports (Figure Skating, Speed Skating, and Ice Hockey)

Skating sports vary by discipline (see Table 51.2). Figure skating requires a combination of speed, agility, power, and flexibility (Mannix et al., 1996). Figure skaters also need to be artistic and to some extent acrobatic, especially when attempting quadruple jumps. The continuously evolving and technically difficult programs have resulted in the demand for greater fitness. Figure skating is classified as an aesthetic sport, and the emphasis on a lean physique prevails as does disordered eating (Sundgot-Borgen & Torstveit, 2010; see Chapter 42), despite recent changes in scoring. Of all figure skating disciplines, pair skating remains the one with greatest concerns for low body mass and eating disorders.

Speed skating is divided into long- and short-track disciplines. Short-track speed skating is characterized by a shorter track (~111 m) (see Table 51.1). Athletes race in a pack and race tactics are critical. Short track is unique in that race time and position are both important to advance from heat to heat. Short-track athletes are small, muscular, and agile. Their training is mostly on ice, as so many unique aspects of the sport are best practiced on ice. Due to this, the team environment is highly intense. In addition, athletes strive to be as light and agile as possible, and often restrict energy intake or follow a strict dietary regimen. This can compromise energy levels and increase the risk of illness or injury (unpublished observation, N. Meyer). No data exist on the physiological demands of short track speed skating, except for one recent study on muscle oxygenation, showing a clear left to right leg difference (Hesford et al., 2012). Skaters often hold a very low position and stay on the right leg through the turns. High lactate levels are expected, especially originating from the right versus left leg.

Long-track speed skating is divided into sprint and all-round disciplines (see Table 51.2). Compared to short track, long-track speed skating makes use of a 400 m track. All-round skaters are endurance athletes with a moderately high aerobic capacity, whereas the sprinters are best compared to 400 m sprinters (Foster & De Koning, 1999). While tactics are important for short track, pacing strategies prevail in long-track speed skating. Short distances are mostly anaerobic, but even the 1000 m requires some energy produced through oxidative pathways (Foster & De Koning, 1999). The most remarkable attribute of long-track speed skaters is the low trunk position, a performance-determining factor (Foster & De Koning, 1999). Long-track skaters experience blood occlusion, oxygen desaturation, and extremely high muscle and blood lactate levels (Foster & De Koning, 1999). Finally, long-track speed skaters use altitude training camps and their training loads are very high at key times of the year.

Ice hockey is described as a physically demanding team sport, with high-intensity bouts interspersed with periods of low intensity or rest. Each game consists of three 20-minute periods. Ice hockey is a team sport and contracts both glycolytic and oxidative energy systems (Cox et al., 1995). Today's ice hockey players are leaner and fitter. As with any other team

sport, positions played in ice hockey evoke physiological differences, and thus, nutritional strategies need to be adjusted accordingly.

Nutrition Issues

Figure Skating The main nutritional issue for figure skaters is the marginal energy and nutrient intake relative to increased requirements during training and competition. To date, no studies have measured figure skaters' energy expenditure or glycogen utilization rates. Most research on figure skaters has reported the dietary intakes using dietary records and surveys. Results show that figure skaters do not meet recommended levels for energy and certain nutrients. In fact, figure skaters appear to skip breakfast and underfuel during training (Ziegler et al., 2002). Some research in aesthetic sports suggests that incurring a negative energy balance throughout the day is associated with higher body fat levels (Deutz et al., 2000). According to the intensity and volume of training, figure skaters should consume sufficient carbohydrates throughout the day (3–5 g/kg/day for technical training and 5–7 g/kg/day for intense training and competition) to meet energy and nutrient needs (Burke et al., 2011). Adding a sport drink or sweetened tea during skating could assist in maintaining blood glucose levels and hydration status.

The risk of restrictive eating patterns is high in figure skaters (Sundgot-Borgen & Torstveit, 2010) and attempts should be made to screen all athletes, use an effective referral system, and have an institutional eating disorder policy in place for the purpose of treatment and prevention.

Speed Skating Energy expenditure in long-track speed skaters is high, ranging from approximately 3000 to nearly 6000 kcal/day, depending on the intensity of training (Ekelund et al., 2002). There are no data on short-track speed skaters. Short-track skaters perform up to five high-intensity training sessions per day. For both long- and short-track speed skaters, adequate energy intake before and during periods of intense training is critical. Eating too little, either due to restrictive eating and/or lack of appetite can lead to underperformance. Considering the frequent cases of upper respiratory tract infections, injuries, mononucleosis, and signs of overtraining observed in long-track speed skating (Foster & De Koning, 1999), the emphasis should be on meeting energy and macronutrient needs during this training period (Meyer et al., 2011).

Due to the intense nature of speed skating (long and short track), the primary fuel for performance comes from muscle glycogen. According to Green (1978), glycogen utilization rates during bouts of continuous and intermittent skating are high. Especially in long-track speed skating, the low trunk position, associated blood occlusion (Szmedra et al., 2001), and high lactate levels and concomitant rise in acidity all contribute to fatigue. A daily high carbohydrate intake (6–12 g/kg/day) ensures repletion of muscle glycogen stores during phases of intense training. Supplementing with sport drinks, gels, or other carbohydrate-rich sources (30–60 g/h) is especially easy when athletes transition from ice to dry land training (Meyer & Parker-Simmons, 2009).

While short-track athletes probably expend less energy than long-track speed skaters, a moderately high carbohydrate intake is still important to support the intensity of training. If these athletes restrict energy and carbohydrates, they should at least increase their energy intake prior to racing, due to multiple heats and the risk of glycogen depletion (Meyer et al., 2011). In addition, athletes should supplement with carbohydrate-rich fluids/foods to maintain blood glucose levels when advancing from heat to heat. In contrast, long-track speed skaters may not need as much carbohydrate on race day, as most days these athletes compete only once or twice. Due to the enormous muscle mass of sprinters, they need sufficient protein, and should use recovery nutrition diligently as soon as possible after training and competition. Due to the importance of leanness in both short- and long-track speed skating, restrictive eating should be expected (Meyer & Parker-Simmons, 2009).

Finally, adequate hydration in speed skaters is important. On ice, speed skaters' sweat rates and fluid intakes are highly variable (unpublished observation, N. Meyer), and thus, individual guidelines are best provided according to sweat rate, fluid intake (i.e., some skaters may be drinking too much), training phase, and environmental condition (Meyer et al., 2011).

Ice Hockey For ice hockey players, the two main nutritional issues are high rates of glycogen utilization and sweat loss. After 60 minutes of intermittent play time (10× 1 minute on with 5-minute recovery time), a 70% reduction in muscle glycogen should be expected (Green et al., 1978). Thus, hockey players need a higher carbohydrate intake during intense, on-ice training (6–10 g/kg/day). Ice hockey is a team sport and is surrounded by a culture that makes it difficult to infuse more healthful and performance-enhancing nutritional strategies.

Sweat rates are high in ice hockey players and the uniform worn on ice makes it difficult to dissipate heat (Green et al., 1978). Recent data in junior players showed an average sweat rate of 1.8 l/hour. More than half of the players began practice in a dehydrated state and over one-third of the athletes lost more than 1% of initial body weight (Palmer & Spriet, 2008). There seems to be a need for more education in ice hockey players and changing the environment to promote appropriate fueling tactics may be necessary. Since both carbohydrate and fluid intake are needed to support intense on-ice training, providing sport drink by the rink may be a simple strategy to make the athletes drink it (Palmer et al., 2010).

Sliding Sports (Bobsled, Skeleton, and Luge)

The sliding sports include bobsled, skeleton, and luge (see Table 51.1). Skeleton is an individual sport performed on an ice track, also used by luge and bobsled athletes. Skeleton athletes push their sleds forward in a crouched position either with one or both hands for 15–30 seconds, then load to a prone, head-first position on top of the sled. In luge, athletes sit on the sled and push off from two handles followed by multiple arm strokes or paddles to accelerate before adopting a supine position on the sled. Finally, bobsled is made up of a crew. The crew begins by pushing the sled from a standing start, and then loads the sled, entering the track.

For all three sports, the pushing phase at the start and technical driving ability are critical and performance determining (Sands et al., 2005). Much of talent identification in bobsled and skeleton is directed toward sprint speed ability, while upper extremities, back extensors, and grip strength are important factors for luge (Platzer et al., 2009). All three sports have a weight limit (sled and athletes), but their risk for eating disorders is small (unpublished observation, N. Meyer). Interestingly, skeleton and luge athletes should also not "bulk up" nor become too lean, as this will change aerodynamics (Platzer et al., 2009). For all three sports, "ballast" or extra weight may be added to sleds to increase speed.

Nutrition Issues

No published data are currently available on the diets of bobsled, skeleton, and luge athletes. Estimates of energy expenditure depend on training volume and intensity including dry land training (e.g., weight lifting, sprint training, and plyometrics) and on-ice training. On-ice training is less energetically challenging due to the low number of runs possible using the cumbersome logistics of getting back up to the track start (unpublished observation, N. Meyer). Macronutrient intakes should be adjusted to strength/power athletes using a low to moderate carbohydrate intake (e.g., 3–5 g/kg/day and 5–7 g/kg/day) and sufficient protein spread throughout meals, snacks and for recovery. Fluid needs are expected to be relatively low during on-ice training and higher in summer environments. Best is to provide guidelines according to measured sweat rates.

Common Nutrition Issues Among Winter Sport Athletes

The sport-specific discussion of this chapter highlighted the major nutrition issues, including carbohydrates. Winter sport athletes also need adequate protein throughout the day, especially when training intensity and/or volume increase and energy is restricted for weight loss. Most winter sport athletes get adequate protein (Sjödin et al., 1994), except perhaps for ski jumpers (Rankinen et al., 1998). Fat intake of winter sport athletes ranges from 25% to 40% of total energy intake (Meyer & Parker-Simmons, 2009). Fat intake may be higher in those athletes with less experience and poor knowledge, in cross-country skiers during intense training

(Sjödin et al., 1994) and in those sports where a low body weight is not a decisive performance factor. Educating athletes on the amount and type of fat and how to balance training meals, adding oils, nuts, seeds, and fatty fish should be part of nutrition education (Meyer et al., 2011).

Winter sport athletes have unique micronutrient needs that are exacerbated when training is intense, integrates environmental extremes, and phases of energy restriction are imposed. In recent years, the use of altitude exposure has become a common strategy to increase hemoglobin mass. While earlier data showed a 35–40% prevalence of iron deficiency and depletion (Meyer & Parker-Simmons, 2009), more recent data on blood profiles in skiers (alpine and cross-country) show rising hemoglobin and hematocrit levels (Banfi et al., 2010; Morkeberg et al., 2009). Nevertheless, athletes should still be tested and adequate time to remedy low iron status should be provided, especially well ahead of altitude exposure (Stray-Gundersen et al., 1992).

Other micronutrients of interest for winter sport athletes are antioxidants and vitamin D. While the supplementation of certain micronutrients (e.g., iron and vitamin D) may be of benefit if status is low, ingestion of antioxidant supplements in the absence of elevated needs may be counterproductive (Powers et al., 2011; see Chapters 19, 20, and 21). Winter sport athletes need to use caution when making decisions related to dietary and sport supplements due to the risk of contamination (see Chapter 23). Limited dietary/sport supplements may benefit winter sport athletes. These include blood buffers, dietary sources of nitrate, creatine, and caffeine (Meyer et al., 2011).

In summary, most winter sport athletes are exposed to altitude and cold. Variably, these environments will affect the athlete's nutritional needs. Winter sport athletes have a range of nutrition issues mostly targeting energy, carbohydrate, and fluid intake. Winter sport athletes have varying degrees of physiques and most of them may be classified as weight-sensitive sports. Thus, adequate screening, treatment, and prevention strategies should be employed. Finally, winter sport athletes may be at risk for low iron and vitamin D status, and thus, should be tested and monitored before using dietary/supplement intervention strategies.

References

Askew, E.W. (1995) Environmental and physical stress and nutrient requirements. *American Journal of Clinical Nutrition* 61, 631S–637S.

Banfi, G., Tavana, R., Freschi, M., et al. (2010) Reticulocyte profile in top-level alpine skiers during four consecutive competitive seasons. *European Journal of Applied Physiology* 109, 561–568.

Barnholt, K.E., Hoffman, A.R., Rock, P.B., et al. (2006) Endocrine responses to acute and chronic high-altitude exposure (4,300 meters): modulating effects of caloric restriction. *American Journal of Physiology, Endocrinology, and Metabolism* 290, E1078–E1088.

Brouns, F., Saris, W.H., & Ten Hoor, F. (1986) Nutrition as a factor in the prevention of injuries in recreational and competitive downhill skiing. Considerations based on the literature. *Journal of Sports Medicine and Physical Fitness* 26, 85–91.

Burke, L.M., Hawley, J.A., Wong, S.H., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Sciences* 29(Suppl. 1), S17–S27.

Butterfield, G.E. (1999) Nutrient requirements at high altitude. *Clinics in Sports Medicine* 18, 607–621, viii.

Cox, M.H., Miles, D.S., Verde, T.J., et al. (1995) Applied physiology of ice hockey. *Sports Medicine* 19, 184–201.

Deutz, R.C., Benardot, D., Martin, D.E., et al. (2000) Relationship between energy deficits and body composition in elite female gymnasts and runners. *Medicine & Science in Sports & Exercise* 32, 659–668.

Ekblom, B. & Bergh, U. (2000) Cross-country skiing. In *Nutrition in Sport* (ed. R.J. Maughan), pp. 656–662. Blackwell Science, Oxford.

Ekelund, U., Yngve, A., Westerterp, K., et al. (2002) Energy expenditure assessed by heart rate and doubly labeled water in young athletes. *Medicine & Science in Sports & Exercise* 34, 1360–1366.

Ferguson, R.A. (2010) Limitations to performance during alpine skiing. *Experimental Physiology* 95, 404–410.

Foster, C. & De Koning, J.J. (1999) Physiological perspectives in speed skating. In *Handbook of Competitive Speed Skating* (eds. H. Gemser, J.J. De Koning & G.J. Van Ingen Schenau), pp. 117–137. International Skating Union, Lausanne, Switzerland.

Fulco, C.S., Kambis, K.W., Friedlander, A.L., et al. (2005) Carbohydrate supplementation improves time-trial cycle performance during energy deficit at 4,300-m altitude. *Journal of Applied Physiology* 99, 867–876.

Green, H.J. (1978) Glycogen depletion patterns during continuous and intermittent ice skating. *Medicine & Science in Sports* 10, 183–187.

Green, H.J., Daub, B.D., Painter, D.C., et al. (1978) Glycogen depletion patterns during ice hockey performance. *Medicine & Science in Sports* 10, 289–293.

Hesford, C.M., Laing, S., Cardinale, M., et al. (2012) Asymmetry of quadriceps muscle oxygenation during elite

short-track speed skating. *Medicine & Science in Sports & Exercise* 44, 501–508.
Jeukendrup, A.E. (2011) Nutrition for endurance sports: marathon, triathlon, and road cycling. *Journal of Sports Sciences* 29(Suppl. 1), S91–S99.
Kayser, B. (1992) Nutrition and high altitude exposure. *International Journal of Sports Medicine* 13(Suppl. 1), S129–S132.
Lakie, M. (2010) The influence of muscle tremor on shooting performance. *Experimental Physiology* 95, 441–450.
Mannix, E.T., Healy, A., & Farber, M.O. (1996) Aerobic power and supramaximal endurance of competitive figure skaters. *Journal of Sports Medicine and Physical Fitness* 36, 161–168.
Meyer, N.L. & Parker-Simmons, S. (2009) Winter Sports. In *Practical Sports Nutrition*, 1st edn (eds. L.M. Burke), pp. 335–358. Human Kinetics, Champaign, IL.
Meyer, N.L., Manore, M.M., & Helle, C. (2011) Nutrition for winter sports. *Journal of Sports Sciences* 29(Suppl. 1), S127–S136.
Morkeberg, J., Saltin, B., Belhage, B., et al. (2009) Blood profiles in elite cross-country skiers: a 6-year follow-up. *Scandinavian Journal of Medicine and Science in Sports* 19, 198–205.
Müller, W., Groschl, W., Müller, R., et al. (2006) Underweight in ski jumping: the solution of the problem. *International Journal of Sports Medicine* 27, 926–934.
Palmer, M.S. & Spriet, L.L. (2008) Sweat rate, salt loss, and fluid intake during an intense on-ice practice in elite Canadian male junior hockey players. *Applied Physiology, Nutrition and Metabolism* 33, 263–271.
Palmer, M.S., Logan, H.M., & Spriet, L.L. (2010) On-ice sweat rate, voluntary fluid intake, and sodium balance during practice in male junior ice hockey players drinking water or a carbohydrate-electrolyte solution. *Applied Physiology, Nutrition and Metabolism* 35, 328–335.
Phillips, S.M. & Van Loon, L.J. (2011) Dietary protein for athletes: from requirements to optimum adaptation. *Journal of Sports Sciences* 29(Suppl. 1), S29–S38.
Platzer, H.P., Raschner, C., & Patterson, C. (2009) Performance-determining physiological factors in the luge start. *Journal of Sports Sciences* 27, 221–226.
Powers, S., Nelson, W.B., & Larson-Meyer, E. (2011) Antioxidant and vitamin D supplements for athletes: sense or nonsense? *Journal of Sports Sciences* 29(Suppl. 1), S47–S55.
Rankinen, T., Lyytikainen, S., Vanninen, E., et al. (1998) Nutritional status of the Finnish elite ski jumpers. *Medicine & Science in Sports & Exercise* 30, 1592–1597.
Rusko, H. (2003) Physiology of cross country skiing. In *Handbook of Sports Medicine and Science—Cross Country Skiing* (eds. H. Rusko), pp. 1–31. Blackwell Science Ltd, Oxford.
Sands, W.A., Smith, L.S., Kivi, D.M., et al. (2005) Anthropometric and physical abilities profiles: US National Skeleton Team. *Sports Biomechanics* 4, 197–214.
Seifert, J.G., Luetkemeier, M.J., White, A.T., et al. (1998) The physiological effects of beverage ingestion during cross country ski training in elite collegiate skiers. *Canadian Journal of Applied Physiology* 23, 66–73.
Seifert, J.G., Lutkemeier, M.J., White, A.T., et al. (2000) Fluid balance during slalom training in elite collegiate alpine racers. In *2nd International Congress on Skiing and Science* (eds. E. Müller, H. Schwameder, C. Raschner, et al.), pp. 634–640. Verlag Dr. Kovac, St. Christoph, Austria.
Sjödin, A.M., Andersson, A.B., Hogberg, J.M., et al. (1994) Energy balance in cross-country skiers: a study using doubly labeled water. *Medicine & Science in Sports & Exercise* 26, 720–724.
Stöggl, T., Enqvist, J., Müller, E., et al. (2010) Relationships between body composition, body dimensions, and peak speed in cross-country sprint skiing. *Journal of Sports Sciences* 28, 161–169.
Stray-Gundersen, J., Hochstein, A., Delemos, D., et al. (1992) Failure of red cell volume to increase to altitude exposure in iron deficient runners. *Medicine & Science in Sports & Exercise* 24, S90.
Sundgot-Borgen, J. & Torstveit, M.K. (2010) Aspects of disordered eating continuum in elite high-intensity sports. *Scandinavian Journal of Medicine and Science in Sports* 20(Suppl. 2), 112–121.
Szmedra, L., Im, J., Nioka, S., et al. (2001) Hemoglobin/myoglobin oxygen desaturation during Alpine skiing. *Medicine & Science in Sports & Exercise* 33, 232–236.
Tesch, P.A. (1995) Aspects on muscle properties and use in competitive Alpine skiing. *Medicine & Science in Sports & Exercise* 27, 310–314.
Vallerand, A.L., Zamecnik, J., & Jacobs, I. (1995) Plasma glucose turnover during cold stress in humans. *Journal of Applied Physiology* 78, 1296–1302.
Ziegler, P.J., Jonnalagadda, S.S., Nelson, J.A., et al. (2002) Contribution of meals and snacks to nutrient intake of male and female elite figure skaters during peak competitive season. *Journal of the American College of Nutrition* 21, 114–119.

Chapter 52

Team Sports

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Introduction

Team sports encompass a wide range of activities that are not easy to organize into a single physiological stress category that thereby requires a distinct nutritional approach. Furthermore, position-specific roles, player substitution rules, season length, and playing environment within each sport contribute even more variety (Table 52.1). For instance, the carbohydrate needs of a defensive lineman in American football differ substantially from those of soccer midfielder; the hydration strategies for a center fielder in baseball during summer season are probably not the same as those for an outside centre in a rugby Winter season; and the muscle mass goals for a basketball center are again different from those of a field hockey midfielder. From this it is clear that the nutritional approach to team sports cannot be pinpointed as that for, say, a middle distance runner or a discus thrower, and the dietitian must therefore approach each case individually.

Several authors point to a limitation when studying team sports: no two games are alike (Gregson et al., 2010). This means that it is hard to standardize conditions in order to carry out studies to assess the effects of nutrition or supplement intervention. Most physical aptitude tests performed on team sport players assess specific fitness components such as aerobic capacity, leg power, or strength, but tests that replicate actual game conditions are rare. One such rarity is the Loughborough Intermittent Shuttle Test (LIST) (Nicholas et al., 2000), which lasts 90 minutes and attempts to replicate movement patterns encountered in a soccer match, although some authors do not consider it valid since it does not measure skill (Currell & Jeukendrup, 2008). It may also require greater effort than players expend in a game. Only a handful of studies have used the LIST to test the effect of nutrition on performance (Ali & Williams, 2009; Bailey et al., 2011; Erith et al., 2006; Leiper et al., 2005; Morris et al., 2003; Phillips et al., 2011; Taylor et al., 2011; Thompson et al., 2001), and similar tests for other sports are hard to find. This methodological complexity in measuring team sports impoverishes the body of scientific knowledge of the effects of nutrition on team sport performance, with many existing recommendations borrowing from studies on other athletes like runners and cyclists, but with a large contribution from observation, trial and error, and logical inferences.

Besides confronting the varied biological demands placed on these athletes and the relative research limitations, the team dietitian must also face a complex sociological scenario where interpersonal relationships between team members, coaching and medical staff, club authorities, the kit men, player managers, family members, product sponsors, and sometimes even media professionals and fans, set a stage where providing nutritional advice may not be as simple as it should be. Problems typically surface when the dietitian recommends a sports beverage that happens to be the competition brand of the team sponsor, has to

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Table 52.1 Team sport characteristics

Type of sport	Sport	Season (duration)	Game frequency	Game duration	Number of players on field	Substitutions per game
Strength-power	American football	Fall–Winter	1 × week	4 × 15′ periods	11	Unlimited
	Rugby union	Fall–Winter	1 × week	2 × 40′ halves	15	7
	Rugby league	Fall–Winter	1 × week	2 × 40′ halves	13	4
Endurance-based	Soccer	Year-round	1–2 × week	2 × 45′ halves	11	3
	Field hockey	Fall–Winter	1–2 × week	2 × 35′ halves	11	5
Court	Volleyball	Winter	1–3 × week	2–5 sets	6	18
	Basketball	Winter	1–3 × week	4 × 15′ quarters	5	Unlimited
	Handball	Winter	1–2 × week	2 × 35′ halves	7	9
Bat-and-ball	Baseball	Summer	1–3 × week	9 innings	9	Unlimited
	Softball	Summer	1–3 × week	9 innings	9	9
	Cricket	Summer	Variable	Variable	11	Only if injury

dispute the weight loss strategy of a player with a spouse or parent, criticizes the amino acid supplement the strength-coach just recommended, bans the locker room pastries the kit men bought for the players, or selects a pregame menu that contradicts the head coach's belief system. It is often the case that in order to fully implement an adequate nutritional intervention in a team, the dietitian must do so at a rate and span that is circumscribed by the aforementioned limitations, treading carefully to avoid, as much as possible, interpersonal conflict. While many times frustrating in the short run, perseverance, patience, and people skills can eventually lead to a successful nutritional program in the long run.

Specifics of Team Sports

Understanding the variation in team sport characteristics highlights the different game durations, season lengths, substitution allowances, game frequencies, and rest intervals affecting energy, substrate, and fluid demands, which must be replenished with a tailored nutrition program. Certain sport characteristics allow dietitians to implement optimum nutritional delivery strategies while others pose obstacles. For example, it is much easier to rehydrate players in sports like gridiron football or basketball, which have several rest intervals and time-outs and allow several substitutions, than in soccer, which has two continuous long halves with few interruptions and allows only three substitutions. If modifying body mass and composition is an issue, having 6–7 months during the off-season, as in rugby, will also permit goal-specific planning and work. However, in soccer, with a 10-month-long season and up to 50 or more games per year, this is difficult, since in-season specific body-modifying training and nutrition take a back seat to peaking in games.

Some sports like basketball, volleyball, and baseball require a large amount of traveling to play series of up to three games on consecutive days. Being on the road or flying, and spending time in hotels away from home naturally alters a player's regular nutrition regimen, since meals may be skipped, and food choices may be whatever is available instead of what is ideal. Successive daily games, even when efforts are not glycogen-depleting or dehydrating and frequent substitutions occur, still place a physiological stress that can be cumulative but that can also be ameliorated by adequate nutrition planning (Mujika & Burke, 2010).

Nutrient Intake of Team Sport Athletes

The dietary intake of team sport athletes is generally less than that of endurance athletes, but higher than that of strength and aesthetic athletes. Analyzing the reported intake from 3-day records of team sport athletes published in the last 30 years, weighted averages for energy intake are 15.3 MJ/day (3660 kcal) and 8.6 MJ/day (2064 kcal) for males (n = 819) and females (n = 283), respectively (Holway & Spriet, 2011). The macronutrient distributions are 49%, 17%, and 34% for males and 50%, 15%, and 35% for females for carbohydrates, proteins, and fats, respectively. Since some of the recommended intakes are 55–65% for carbohydrates and less than 30% for fats (Clark, 1994), the actual intake of some of these athletes does not meet these goals. Another way in which macronutrients are recommended is relative to body mass or weight, in grams per kilogram. Relative to body mass, male team sport athletes reported an average of 5.6 ± 1.3 g/kg/day for carbohydrate intake (Figure 52.1), and females 4.0 ± 0.7 g/kg/day, which is below expert committee recommendations of 6–10 g/kg/day (Rodriguez et al., 2009). Only a few studies report mean carbohydrate intakes above 8 g/kg/day, and in soccer players (Hickson et al., 1987; Jacobs et al., 1982; Rico-Sanz, 1998). In female team athletes, the highest mean carbohydrate intake, 5.2 g/kg/day, was also in soccer players (Clark et al., 2003). Athletes' reported intakes (Burke et al., 2001) as well as on-field experience in working with team sport athletes indicates that these smaller amounts seem to be appropriate, coinciding with the FIFA/FMARC 2006 soccer recommendation level of 5–7 g/kg/day (Nutrition for football: the FIFA/F-MARC Consensus Conference, 2006). Regrettably, data are lacking for some sports such as rugby union and cricket. Most of the reported intakes correspond to training days, while more data are needed for match days. It is important to recognize too the large individual variability in these reported intakes: some players are clearly achieving large intakes, but others have intakes that are far below the recommendations. Again, interpretation of these data requires consideration of the stage of the season and the training load on the days when intake was assessed. Coaches are often reluctant to allow measurements to be made during periods of intensive training or at key points of the season in case players are distracted and lose focus.

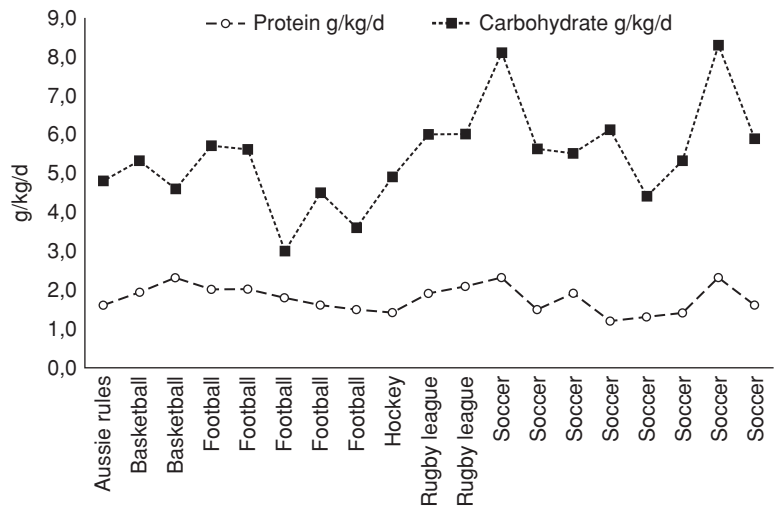

Figure 52.1 Carbohydrate and protein intake (g/kg/day) of male athletes in team sports reported in the literature between 1980 and 2010.

Modifying Size and Body Composition

It is often the case that modifying size and body composition to improve performance can actually compromise health (Jonnalagadda et al., 2001). For example, the size of gridiron football players has been increasing for several decades, with currently about 21% of 2616 NFL players in the 2009 roster weighing over 136 kg (300 lb) (http://www.nfl.com/players/search?category=team&playerType=current). According to Kraemer et al. (2005), body size has increased for offensive and defensive linemen but not for other positions since the 1970s, but height and body composition have remained similar in all positions. This means that linemen have both more muscle and adipose than 30 years ago. This issue presents a dilemma for the dietitian, but it is a reality of sport. On the other hand, team sport athletes may not need to drop their body fat to extremely low levels as in aesthetic or gravity-sensitive sports, reducing, but not entirely eliminating, the incidence of eating disorders (see Chapter 42). Throughout the season, body fat can naturally fluctuate by around 2–4% (Casajus, 2001), and care must be taken to assess current body composition status by comparing it to the appropriate seasonal standard, instead of constantly striving to pin it at the minimum value. It is good practice to have position-specific seasonal reference standards of body composition as a guide (Holway & Garavaglia, 2009).

Assessing and monitoring size and body composition in team sports is usually done at the start of preseason training and at intervals throughout the season, with closer supervision of players who need to modify muscle and/or adipose tissues. Since most teams do not have laboratories with expensive body composition equipment, and the sports dietitian must often assess players in a less-than-ideal location with the added constraint of limited time availability, it is best to use surface anthropometry when evaluating size and composition of team sport players (see Chapter 6). Measures such as height and body mass, together with a series of skinfold tests from several body locations can be carried out quickly and provide a good amount of information to the dietitian. If time permits, trunk and limb girths and bone breadths can further contribute valuable information on muscle and skeletal structures (Holway & Garavaglia, 2009). Care must be taken to measure using a standardized protocol and calibrated equipment (Hume & Marfell-Jones, 2008).

Simultaneous nutrition and exercise interventions to change size and body composition require effort, discipline, and time, and ideally it is done in the off-season. While some sports like rugby and basketball have competitive periods that last up to 6 months, allowing sufficient time to work on modifying muscle and/or adipose tissues, other sports like soccer have seasons that last up to 10 months with over 50 matches. Hence in soccer, for instance, important changes in muscle and fat are best completed in the youth divisions. The main issue in size modification is achieving a nutritional surplus or deficit with respect to energy balance (Rodriguez et al., 2009). This can be accommodated with food, although in some instances energy-dense supplements may help in achieving lean body mass gain (Maughan et al., 2007), but care must be taken to avoid unwanted fat gain. No nondoping nutritional supplements have been shown to be effective in promoting significant fat losses (Pittler & Ernst, 2004). A usual scenario of an athlete needing to gain weight is that of a tall, ectomorph player in his late teens that may be training with his age-division team, the first team, and perhaps also some type of select side, as well as trying to finish the growth process. The sports dietitian must assess the situation and determine whether the inability to gain muscle stems from excessive training or undernutrition. A coordinated approach with the athletic trainer is of utmost importance for the success of a body composition change strategy. Many young team athletes comply with adequate nutrition from Monday to Friday, but slack off on weekends, when they miss important meals and sleep, and may overindulge in alcohol, slowing or halting progress altogether. Muscle gains must not be indiscriminate: there is a tendency in rugby and gridiron football or baseball to maximize muscle mass, when the ideal amount that will produce the best power-to-weight ratio and acceleration is very likely less than maximum hypertrophy (Cronin & Hansen, 2005).

Fat loss can also be difficult for some athletes, particularly older athletes who now need to make extra efforts in their dietary habits to avoid gaining fat mass. Fat has its own individual rate of loss, so a proper time frame must be allotted for the plan to work effectively and weight loss expectations should not surpass biological reality in terms of quantity and speed. A conservative approach can be half-a-kilogram of adipose tissue per week (Garthe et al., 2011), although individual differences play a role here and some players may be able to speed up this rate while others might need more time. This rate of fat loss is adequate to prevent unwanted muscle mass losses, but speeding up the process can result in lean mass losses, an increased chance of injury, impaired immune function, and slowing recovery (Rankin, 2002).

Nutrient Timing

Although the subject of nutrient timing currently receives a lot of attention (Stephens & Braun, 2008), it is more important for athletes who train twice daily and have a large energy and glycogen expenditure, such as endurance-based athletes. Team sports athletes typically play matches once a week in the case of soccer, rugby, gridiron football, and field hockey, but court sports like basketball, volleyball, and handball and batting sports like cricket, baseball, and softball generally play several days in a row. Many soccer teams also play twice a week, whereby these teams imitate their court and bat counterpart sports and rotate players in the roster to provide ample recovery. If you have one whole week to recover between matches and train once-a-day, there is ample opportunity for nutritional recovery, but if you must play three days in a row, even if the court or batting sport is less dependent on carbohydrate metabolism and many substitutions take place, progressive glycogen depletion and dehydration must be accounted for with proper nutrient timing (Mujika & Burke, 2010; Reilly & Ekblom, 2005). This entails a recovery meal or supplement consumed within the first hour after training or playing and containing 1–1.5 g/kg body mass carbohydrates for glycogen repletion, fluids for rehydrating, and some 10–20 g of protein for tissue reparation (Howarth et al., 2009). In many Western countries, low-fat chocolate milk is a popular choice, while other societies use a drink based on soymilk and sugar. The menu choices are many and can cater to the local cultural preference, budget constraints, and practical aspects such as food preparation, transport, and conservation.

Pre-event or training meals must supply carbohydrates and protein but have to be easy-to-digest, so a good suggestion is to avoid unfamiliar foods and those that are very high in fat and fiber (Williams & Serratosa, 2006). Digestion times vary, but it is generally agreed that between 2 and 4 hours is adequate. Some athletes prefer a meal and then a small snack or sports drink in the hour prior to playing or training. It is best if the type of carbohydrate in the pre-event meal will supply a sustained rate of glucose to the blood stream, as with pasta, rice, and legumes, instead of very refined sources.

During the game or training session, if longer than 1 hour, sports drinks and carbohydrate snacks can aid in maintaining fuel and fluid levels to ensure optimum performance (Sawka et al., 2007).

Dietary Planning

One of the main concerns when planning nutrition programs for team sport athletes is adequate energy (Rodriguez et al., 2009). Missing this important target can mean unwanted fat gain or muscle loss with inadequate recovery. A second concern is achieving a carbohydrate dose that is in the proper range. The difficulty with carbohydrates is that, as with energy balance, too much or too little can negatively affect performance, and there is a narrow ideal range for each player. In our experience, the prescription approach using grams per kilogram per day will work fairly well for athletes up to around 85 kg body mass, but beyond that this approach will tend to overestimate carbohydrate needs. Land-based large athletes (as opposed to swimmers and rowers who have their weight supported by water) do not cover the distances that lighter athletes do, and are generally assigned to positions that require less field movement and more strength and scrimmaging tasks. Their energy and carbohydrate needs follow an exponentially decreasing curve (Figure 52.2)

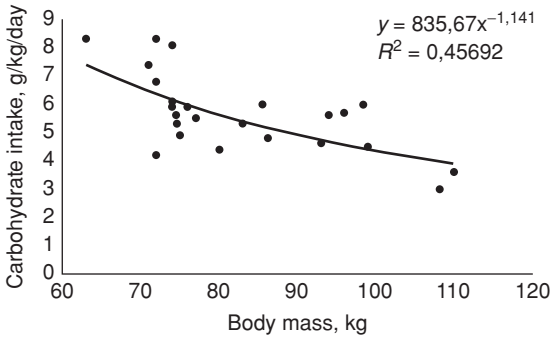

Figure 52.2 Diminishing carbohydrate intake relative to body mass in team sport athletes. Data from Holway and Spriet (2011).

relative to their body mass, and hence their requirements are less than those generally suggested for athletes weighing 65–85 kg. While their fat intake is similar to that of lighter team mates relative to body mass, their protein intake shows a tendency to increase, so it might be the case that their protein needs are larger, or that years of trial-and-error have led them to choose this macronutrient proportion. An alternative hypothesis might be that larger athletes in more strength-related field tasks subscribe to the strength-athlete preference for a higher-protein diet. Unfortunately, very little information exists on the nutritional intake of team sport athletes weighing over 100 kg.

When planning team or individual menus for team sport players, care should be taken to ensure that the three or four daily meals contain enough carbohydrate sources to cover training or game needs, and that these sources are spread out throughout the day and not concentrated in one or two meals. The carbohydrate amount, as mentioned before, varies according to the sport and position, but a good starting guide is to use between 4 and 8 g/kg/day, with a little more for special situations such as twice-a-day preseason training sessions; large athletes (i.e., those over 100 kg) may have needs at the lower end of the per-body-mass spectrum. Good carbohydrate sources are whole grains, legumes, fruits, and starchy and fibrous vegetables, with additional intake coming from dairy or vegetable milks. If daily carbohydrate needs cannot be covered with these foods, then sugars in drinks, bars, and gels can help cover the requirement. Protein needs are usually more-than-adequately covered in these athletes, often at the expense of carbohydrates, and 1.2–1.6 g/kg/day are adequate. Players may choose their sources from lean flesh foods or plants if vegetarian. As with carbohydrates, it is best to spread the protein sources throughout the day (Phillips & Van Loon, 2011). Fats should make up the remaining energy budget, and include sources of essential fatty acids found in cold-water fish or nuts and seeds.

Hydration

Hydration can be a concern for team sport athletes (Burke & Hawley, 1997), albeit the duration of games and matches is shorter and permits more substitutions, breaks, and opportunities to rehydrate than in endurance individual sports like road cycling or iron man distance triathlons. Weather conditions do of course affect sweat rates, as when baseball or cricket are played during the summer season, but also carrying heavy uniforms, being overweight, undergoing multiple daily training sessions, and being a "heavy sweater" contribute as well (Casa et al., 2005). Another important aspect that can hamper the proper hydration status is when athletes attempt severe diets and try to lose weight at the start of preseason training. The pressure to lose weight to make the team or conform to some rigid standard imposed by a staff member can lead an athlete to improper rehydration, sometimes with adverse health consequences. For instance, a female volleyball coach might weigh the players before practice in the morning, so they would skip breakfast and refrain from drinking prior to training in an effort to ensure a loss at the scale. An extreme case was that of 152-kg Korey Stringer, a professional football player who collapsed with heat exhaustion and died in 2001 while trying to lose weight and train in the summer. The sports dietitian can control body mass before and after practice or games to assess sweat loss rates and also to educate the players as to how much they should rehydrate.

It is clear that sweat rate among players is highly individual, and assessing fluid losses dur-

ing two or three sessions will help identify the individuals who are more prone to having their performance and health hampered by severe dehydration (Maughan & Shirreffs, 2010). Field experience with several team sports has shown us that usually about 15% of players fall into this category, and firm hydration education with these players must be implemented to ensure they abide. Other than assessing pre- and post-training weight differences, the sports dietitian can measure urine-specific gravity with a refractometer (Armstrong et al., 2010). When these tests are performed, it is often the case that many players show up for practice or games in a dehydrated state, so here education strategies or on-the-premises hydration strategies must be implemented. A recent concern is salt loss in athletes, possibly leading to muscle cramping and electrolyte imbalances (Maughan & Shirreffs, 2010). Sweat patches and their analysis are difficult to come by for most sports dietitians, but identifying salt marks on the skin or dark clothing of a player can aid in identifying such cases (Figure 52.3). These athletes may benefit from additional salt intake in meals and beverages surrounding the training session or game. Urine color charts placed in toilets can be of help in making players aware of dehydration provided they are also aware that food fortification and multivitamins can alter urine color (Armstrong et al., 1998).

Figure 52.3 Salt marks in a professional soccer player.

Hydration practices during games vary widely. For example, in professional soccer, we have witnessed some players barely sip water at the halftime break while their teammate sitting next to them drinks two cans of an energy drink. Would they perform better if they drank a liter of a sports drink? Perhaps, but it is hard to measure. Performing studies during official matches is usually very difficult because the coaching staff would, understandably, avoid any distractions, so not much information is available from real match situations.

Supplementation

Nutritional supplements are probably at the forefront of confrontation between sports dietitians and athletic trainers and physical therapists. There may be several reasons for this that are beyond the scope of this chapter, but it is a reality that must be dealt with. In general, dietitians are much less prone to supplement than other sport team staff, and sports nutritionists feel their area of expertise has been invaded; after all, dietitians do not plan the strength program for the athletes or enforce tactical strategies on the coaches. Nevertheless, it is perhaps not a good idea for the dietitian beginning work in a team to generate outright confrontation, but to gradually reach consensus through frank discussion with other staff members. Whatever the strategy chosen to address this problem, the athlete must not receive contradicting messages from staff members regarding nutritional supplements.

As to the efficacy of nutritional supplements for team sport athletes, it is generally agreed and supported by science that sports beverages, food replacement bars and drinks, caffeine, creatine, and sometimes protein or a carbohydrate–protein supplement can enhance recovery and/or performance (Maughan et al., 2007). Supplement fashions come and go, like the hydroxy-methylbutyrate for muscle gains (Nissen et al., 2000) in rugby players, or chromium picolinate in football players (Evans & Pouchnik, 1993), androstenedione in baseball, or the current nitric oxide (Liu et al., 2009) craze in strength sports; however, the "test of time" allows few survivors, notably those mentioned above. Players are constantly bombarded with marketing claims

for nutritional supplements, and locker room gossip and strength-and-fitness gurus also fuel trends, sometimes through ignorance and other times for outright financial profit. It is sometimes the case that nonbanned nutritional supplements are claimed by some athletes to be the reason behind their sudden eye-opening success, only to confess later to steroid use. In this case, nutritional supplements act as a screen to justify impressive body composition changes or performance improvements, when the real contributor is a banned substance like anabolic steroids, growth hormone, erythropoietin, or some stimulant. Despite the subsequent discovery that the real actor was a banned substance and not the purported nutritional supplement, legions of athletes are coaxed into believing pharmacological improvements are possible from nutrients. The sport nutritionist has the tough task of recommending supplements only as an auxiliary complement to food, of explaining the need to have scientific backing for safety, efficacy, and legality for these substances, and of downplaying outrageous claims for pills and powders, as well as alerting players and support staff to the potential doping risk due to contamination (Maughan et al., 2011).

As with other biological issues, athletes respond differently to supplements. Creatine is one supplement in particular that generates an array of responses in team sport athletes: some respond very well and want to continue taking it, a second group notices no effect whatsoever, and a third group feels this supplement is detrimental to their performance. Aside from the individuality in response to this supplement, other factors may contribute to the variety in responses, such as dose, or current intake from flesh foods. Some players who need to cover distances running and sprinting report feeling tightness and stiffness in the muscles and some go even further to claim muscle injuries, despite the lack of scientific backing for this assertion (Greenwood et al., 2003). Others report feeling "too heavy" for their preference. One possible cause may be that the creatine loading dose protocol was used, whereby around 100 g of creatine are ingested in 5 days. While this may work well in strength athletes who do not need to transport their body mass several kilometers per game, in some players this does not work too well. However, when they take in smaller doses, such as 2–5 g/day, without the loading protocol, then they feel better. The bottom line with creatine in team sport athletes is that it may benefit some while it may hamper the performance of others. Since individual response is so prominent, if the choice to try creatine has been made, players should start with a conservative dose, less than 5 g/day, and evaluate the response over a few weeks. Athletes consuming a large daily amount of creatine from food sources such as flesh foods may already have elevated muscle creatine stores and may not notice improvements when supplementing.

In recent years, supplements such as beta-alanine (Stellingwerff et al., 2011) and nitrates in beetroot juice (Bailey et al., 2010) have been showing interesting results in high-intensity endurance protocol, but it yet remains to be determined whether they have any utility in team sports.

One critical underlying fact for any supplement in a team sports setting is flavor. If the taste is foul, it will be very hard for these types of athletes to adhere to the supplement regimen for an extended period of time. Another important factor to increase the odds that these players adhere to a supplement program is for the sports medicine staff to provide it on the club premises at the locker room training or recovery table. When the supplement is given to the athletes to consume at home many will not keep up the regular intake schedule. Getting the support of the team or locker room leaders when introducing new supplements or dietetic approaches is also important, since in the psychology of team groups the opinion of leaders can influence the perception of the other players.

Cultural Issues

It is important for the team sports dietitian to establish a proper rapport with all members of the medical and coaching staff, the team and player managers, the catering staff at the club and at team hotels, even player family members and club support staff such as kit men, security and cleaning personnel, as well as the players. Building rapport with these people can help the dietitian deliver adequate sports nutrition and education

to the players and avoid conflicts. For example, depending upon culture, it may be the case that if a nutrition issue is uncomfortable to some players, they will not discuss it directly with the dietitian, but may likely express their opinions to the masseuse or physical therapist. Hence a good relationship with this therapist can be a good source of feedback from the players. In other cultures, the locker room kit men like to pamper their athletic idols with a tray of pastries before training sessions, and if the dietitian outright bans that practice without discussing the matter with the kit man, he or she might be generating unnecessary conflict. Kit men can be great aids to the dietitian in stocking and helping deliver sports drinks, setting up the nutrition-training table, and even setting up a nutrition recovery meal when the dietitian needs to miss a practice session. The janitors can be key players in this elaborate team sport sociological scenario, since they know from the residue collection what the players have been eating in their rooms. I learned from them that whenever my previous-day dinners were too low in fat and high in fiber, junk food smuggling and consumption in their rooms would skyrocket. As mentioned before, a good relationship with the athletic coach is imperative, and differences must be ironed out as best as possible in a private setting.

References

Ali, A., & Williams, C. (2009). Carbohydrate ingestion and soccer skill performance during prolonged intermittent exercise. *Journal of Sports Sciences* 27(14), 1499–1508.

Armstrong, L.E., Soto, J.A., Hacker Jr, F.T., et al. (1998) Urinary indices during dehydration, exercise, and rehydration. *International Journal of Sport Nutrition* 8(4), 345–355.

Armstrong, L.E., Pumerantz, A.C., Fiala, K.A., et al. (2010) Human hydration indices: acute and longitudinal reference values. *International Journal of Sport Nutrition and Exercise Metabolism* 20(2), 145–153.

Bailey, S.J., Fulford, J., Vanhatalo, A., et al. (2010) Dietary nitrate supplementation enhances muscle contractile efficiency during knee-extensor exercise in humans. *Journal of Applied Physiology* 109(1), 135–148.

Bailey, D.M., Williams, C., Betts, J.A., et al. (2011) Oxidative stress, inflammation and recovery of muscle function after damaging exercise: effect of 6-week mixed antioxidant supplementation. *European Journal of Applied Physiology* 111(6), 925–936.

Burke, L.M. & Hawley, J.A. (1997) Fluid balance in team sports. Guidelines for optimal practices. *Sports Medicine* 24(1), 38–54.

Burke, L.M., Cox, G.R., Cummings, N.K., et al. (2001) Guidelines for daily carbohydrate intake: do athletes achieve them? *Sports Medicine* 31(4), 267–299.

Casa, D.J., Clarkson, P.M., & Roberts, W.O. (2005) American College of Sports Medicine roundtable on hydration and physical activity: consensus statements. *Current Sports Medicine Reports* 4(3), 115–127.

Casajus, J.A. (2001) Seasonal variation in fitness variables in professional soccer players. *Journal of Sports Medicine and Physical Fitness* 41(4), 463–469.

Clark, K. (1994) Nutritional guidance to soccer players for training and competition. *Journal of Sports Sciences* 12(Spec. No.), S43–S50.

Clark, M., Reed, D.B., Crouse, S.F., et al. (2003) Pre- and post-season dietary intake, body composition, and performance indices of NCAA division I female soccer players. *International Journal of Sport Nutrition and Exercise Metabolism* 13(3), 303–319.

Cronin, J.B. & Hansen, K.T. (2005) Strength and power predictors of sports speed. *Journal of Strength and Conditioning Research* 19(2), 349–357.

Currell, K. & Jeukendrup, A.E. (2008) Validity, reliability and sensitivity of measures of sporting performance. *Sports Medicine* 38(4), 297–316.

Erith, S., Williams, C., Stevenson, E., et al. (2006) The effect of high carbohydrate meals with different glycemic indices on recovery of performance during prolonged intermittent high-intensity shuttle running. *International Journal of Sport Nutrition and Exercise Metabolism* 16(4), 393–404.

Evans, G.W. & Pouchnik, D.J. (1993) Composition and biological activity of chromium-pyridine carboxylate complexes. *Journal of Inorganic Biochemistry* 49(3), 177–187.

Garthe, I., Raastad, T. & Sundgot-Borgen, J. (2011) Long-term effect of weight loss on body composition and performance in elite athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 21(5), 426–435.

Greenwood, M., Kreider, R.B., Greenwood, L., et al. (2003) Cramping and injury incidence in collegiate football players are reduced by creatine supplementation. *Journal of Athletic Training* 38(3), 216–219.

Gregson, W., Drust, B., Atkinson, G., et al. (2010) Match-to-match variability of high-speed activities in premier league soccer. *International Journal of Sports Medicine* 31(4), 237–242.

Hickson Jr, J.F., Johnson, C.W., Schrader, J.W., et al. (1987) Promotion of athletes' nutritional intake by a university foodservice facility. *Journal of the American Dietetic Association* 87(7), 926–927.

Holway, F.E. & Garavaglia, R. (2009) Kinanthropometry of Group I rugby players in Buenos Aires, Argentina. *Journal of Sports Sciences* 27(11), 1211–1220.

Holway, F.E. & Spriet, L.L. (2011) Sport-specific nutrition: practical strategies for team sports. *Journal of Sports Sciences* 29(Suppl 1), S115–S125.

Howarth, K.R., Moreau, N.A., Phillips, S.M., et al. (2009) Coingestion of protein with carbohydrate during recovery from endurance exercise stimulates skeletal muscle protein synthesis in humans.

Journal of Applied Physiology 106(4), 1394–1402.

Hume, P. & Marfell-Jones, M. (2008) The importance of accurate site location for skinfold measurement. *Journal of Sports Sciences* 26(12), 1333–1340.

Jacobs, I., Westlin, N., Karlsson, J., et al. (1982) Muscle glycogen and diet in elite soccer players. *European Journal of Applied Physiology and Occupational Physiology* 48(3), 297–302.

Jonnalagadda, S.S., Rosenbloom, C.A. & Skinner, R. (2001) Dietary practices, attitudes, and physiological status of collegiate freshman football players. *Journal of Strength and Conditioning Research* 15(4), 507–513.

Kraemer, W.J., Torine, J.C., Silvestre, R., et al. (2005) Body size and composition of National Football League players. *Journal of Strength and Conditioning Research* 19(3), 485–489.

Leiper, J.B., Nicholas, C.W., Ali, A., et al. (2005) The effect of intermittent high-intensity running on gastric emptying of fluids in man. *Medicine & Science in Sports & Exercise* 37(2), 240–247.

Liu, T.H., Wu, C.L., Chiang, C.W., et al. (2009) No effect of short-term arginine supplementation on nitric oxide production, metabolism and performance in intermittent exercise in athletes. *Journal of Nutritional Biochemistry* 20(6), 462–468.

Maughan, R.J. & Shirreffs, S.M. (2010) Development of hydration strategies to optimize performance for athletes in high-intensity sports and in sports with repeated intense efforts. *Scandinavian Journal of Medicine & Science in Sports* 20(Suppl. 2), 59–69.

Maughan, R.J., Depiesse, F., & Geyer, H. (2007) The use of dietary supplements by athletes. *Journal of Sports Sciences* 25(Suppl. 1), S103–113.

Maughan, R.J., Greenhaff, P.L., & Hespel, P. (2011) Dietary supplements for athletes: emerging trends and recurring themes. *Journal of Sports Sciences* 29(Suppl. 1), S57–S66.

Morris, J.G., Nevill, M.E., Thompson, D., et al. (2003) The influence of a 6.5% carbohydrate-electrolyte solution on performance of prolonged intermittent high-intensity running at 30 degrees C. *Journal of Sports Sciences* 21(5), 371–381.

Mujika, I. & Burke, L.M. (2010) Nutrition in team sports. *Annals of Nutrition & Metabolism* 57(Suppl. 2), 26–35.

Nicholas, C.W., Nuttall, F.E., & Williams, C. (2000) The Loughborough Intermittent Shuttle Test: a field test that simulates the activity pattern of soccer. *Journal of Sports Sciences* 18(2), 97–104.

Nissen, S., Sharp, R.L., Panton, L., et al. (2000) Beta-hydroxy-beta-methylbutyrate (HMB) supplementation in humans is safe and may decrease cardiovascular risk factors. *Journal of Nutrition* 130(8), 1937–1945.

Nutrition for football: the FIFA/F-MARC Consensus Conference (2006) *Journal of Sports Science* 24(7), 663–664.

Phillips, S.M. & Van Loon, L.J. (2011) Dietary protein for athletes: from requirements to optimum adaptation. *Journal of Sports Sciences* 29(Suppl. 1), S29–S38.

Phillips, S.M., Turner, A.P., Sanderson, M.F., et al. (2011) Carbohydrate gel ingestion significantly improves the intermittent endurance capacity, but not sprint performance, of adolescent team games players during a simulated team games protocol. *European Journal of Applied Physiology* 112(3), 1133–1141.

Pittler, M.H. & Ernst, E. (2004) Dietary supplements for body-weight reduction: a systematic review. *American Journal of Clinical Nutrition* 79(4), 529–536.

Rankin, J.W. (2002) Weight loss and gain in athletes. *Current Sports Medicine Reports* 1(4), 208–213.

Reilly, T. & Ekblom, B. (2005) The use of recovery methods post-exercise. *Journal of Sports Sciences* 23(6), 619–627.

Rico-Sanz, J. (1998) Body composition and nutritional assessments in soccer. *International Journal of Sport Nutrition* 8(2), 113–123.

Rodriguez, N.R., Di Marco, N.M., & Langley, S. (2009) American College of Sports Medicine position stand. Nutrition and athletic performance. *Medicine & Science in Sports & Exercise* 41(3), 709–731.

Sawka, M.N., Burke, L.M., Eichner, E.R., et al. (2007) American College of Sports Medicine position stand. Exercise and fluid replacement. *Medicine & Science in Sports & Exercise* 39(2), 377–390.

Stellingwerff, T., Anwander, H., Egger, A., et al. (2011) Effect of two beta-alanine dosing protocols on muscle carnosine synthesis and washout. *Amino Acids* 42(6), 2461–2472.

Stephens, B.R. & Braun, B. (2008) Impact of nutrient intake timing on the metabolic response to exercise. *Nutrition Reviews* 66(8), 473–476.

Taylor, C., Higham, D., Close, G.L., et al. (2011) The effect of adding caffeine to postexercise carbohydrate feeding on subsequent high-intensity interval-running capacity compared with carbohydrate alone. *International Journal of Sport Nutrition and Exercise Metabolism* 21(5), 410–416.

Thompson, D., Williams, C., Kingsley, M., et al. (2001) Muscle soreness and damage parameters after prolonged intermittent shuttle-running following acute vitamin C supplementation. *International Journal of Sports Medicine* 22(1), 68–75.

Williams, C. & Serratosa, L. (2006) Nutrition on match day. *Journal of Sports Sciences* 24(7), 687–697.

Chapter 53

Weight-Category Sports

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Introduction

To make/cut weight is part of the culture for athletes competing in weight-category sports as it may give the athlete an advantage over an opponent in terms of strength, speed, agility, and/or leverage. Making weight refers to the process of losing weight to qualify for a weight class below the athlete's natural weight (Oppliger et al., 2003). Health- and nutrition-related concerns regarding weight loss practices of athletes competing in weight-category sports have been raised since the 1940s (Tuttle, 1943). Measures have been put in place to try and limit the use of harmful weight-loss strategies, such as rule changes on making weight by the National Collegiate Athletic Association (NCAA) in the United States after the untimely deaths of three young wrestlers due to acute dehydration and hyperthermia while trying to make weight for wrestling competitions (AMA, 1998; Wrestling Rules Committee, 1999), as well as the International Federation of Rowing Association (FISA). Since the 1960s, bodies such as the American Medical Association (AMA) and the American College of Sports Medicine (ACSM) have also advocated against harmful weight loss practices due to the negative and unhealthy effect these practices could have on the athlete (ACSM, 1976; AMA, 1967; Oppliger et al., 1996; Tipton & Tcheng, 1970). However, many athletes still make use of harmful weight loss practices today and organizations still have weigh-in rules and weight categories that lend themselves toward stimulating the use of these negative and sometimes dangerous weight loss practices.

This chapter will focus on the weight-loss strategies and techniques of athletes competing in Olympic weight-class sports (including international style wrestling, boxing, judo, taekwondo, weightlifting, and lightweight rowing) as well as possible health- and performance-related consequences pertaining to popular weight-loss strategies in these athletes. Other weight-category sports, not specifically covered in this chapter, use similar weight-loss strategies and, therefore, face similar nutritional, health, and performance consequences.

Weight-Loss Strategies and Techniques

Athletes make use of various weight-loss strategies and techniques in their quest to qualify for a certain weight category. Some strategies are rapid and extreme while others are gradual and, generally, healthier options (see Figure 53.1). Athletes usually aim to lose body fat and/or body water but maintain lean body mass (LBM) when making weight. During a competitive season, weight-category athletes will go through a cycle of weight loss and regain that can be repeated on average 15 times per season, a process referred to as weight cycling. After weigh-in, athletes will consume

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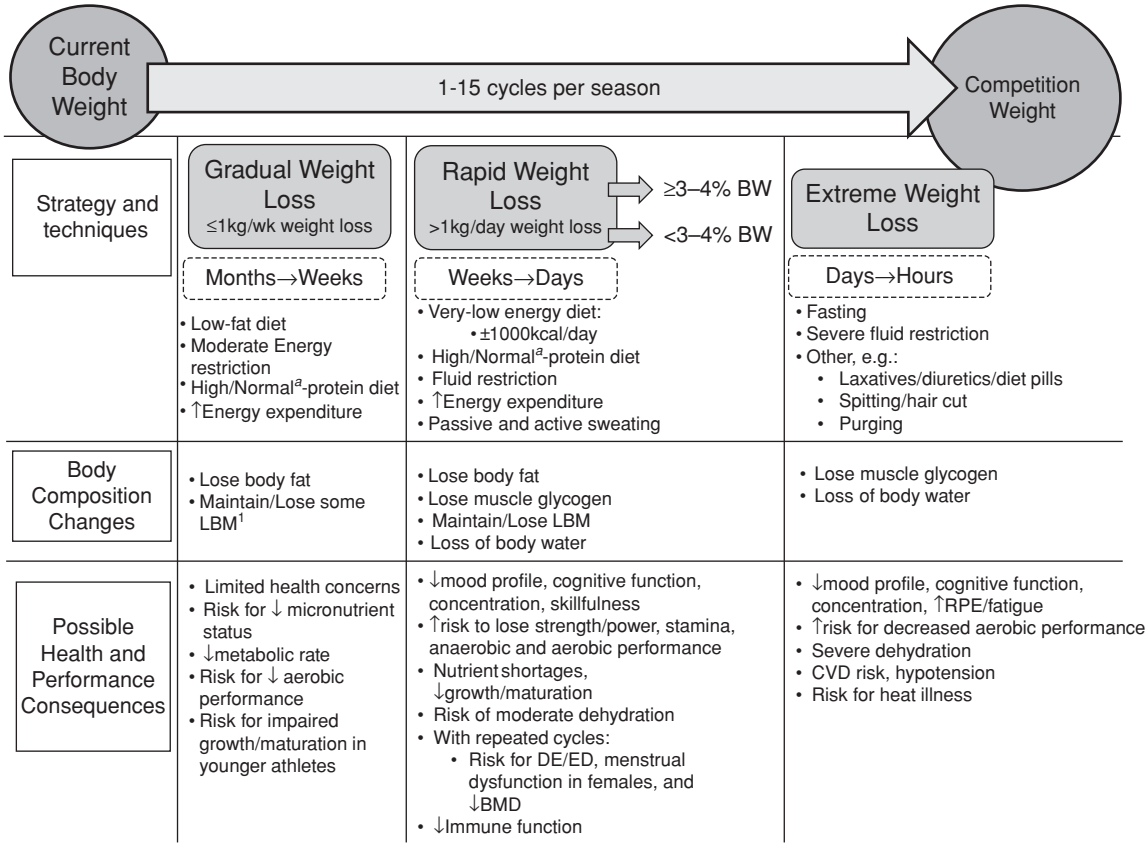

Figure 53.1 Common weight loss strategies and techniques used and the associated body composition changes, as well as possible health and performance consequences. LBM, lean body mass; DE/ED, disordered eating behavior/eating disorder; RPE, rating of perceived exertion; BMD, bone mineral density; CVD, cardiovascular disease.

[a]When following a high-protein diet, LBM can be maintained.

large amounts of food and fluids to try and regain as much as possible of their lost weight, only to try and lose it again before the next weigh-in session. Before implementation of the new NCAA rules on weight cutting, some US college wrestlers reported weight cycling more than 100 times over a period of approximately 6 years with an average of 15 weight-cycling events during a season: the greatest acute weight loss ranged from 1.4 to 20.5 kg at any one time during a season (Steen & Brownell, 1990). While going through these weight cycles, athletes generally strive either to maintain their physical performance and health while making weight or to recover their performance to pre-weight-loss levels after weigh-in.

Weight-loss strategies include restriction of food and/or fluids and/or increased exercise. Common techniques used to restrict food intake include fasting, meal skipping, taking diet pills, vomiting, and binging/purging. Use of saunas, hot tubs, heated training rooms, rubber or plastic sweat suits, spitting, taking laxatives, and/or diuretics are often used as dehydration techniques. Increased exercise is used to facilitate not only weight loss but also dehydration even further by exercising in hot environments or with rubber/plastic suits. Athletes

have reported the use of combinations of four or more of these techniques when cutting body weight (BW) (Artioli et al., 2010; Slater et al., 2005a; Steen & Brownell, 1990).

In Figure 53.1, popular weight-loss strategies and techniques are summarized, along with associated body composition changes and possible health- and performance-related concerns specific to the strategy and techniques used by weight-category athletes.

Wrestlers and Judokas

Rule changes by the NCAA have proved to be effective in decreasing the frequency of use of dangerous rapid weight-loss (RWL) strategies among US college and high school wrestlers, as well as decreasing the amount of weight lost before competitions (Oppliger et al., 2003). Nevertheless, even after the introduction of these rules, 5–8% still report the use of methods such as fasting and the use of saunas and rubber or plastic suits on 3 or more days per week (Davis et al., 2002; Oppliger et al., 2003). However, since these rules have not been adopted for international style wrestling (including freestyle and Greco-Roman), it seems that wrestlers abide by them only during the regulated scholastic season. In fact, the frequency with which RWL strategies, including banned strategies such as saunas or rubber/plastic suits, were still being used ranged between 40% and 60% in a group of wrestlers (aged 15–18 years) competing in the 1997 and 1998 US National wrestling championships (Alderman et al., 2004). Additionally, an average weight gain of 3.4 ± 1.8 kg (~5% gain in BW) was found between weigh-in and competition. This is similar to weight-loss levels found among wrestlers before implementation of the NCAA rules (Steen & Brownell, 1990). Artioli et al. (2010) found that among a group of judo athletes (men = 607, women = 215) competing at all levels, many (48%) made use of RWL strategies and lost between ~2% and 5% of BW but some lost more than 5% BW over a short period of time (less than 5 days) before competing, similar to international style wrestlers. These RWL strategies included techniques such as the use of plastic/rubber suits. Most of these athletes had between two and five weight-cycling events but some had as many as 10 weight-cycling events a year.

Lightweight Rowers

The international governing body of rowing (FISA) discourages lightweight rowers from using RWL strategies (i.e., food and fluid restriction, increased exercise). Looking at the weight loss practices of elite Australian lightweight rowers (n = 100), Slater et al. (2005a) found 91% made use of two or more weight loss practices, with 60% of all respondents using four or more weight loss practices. The most popular weight loss practices were gradual dieting, fluid restriction, and increased training load. Self-reported weight loss in the week prior to a regatta was as high as 6 kg in men and 4.5 kg in women, with daily BW fluctuations of 0.5–1.0 kg during the regatta. Some oarsmen and oarswomen had, however, daily variations up to 4 and 2 kg, respectively. Most of the male and female rowers reported that making weight during a multiday regatta was easy while a small percentage (13% of men and 18% of women) reported they found it more difficult toward the end of the regatta. The researchers found more rowers to be in a hypohydrated state than anticipated considering their responses to the questionnaire regarding fluid restriction and/or sweat promotion strategies. It is speculated that the rowers were not aware that they are often in a hypohydrated state, or they did not want to disclose the use of dehydration techniques.

Boxers and Taekwondo Athletes

Boxers usually make use of extreme weight-loss strategies (i.e., meal skipping, fasting, restriction of fluid intake, and training with sweat suits) when making weight, also referred to as "drying out" (Morton et al., 2010; Smith et al., 2001). It has been reported that boxers lose up to 5% of BW in the 7 days preceding competition, but some have also reported to lose as much as 4% of BW 3 hours prior to official weigh-in (Smith, 1998; Smith et al., 2000). Unpublished data on elite taekwondo athletes competing in the 2004 European Championships showed that food restriction, increased exercise, use

of saunas, and fluid restrictions as well as special diets were used to make weight. On average, rapid weight gain of 1.5% BW in male and 1.3% BW in female athletes was reported, but some individuals gained up to 14% BW (Van Dijk et al., 2006).

Table 53.1 summarizes the number of weight categories and the weigh-in procedures for Olympic weight-category sports. It also highlights the duration and frequency of competitions.

Table 53.1 Weigh-in procedure of different weight-category sports

Sport	Duration and frequency	Number of weight categories	Weigh-in procedure
Wrestling Senior International (Greco-Roman and freestyle)	1 bout = 3 × 2 minute rounds. Each weight category is contested over 1 day. Not more than 4 bouts each day of competition. Competition session lasts 4 hours or less.	♂ = 7 ♀ = 7 ♂ = 4[a]	One weigh-in (30-minute period) on the evening before the tournament/competition starts.
Weightlifting	Two lifts must be executed in the following sequence: (a) the snatch; (b) the clean and jerk. A maximum of three attempts are allowed in each lift. Each weight category is contested over 1 day.	♂ = 8 ♀ = 7	One weigh-in in the morning. Weigh-in begins 2 hours before the start of the competition and lasts 1 hour.
Boxing Amateur	1 bout = 3 × 3 minute rounds. Competition every second day with 4–5 bouts during the tournament.	♂ = 11 ♀ = 13 ♂ = 3[a]	All boxers have weigh-in the morning of the first competition day. During tournaments, only those drawn to box have to weigh-in on the morning. There are at least 3 hours between weigh-in and start of the competition.
Judo	1 bout = 5 minutes. Each weight category is contested over 1 day. 4–5 bouts with minimum 10 minutes between bouts.	♂ = 8 ♀ = 7	Weigh-in on the morning of the competition (1-hour period). At least 2 hours between weigh-in and start of the competition.
Taekwondo	1 bout = 3 × 3 minute rounds. Each weight category is contested over one day. 5–8 bouts during competition.	♂ = 7 ♀ = 7 ♂ = 4[a] ♀ = 4[a]	Weigh-in on the evening before competition. One weigh-in at the start of competition.
Lightweight rowing	Race is over a 2000-m course (about 6–7 minutes). Compete every second day over a period of 7 days.	♂ (♀) Lightweight: Average weight of crew not more than 70 (57) kg. Individual weight more than 72.5 (59) kg not accepted. Maximum weight for a single sculler: 72.5 (59) kg.	Weigh-in each day and for each event. Weigh-in not less than 1 hour and not more than 2 hours before start of race.

[a]Olympic weight classes.

Performance, Health, and Nutritional Concerns

As illustrated in Figure 53.1, there are many possible nutritional, performance, and health concerns that could feature when athletes are making weight. The extent and severity of each of these will depend on the nature and extremity of the weight-loss strategy, the frequency of use, and time for which low mass is sustained, as well as the individual athlete's response to it. Many athletes report that they perceive their performance to be impaired by weight-loss efforts and yet almost all practice some kind of weight-loss technique annually. In this section, some of these nutritional, performance, and health concerns will be discussed in more detail than others since many have already been reviewed in detail elsewhere in this book.

Nutritional Concerns

Nutrition professionals who work with weight-category athletes must familiarize themselves with the rules of the sport, weight categories, weigh-in procedures, weight-loss strategies mostly used, and annual training cycles before attempting to develop a nutrition care plan. The athlete's nutrition needs should be individualized as they move in and out of season, and must be adapted according to their weight-loss needs. It is advised that a yearly weight management plan is implemented but that there is some room for weight gain during off-season. Weight-class athletes are encouraged to be no more than about 3% above their desired competition weight during the competition season. If changes in body composition are desired, these should be achieved during the off-season and while preparing for the competition season (see Chapter 41).

Rapid versus Gradual Weight Loss Gradual weight loss techniques are promoted during the pre-competition time (≤1.0 kg BW loss/week) to promote loss of body fat and maintenance of LBM, allowing possible performance impairments due to large food and fluid restrictions in the competition season to be minimized. Maintaining competition BW during tournaments is also easier when gradual weight loss is implemented before the start of the season since weight gain can be rapid after weigh-in, especially when dehydration techniques are used. There is some evidence to suggest that gradual weight loss improves muscle mechanical functioning indirectly since less dehydration is needed to make weight before weigh-in (Fogelholm et al., 1993; Garthe et al., 2011; Koral & Dosseville, 2009). Athletes should be encouraged to focus on low-fat, nutrient-dense foods during the off-season. More practical information on how to lose body fat and change body composition can be found in Chapter 41. Depending on the athlete's needs, the last 1–2 kg can be lost 1 day prior to weigh-in via RWL techniques in order to limit losses in LBM and thereby limit strength losses (Morton et al., 2010).

When making weight prior to weigh-in, athletes are encouraged to limit RWL to <3–4% BW (depending on time from weigh-in to competition and temperature during competition). This will help to minimize decrements in performance as well as symptoms related to RWL strategies such as dizziness, hot flashes, nausea, headache, and nosebleeds (Alderman et al., 2004). RWL (>1 kg weight loss/day) strategies are often implemented from 7 days to 24 hours before weigh-in.

Food Restrictions Decreasing food intake is often used by athletes competing at all levels to make weight, either through moderate or severe energy restrictions (Alderman et al., 2004; Oppliger et al., 2003). This often results in short-term nutrient shortages, losses in LBM, and glycogen depletion (Sundgot-Borgen & Garthe, 2011). Prolonged periods of energy restriction might result in decreased nutritional status and increase the risk for various adverse health outcomes. Even though weight-category sports rely mostly on anaerobic energy systems, with the exception of lightweight rowing, carbohydrate availability during competition and key training sessions remain important. Insufficient carbohydrate intake is associated with decreased aerobic performance mostly due to decreased glycogen levels, but has also been linked with decrements in muscle strength, increased rate of perceived exertion, and negative mood profile (increased confusion, anger, tension, and fatigue, decreased vigor) (Burge et al., 1993; Houston et al.,

1981; Koral & Dosseville, 2009). Following a high-carbohydrate diet (60–70% of energy intake or about 3 g/kg BW/day) while on an energy-restricted budget can help to maintain performance levels (Burke et al., 2011; Horswill et al., 1990; Rankin et al., 1996). Higher protein intakes can help to maintain LBM while restricting energy intake and promoting a greater loss in body fat, and daily protein intakes of 1.2–2.3 g/kg BW are recommended (Tipton, 2011).

Fluid Restrictions Dehydration is often used as an RWL strategy, but is associated with various health and performance risks (i.e., reduced muscle strength and aerobic performance, impaired thermoregulation), which are covered in more detail in Chapter 15. Athletes that make use of dehydration techniques should be warned to limit weight loss to not more than 2–3% BW in order to minimized performance decrements, but also to enable complete rehydration before competition starts. Including sodium in the recovery beverage is important to help facilitate replacement of lost electrolytes as well as fluid absorption and maintain the thirst drive (see Chapters 14 and 16 for more information on rehydration guidelines) (Burge et al., 1993; Slater et al., 2006).

Performance Concerns

Inconsistent results regarding the effect of RWL strategies on performance outcomes are found in the literature and there are different possible explanations for this variance, some of which include laboratory-based versus field-based settings and performance tests, small sample sizes, magnitude of weight loss, age of athletes, different periods of dehydration and/or energy restriction, different RWL techniques being used, and lack of rehydration/recovery. In general, there seems to be a consensus that performance decrements can be minimized if the competition weight differs by less than 3–4% from the pre-RWL BW. The period after weigh-in and before competing is, therefore, a crucial refeeding and recovery opportunity, as the risk for decreased performance is much greater when no recovery takes place after weigh-in (Artioli et al., 2010; Koral & Dosseville, 2009; Slater et al., 2005b). It should also be noted that many studies investigating the effect of RWL strategies on performance and mood outcomes are done on experienced athletes that often make use of weight cycling. Thus, even though some of these studies found no or a small but not-significant effect of RWL on measured performance outcomes, this might not be the case when working with athletes not accustomed to weight cycling.

Mood Profile and Rating of Perceived Exertion Mood plays an important role in the performance of athletes competing in sports with short and intense bursts of play/combat combined with a high level of open skill and in sports judged by self-referenced criteria (Beedie et al., 2000). Decrements in mood profile have been shown among weight-category athletes who have lost more than 3–4% BW through RWL techniques. Changes in psychological factors include increased confusion, anger, tension, fatigue, rating of perceived exertion (RPE) with concomitant decrements in vigor (Hall & Lane, 2001; Horswill et al., 1990; Koral & Dosseville, 2009; Marttinen et al., 2011). Some may, however, perceive an increase in anger as a positive psychological change, especially when competing in combat sports such as boxing and karate. Koral and Dosseville (2009) investigated the effect of a combined weight-loss strategy (21 days on gradual weight loss and 6 days on RWL program) on judo performance and mood among elite male and female judo athletes before a national championship. After 3.9% BW loss, there was a significant increase in confusion and tension, as well as a decrease in vigor among athletes compared with baseline levels, which is similar to what others found after RWL. In contrast to others, fatigue and anger levels did not increase, which suggests that this combined method of weight loss could affect mood to a lesser extent. It should, however, be noted that the final measurement of mood profile took place one day prior to the championship and not immediately before competing.

Strength, Aerobic, and Anaerobic Performance There are reports in the literature that anaerobic performance, as well as concentrations of adenosine triphosphate and phosphocreatine

in skeletal muscle are not reduced after dehydration (Houston et al., 1981), and that the excitability and recruitment of muscle fibers is not affected by dehydration (Costill et al., 1976). This, in combination with evidence that high-intensity, short duration exercise is not dependent on oxygen and glucose delivery (Horswill, 1992; Horvath & Horvarth, 1973), can be used as arguments that RWL strategies involving short-term fluid and food restrictions will not negatively influence strength, stamina, and anaerobic performance of athletes competing in events that involve maximum efforts of short duration. Recent studies (Smith et al., 2000, 2001) among amateur boxers have found that RWL of about 3–4% BW through food and fluid restrictions or exercise-induced thermal dehydration did not result in significant decrements in boxing performance tasks. However, the sample size of these studies was small (n = 7/8) and variations in individual performances were often large, so these findings should be interpreted with care. Interestingly, after power and effect size analysis using η^2 procedures, there was a mean fall in boxing performance of 27% after RWL caused by exercise-induced thermal dehydration (Smith et al., 2000). Similarly, no differences were found in anaerobic performance or strength after RWL (about 3–8% BW), with or without a recovery period among wrestlers and judo athletes (Artioli et al., 2010; Fogelholm et al., 1993; Koral & Dosseville, 2009; Marttinen et al., 2011) while others found decrements in strength and anaerobic performance (Horswill et al., 1990; Houston et al., 1981; Webster et al., 1990). Koral and Dosseville (2009) did find a significant decrease in 30-second repetitions of judo movements, which is of concern since the average confrontation time in competition conditions is usually 30 seconds interspersed with about 10 seconds rest. Gutiérrez et al. (2003) found that sauna-induced weight loss of about 1.5 kg among male and female non-weight-category athletes not accustomed to making weight, did not affect strength or jumping performance in the men but significantly decreased jumping performance in the women. These tests were undertaken after a 1-hour recovery period, during which athletes were not able to restore BW losses fully.

RWL (about 4–6% BW) without sufficient recovery has shown to impair rowing performance (Burge et al., 1993), but the magnitude in 2000-m rowing performance decrement was decreased substantially in two studies by Slater et al. (2005b, 2006) when RWL was followed by an aggressive nutrition recovery strategy (see Table 53.2). Furthermore, when this strategy was implemented during a multiday regatta to facilitate recovery of at least three quarters of the BW loss experienced in the 24 hours before weigh-in (recovery period = 12–16 hours), the initial small negative effect on performance was eliminated when repeated over several days (Slater et al., 2006). It is important to note that both dehydration and exercising in the heat were found to compromise rowing performance independently and when they are combined to exacerbate performance decrements further (Slater et al., 2005b).

Health Concerns

Disordered Eating and Eating Disorders Research shows that even though weight-category athletes often make use of pathogenic BW control measures, during season while making weight, this is transient and more related to the culture of the sport (Artioli et al., 2010; Dale & Landers, 1999). There is, however, evidence that those athletes who start to make weight at an early age (about 14 years) are prone to using more extreme methods of weight management (Oppliger et al., 2003). It can also not be ruled out that repeated cycles of making weight can result in the development of disordered eating (DE) patterns in some athletes (Dale & Landers, 1999; Steen & Brownell, 1990). Competing in unrealistic weight categories and/or increased pressure from coaches/trainers, parents, and/or peers to maintain an unrealistic, low BW can trigger episodes of DE behavior. This may progress into a clinical eating disorder (ED) in a small number of athletes (for more detailed information on risk factors, signs, prevention, and treatment of DE/ED, see Chapter 42).

Reproductive Function and Bone Health Sporadic episodes of low energy availability due to

increased exercise energy expenditure and/or decreased energy intake are often part of the competition season weight-category athletes as they go through weight cycling (Slater et al., 2005a). Chronic periods of low energy availability (<30 kcal/kg fat-free mass/day) can result in decreased levels of reproductive hormones in men and women, disruptions in the menstrual cycle, and decreased bone formation in as little as 5 days. The long-term health consequences are not clear, but irreversible bone loss, decreased bone mass, and increased stress fracture risk have been shown among some athletes (Loucks et al., 2011; see Chapter 5). Ingesting sufficient energy to match energy expenditure is essential to maintain reproductive and bone health. In Chapter 5, energy availability and how it relates to the athlete's health is discussed in more detail, while Chapter 20 refers to the importance of adequate dietary calcium intake and vitamin D status for bone health.

Immune Function Micronutrient deficiencies and undernutrition are associated with compromised immune function and, when combined with heavy training sessions and competition, put athletes at increased risk of developing exercise-induced immune function impairment (Gleeson et al., 2004; see Chapter 39). Although most studies on exercise-induced immune depression have been conducted among endurance-type athletes, weight-category athletes who go through cycles of very low energy and nutrient intake, RWL, very low levels of body fat and increased periods of exercise may also be at risk. How this will translate into actual infection risk among these athletes is, however, not clear.

Practical Considerations

Before Weighing-In

If weigh-in is in the morning (8–10 a.m.), there is no need for breakfast before weighing-in starts. However, if weigh-in is scheduled in the evening, there are more concerns around food intake during the day. We suggest that athletes restrict the total volume of food consumed during the day by focusing on energy-and-carbohydrate-dense foods, and have a short exercise bout before weigh-in, rather than fasting before weigh-in. This may limit some of the reported negative consequences (e.g., muscle glycogen depletion) associated with fasting. Examples of compact, energy-and-carbohydrate-dense foods include sports bars, carbohydrate gels, sports drinks, fruit smoothies, dried fruits, and yoghurt-covered rice cakes.

Recovery Strategies

Evidence on the effect of RWL strategies on performance is contradictory, as described earlier. There is, however, evidence that aggressive nutritional recovery strategies after weigh-in, focusing on high carbohydrate, sodium, and fluid intake can limit performance decrements associated with RWL (Rankin et al., 1996; Slater et al., 2006). These nutritional recovery strategies may not be applicable for all weight-category athletes due to the characteristics of the sport (e.g., rapid movements) and individual tolerance, which might result in possible gastrointestinal discomfort during competition due to the ingestion of high volumes of food and/or fluid. Furthermore, there is evidence to suggest that a less aggressive nutritional recovery strategy can also prevent performance impairment of interval-related tasks (Fogelholm, 1993; Hall & Lane, 2001) seen for example in boxing. The main focus during the recovery period should be a high-carbohydrate diet: intakes of 200–275 g carbohydrate within 4–5 hours have been documented (Artioli et al., 2010; Rankin et al., 1996). It is important that every athlete experiment with various nutritional recovery strategies during less important competitions or training in order to find the optimum recovery strategy that fits the unique needs of the athlete considering the type of sport, RWL strategies used, amount of BW loss, and rules of weigh-in. Practical recovery strategies grouped according to recovery time are discussed below.

1–2 hours (e.g., lightweight rowing)

- If dehydration techniques were mainly used, recovery of fluid losses is essential. Replace 150% of BW loss with fluid intake. Fluids should contain sufficient sodium (50—60 mmol/liter) to facilitate fluid absorption and limit urinary output. Alternatively, water and sodium-rich foods

can be ingested to restore hydration levels (see Chapter 16).
- Glycogen levels can be recovered by ingesting easily digested, carbohydrate-rich foods (see example in Table 53.2). Athletes need to find the balance between sufficient intake to hinder performance decrements and what is tolerated (gastrointestinal comfort and taste preference).
- Stay hydrated and maintain blood glucose levels by snacking every 1–3 hours, when waiting for the next heat/round.
- Limit the possible negative effect that a hot and humid environment can have on performance by incorporating cooling strategies, e.g., drinking slushies or using cold towels.

2–5 hours (e.g., judo, boxing)
- Similar nutritional recovery strategies can be used with 1–2 hours of recovery; emphasis is placed on gastrointestinal comfort (e.g., due to rapid movements) when considering type and volume of food and fluids ingested.
- Due to longer recovery time, athletes can experiment with larger volumes of fluids and easily digested carbohydrate-rich solid foods.
- Full rehydration and restoration of BW is not possible with >4% BW loss.

>12 hours (e.g., wrestling, taekwondo, multiday regatta)
- Aggressive nutritional recovery should be implemented and aimed at restoring at least three-quarters of RWL.
- Frequent meals containing (preferably high glycemic index) carbohydrate-rich foods should be ingested.
- 1.0–1.2 g carbohydrate/kg BW/h for the first 4 hours can enhance the rate of muscle glycogen recovery, or combine 0.8 g carbohydrate/kg BW/h and 0.4 g protein/kg BW/h for a similar rate in muscle glycogen recovery while keeping food volumes low.

Conclusion

Professionals in nutrition can find themselves in a dilemma when working with weight-category athletes. On the one hand, they know the risks of RWL strategies and want to act ethically when interacting with these athletes, but on the other hand, they want their athletes to make a given weight category and they are faced with a strong tradition of RWL that is an inherent part of the sport. It is often a balancing act between helping athletes to make weight in the safest possible way and without compromising

Table 53.2 Example of an aggressive nutritional recovery strategy for a 65-kg, lightweight rower

34 mg sodium/kg BW = ~2200 mg sodium
~30 ml fluid/kg BW = ~2.0 liter fluid
2.3 g carbohydrate/kg BW = ~150 g carbohydrate

Practical food and drink choices

Examples of food and beverage intake	Estimated fluid (ml)	Estimated carbohydrate (g)	Estimated sodium (mg)
2 × 1 liter sports drink (~5% solution with electrolytes) and added sodium corresponding to 400 mg (e.g., 2 salt tablets or electrolyte drops in sports drink)	2000	108	1420
3 thick slices of white bread with jam and honey		55	400
Total intake	2000[a]	163	1820

Source: From Slater et al. (2006).

[a]Ingest a larger volume initially, followed by progressive smaller volume as competition approaches.

Other carbohydrate-rich examples that can be experimented with include carbohydrate gels, sports bars, raisins, dried fruits, and bananas. Alternatives to replace sodium include adding extra salt (1 level teaspoon) to recovery food or using salty spreads, e.g., vegemite on bread.

health and performance. Gaining the trust of the coach/trainer and the athlete is probably the most important success factor of any nutrition care plan.

Summary and Recommendations

- Before a nutritional care plan is designed, athletes must undergo a thorough screening including weight history, weight cycling, menstrual history for females, nutritional status, dietary patterns and behavior, and thoughts and feelings about body image, BW, and toward food.
- A 4-day (3 training days and 1 non-training day) or a 7-day diet record (using weighed or household measures) should be conducted and used as the basis for an individualized nutritional care plan.
- Body composition measurements must be done with accurate and reliable tools while in a euhydrated state. Body weight and body composition goals should be realistic and not hamper performance and health outcomes. See Chapter 6 for issues regarding the assessment of body composition.
- Change in body composition should be monitored on a regular basis including a period of at least 2 months after body composition goal(s) has been reached to detect any continued or unwarranted losses or weight fluctuations.
- Body composition changes should be made during off-season to avoid interference with competitions and sport-specific training loads, as this may create stress or overload for the athlete. Additionally, optimal results may also not occur due to conflicting training stimuli.
- Athletes are encouraged to be within 3% of their competition weight during off-season and to limit RWL to <3–4% BW (depending on time from weigh-in to competition and recovery strategies).
- Athletes under the age of 18 should be discouraged from making weight.
- Athletes with a history of menstrual dysfunction and/or stress fracture should be referred for a bone mineral density (BMD) test.
- Athletes should aim for gradual weight loss of 0.5 kg/week. To induce this, an energy deficit of about 2100 kJ (500 kcal) per day is needed. Athletes, especially female athletes, should, however, consume sufficient energy to avoid adverse health consequences (see Chapter 5).
- A multivitamin-mineral supplement and omega-3 fatty acid supplementation can be considered for those that are on a tight energy budget to reduce the risk of micronutrient deficiencies.
- Athletes wanting to make weight should be educated on choosing low-energy, nutrient-rich foods that promote satiety. A variety of foods, frequent meals, hydration, and combining carbohydrate and protein after training should be emphasized for optimum nutrition, to prevent premature fatigue, and to promote recovery during training sessions.
- Depending on the athlete's body composition goals, protein intake can vary between 0.8 and 2.3 g protein/kg BW, carbohydrate intake can be about 3 g/kg BW and fat less than 15% total daily energy intake.
- To limit loss in LBM, combining resistance training with a high-protein diet is recommended.
- For some athletes, it can be beneficial in terms of health and performance, to gain LBM and move up a weight class. The weight gain period should be controlled and stabilized after the athlete has reached his/her goal.
- As athletes mainly consult coaches on making weight, coaches should also be educated on negative health and performance consequences due to inappropriate weight loss strategies, as well as the importance of a yearly weight management plan.

Suggested Modifications in Regulations to Reduce Use of RWL Strategies

- In sports such as wrestling, judo, and taekwondo daily weigh-in sessions are recommended as well as first weigh-in to be less than 2–3 hours before competition.
- Sports federations should be encouraged to have similar weight categories at national and international competitions, as well as more weight categories, especially in the low- and middle-weight classes.
- Changing to a lower weight class during season should not be accepted. This could lead to smaller

weight fluctuations/fewer occasions of weight fluctuation if athletes have to compete in the same weight class throughout the season.

- Accepting weight allowance (e.g., 1–2 kg over weight limit) in smaller tournaments might reduce the frequency of weight cycling in a season.
- Federations should enforce the principle of a "competition certificate" where athletes are measured for a minimum accepted body fat level and a safe hydration level. More research is, however, needed to set these healthy levels and which methods to use in assessing these measurements.

References

Alderman, B.L., Landers, D.M., Carlson, J., et al. (2004) Factors related to rapid weight loss practices among international-style wrestlers. *Medicine & Science in Sports & Exercise* 36(2), 249–252.

American College of Sports Medicine [ACSM] (1976) Position stand on weight loss in wrestlers. *Medicine & Science in Sports & Exercise* 8(2), xi-xiii.

American Medical Association [AMA] (1967) Wrestling and weight control. *Journal of the American Medical Association* 201(7), 131–133.

American Medical Association [AMA] (1998) From the Centers of Disease Control and Prevention. Hyperthermia and dehydration-related deaths associated with intentional rapid weight loss in three collegiate wrestlers—North Carolina, Wisconsin, and Michigan, November–December 1997. *Journal of the American Medical Association* 279(11), 824–825.

Artioli, G.G., Iglesias, R.T., Franchini, E., et al. (2010) Rapid weight loss followed by recovery time does not affect judo-related performance. *Journal of Sports Sciences* 28(1), 21–32.

Beedie, D., Terry, P.C., & Lane, A.M. (2000) The profile of mood states and athletic performance: two meta-analyses. *Journal of Applied Sport Psychology* 12(1), 49–68.

Burge, C.M., Carey, M.F., & Payne, W.R. (1993). Rowing performance, fluid balance, and metabolic function following dehydration and rehydration. *Medicine & Science in Sports & Exercise* 25(12), 1358–1364.

Burke, L.M., Hawley, J.A., Wong, S.H.S., et al. (2011) Carbohydrates for training and competition. *Journal of Sports Science* 29, S17–S27.

Costill, D.L., Cote, R., & Fink, W. (1976) Muscle water and electrolyte following varied levels of dehydration in man. *Journal of Applied Physiology* 40, 6–11.

Dale, K.S. & Landers, D.M. (1999) Weight control in wrestling: eating disorders or disordered eating? *Medicine & Science in Sports & Exercise* 31(10), 1382–1389.

Davis, S.E., Dwyer, G.B., Reed, K., et al. (2002) Preliminary investigation: the impact of the NCAA wrestling weight certification program on weight cutting. *Journal of Strength and Conditioning Research* 16, 305–307.

Fogelholm, M.G., Koskinen, R., Laakso, J., et al. (1993) Gradual and rapid weight loss: effects on nutrition and performance in male athletes. *Medicine & Science in Sports & Exercise* 25(3), 371–377.

Garthe, I., Raastad, T., Refsnes, P.E., et al. (2011) Effect of two different weight-loss rates on body composition and strength and power-related performance in elite athletes. *International Journal of Sport Nutrition and Exercise Metabolism* 21(2), 97–104.

Gleeson, M., Nieman, D.C., & Pedersen, B.K. (2004) Exercise, nutrition and immune function. *Journal of Sports Sciences* 22, 115–125.

Gutiérrez, A., Mesa, J.L.M., Ruiz, J.R., et al. (2003) Sauna-induced rapid weight loss decrease explosive power in women but not men. *International Journal of Sports Medicine* 24, 518–522.

Hall, C.J. & Lane, A.M. (2001) Effects of rapid weight loss on mood and performance among amateur boxers. *British Journal of Sports Medicine* 35, 390–395.

Horswill, C.A. (1992) Applied physiology of amateur wrestling. *International Journal of Sports Medicine* 14, 114–143.

Horswill, C.A., Hickner, R.C., Scott, J.R., et al. (1990) Weight loss, dietary carbohydrate modifications and high intensity, physical performance. *Medicine & Science in Sports & Exercise* 22, 470–476.

Horvath, S.M. & Horvath, E.C. (1973) *The Harvard Fatigue Laboratory: Its History and Contributions*. Prentice-Hall, Englewood Cliffs, NJ.

Houston, M.E., Marrin, D.A., Green, H.J., et al. (1981) The effect of rapid weight loss on physiological functions in wrestlers. *Physician and Sportsmedicine* 9, 73–78.

Koral, J. & Dosseville, F. (2009) Combination of gradual and rapid weight loss: effects on physical performance and psychological state of elite judo athletes. *Journal of Sports Sciences* 27(2), 115–120.

Loucks, A.B., Kiens, B., & Wright, H.H. (2011) Energy availability in athletes. *Journal of Sports Sciences* 29, S7–S15.

Marttinen, R.H., Judelson, D.A., Wiersma, L.D., et al. (2011) Effects of self-selected mass loss on performance and mood in collegiate wrestlers. *Journal of Strength and Conditioning Research* 25(4), 1010–1015.

Morton, J.P., Robertson, C., Sutton, L., et al. (2010) Making the weight: a case study from professional boxing. *International Journal of Sport Nutrition and Exercise Metabolism* 20, 80–85.

Oppliger, R.A., Case, H.S., Horswill, C.A., et al. (1996) American College of Sports Medicine position stand: weight loss in wrestlers. *Medicine & Science in Sports & Exercise* 28(6), ix–xii.

Oppliger, R.A., Steen, S.A.N., & Scott, J.R. (2003) Weight loss practices of college wrestlers. *International Journal of Sport Nutrition and Exercise Metabolism* 13, 29–46.

Rankin, J.W., Ocel, J.V., & Craft, L.L. (1996) Effect of weight loss and refeeding diet composition on anaerobic performance in wrestlers. *Medicine & Science in Sports & Exercise* 28, 1292–1299.

Slater, G.J., Rice, A.J., Sharpe, K., et al. (2005a) Body-mass management of Australian lightweight rowers prior to and during competition. *Medicine & Science in Sports & Exercise* 37(5), 860–866.

Slater, G.J., Rice, A.J., Sharpe, K., et al. (2005b) Impact of acute weight loss and/or thermal stress on rowing ergometer performance. *Medicine & Science in Sports & Exercise* 37(8), 1387–1394.

Slater, G.J., Rice, A.J., Tanner, R., et al. (2006) Impact of two different body mass management strategies on repeat rowing

performance. *Medicine & Science in Sports & Exercise* 38(1), 138–146.
Smith, M.S. (1998) *Sport specific ergometry and the physiological demands of amateur boxing*. Thesis. University College, Chichester, UK.
Smith, M.S., Dyson, R., Hale, T., et al. (2000) The effects in humans of rapid loss of body mass on a boxing-related task. *European Journal of Applied Physiology* 83, 34–39.
Smith, M., Dyson, R., Hale, T., et al. (2001) The effects of restricted energy and fluid intake on simulated amateur boxing performance. *International Journal of Sport Nutrition and Exercise Metabolism* 11, 238–247.
Steen, S.N. & Brownell, K.D (1990) Patterns of weight loss and regain in wrestlers: has the tradition changed? *Medicine & Science in Sports & Exercise* 22(6), 762–768.
Sundgot-Borgen, J. & Garthe, I. (2011) Elite athletes in aesthetic and Olympic weight-class sports and the challenge of body weight and body composition. *Journal of Sports Sciences* 29, S101–S114.
Tipton, K.D. (2011) Conference on 'Nutrition and health: cell to community'. Symposium 2: exercise and protein nutrition. Efficacy and consequences of very-high-protein diets for athletes and exercisers. *Proceedings of the Nutrition Society* 70, 205–214.
Tipton, C.M. & Tcheng, T.K. (1970) Iowa wrestling study. Weight loss in high school students. *Journal of the American Medical Association* 214(7), 1269–1274.
Tuttle, W.W. (1943) The effect of weight loss by dehydration and the withholding of food on the physiologic responses of wrestlers. *Research Quarterly* 14, 158–166.
Van Dijk, F., Garthe, I., & Wisnes, A (2006) *Rapid Weight Loss Practices Among Elite Taekwondo Players*. Publication of the European Taekwondo Union, Oldenzaal, The Netherlands.
Webster, S., Rutt, R., & Weltman, A. (1990) Physiological effects of a weight loss regime practiced by college wrestlers. *Medicine & Science in Sports & Exercise* 22, 229–234.
Wrestling Rules Committee (1999) *NCAA Wrestling Rules*. NCAA Publications, Indianapolis, IN.

Index

3D photonic scanning, 94–5

A-band of muscle fibers, 26
accelerometers, 67–70
acclimation, 28, 30
acclimatization, 28
acetyl coenzyme A (acetyl-CoA), 38–9, 43–5
N-acetylcysteine (NAC), 267, 273
actin filaments, 27, 37
activating transcription factor-2 (ATF-2), 472
activities, energy cost of, 4
acute mountain sickness (AMS), 429, 534
acyl-CoA-binding protein (ACBP), 158
adenosine monophosphate (AMP), 40, 48, 49
adenosine triphosphate (ATP)
  conversion to ADP
    fatigue, 324–5
    glycolysis, 40
    hydrolysis, 38
    intracellular factors, 48–50
    maximal rates of ATP resynthesis, 40
    muscle contraction, 37, 38
    phosphocreatine (PCr) breakdown, 39–40
    resynthesis, 38–9
  glycolytic pathway from G6P to pyruvate, 41–3
  heat conversion, 28
  muscles, 27–8
adipokines, 468
adipose tissue, 47–8, 88
  chronic low-grade inflammation, 468
  subcutaneous (SAT), 90, 93
adipose triacylglycerol (ATGL), 158, 171
adrenaline, 50–51
aerobic metabolism
  amino acid oxidation, 47
  carbohydrate oxidation, 43–5
  dehydration, effect of, 187–8
    endurance exercise, 188–93
  fat oxidation
    fatty acid (FA) uptake by muscle, 46–7
    lipolysis, 45–6
  pre-exercise hypohydration, effect of, 193–4
aging athletes, 369–71, 379–80
  nutritional considerations, 373
    carbohydrates, 374
    energy needs, 373–4
    macronutrient summary, 376
    micronutrients, 375–8
    proteins, 375
  special considerations
    dietary supplement use, 379
    hydration, 378
    nutrient–drug interactions, 378–9
  sports participation and physiological effects
    body weight, 373
    bone mineral density, 372–3
    cardiorespiratory function and cardiovascular disease risk, 371–2
    health benefits, 373
    insulin resistance, 373
    muscle function, 372
air displacement plethysmography (ADP), 92
air quality issues, 435–7
  primary air pollutants, 436
airway hyperreactivity (AHR), 435, 436
β-alanine, 23
  buffering supplement, 327–30
  carnosine elevation, 43
  strength and power events, 558
alcohol, 336–7, 342
  aftermath of use, 341
  diuretic effects, 182–3, 339–40
  glycogen metabolism, 338–9
  hydration and thermoregulation, 339–41
  injuries and incapacity, effect upon, 341–2
  oxidative stress, 339
  performance, 337
  water of oxidation, 176
altitude effects, 427
  acute mountain sickness (AMS), 429, 534
  adaptation to, 427–9
  exercise performance, 432–5
amenorrheic athletes (AmA), 76, 78, 81, 82, 349
  vegetarian diets, 388–9
amino acids
  essential (EAA), 9–10, 148, 149
  gluconeogenesis, 7

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amino acids *(continued)*
limitations in vegetarian diets, 386
metabolism, 8–9
oxidation in aerobic metabolism, 47
requirement, 9–11
ammonia implants, 556
AMP-activated protein kinase (AMPK), 55–6
amputees, 395
anabolic drive, 137
anaerobic metabolism, 51
pre-exercise hypohydration, effect of, 194–5
T1DM, 492–3
androstenediol, 555
androstenedione, 555
anemia, 25
see also iron deficiency
iron-refractory iron-deficiency anemia (IRIDA), 231
sports anemia, 233
anorexia nervosa (AN), 83, 513, 514
diagnostic criteria, 514
anorexigenic hormones, 84
anovulation, 349
anthocyanin, 283
anthropometry, 95–8
antioxidant capacity, 271
antioxidant response element (ARE), 459
antioxidants, 265, 274
balancing performance and redox regulation
antioxidant deficiencies, 272
muscle as redox sensing organ, 273–4
muscular contractions, 272–3
ROS-induced muscle fatigue, 273
enzymatic, 265–6
immunonutrition support, 482
nonenzymatic, 266–9
vegetarian diets, 384
appetite suppression, 83–4
aromatase inhibitors, 379
ascorbic acid (vitamin C), 13
as an antioxidant, 267
antioxidant properties, 56
dietary sources, 219
supplementation, 224
ATPases, 27–8
muscle contraction, 37

banned substances, 555–6
baroreceptors, 25
basal metabolic rate (BMR), 3
estimation from physical attributes, 4
measurement, 3–6
basement membrane, 26
bergamottin, 378
bicarbonate, labeled, 64
biochemistry, 36
energy for muscle force generation
amino acid oxidation in aerobic metabolism, 47
ATP hydrolysis, 38
ATP resynthesis, 38–9, 302–5
buffering excessive pH falls, 43
carbohydrate oxidation in aerobic metabolism, 43–5
fat oxidation in aerobic metabolism, 45–7
glucose uptake from blood, 40–41
glycogen breakdown, 41
glycolysis, 40
glycolytic pathway, 41–3
phosphocreatine breakdown, 39–40
energy metabolism regulation, 48
hormones and cytokines, 50–51
intracellular factors, 48–50
fuel stores in the body, 47–8
metabolic adaptations to exercise
endurance training, 55–6
strength training, 56–7
metabolic response to exercise, 51
high-intensity exercise, 51–2
prolonged exercise, 52–5
skeletal muscle, 36
contraction, 36–7
fiber types, 37–8
bioelectrical impedance analysis (BIA), 98–9
biological value (BV), 10–11
biotin (vitamin H), 13
blood
circulation, 22
hemoglobin concentration, 23
pH, 23
plasma osmolality, 176, 177
pressure, 25
red blood cells (RBCs), 21, 22, 23
total volume, 24
blood doping, 25
body composition assessment, 88–9, 99–100
technique comparison, 89–90
techniques, 90
field methods, 95–9
laboratory methods, 90–95
body mass, definition, 62
body mass index (BMI), 90, 99
body surface area (BSA), 285–6
body temperature, 28
measurement for indirect calorimetry, 67
bovine colostrum, 485
Boyle's gas law, 22
brain-derived neutrotrophic factor (BDNF), 474
brain responses to oral carbohydrate, 127
branched-chain amino acids (BCAAs), 121–2, 207
influences on central fatigue, 207–8
breathing, 21
buffering agents, 324–7, 332–3
summary table, 333
supplements
β-alanine/carnosine, 327–30
creatine (Cr)/phosphocreatine (PCr), 327
supplements, extracellular
sodium bicarbonate, 330–31
sodium citrate, 331–2
bulimia nervosa (BN), 513, 514
diagnostic criteria, 514

caffeic acid, 279
caffeine, 313, 321
  distance running, 582
  diuretic effects, 182–3
  ergogenic effects, 314–15
    central drive and prolonged exercise, 315–16
    central nervous system (CNS), 315
    exercise performance improvement, 316
    low dose studies in humans, 316–18
  fluid balance, temperature regulation and heat tolerance, 321
  habitual consumption, 320–21
  influences on central fatigue, 211
  legal supplement, 314
  metabolic effects, 318–19
    intestinal uptake and glucose oxidation, 319
    muscle glycogen resynthesis, 319–20
  performance-enhancing substance, 556
  research evolution, 313–14
  supplementation for glycogen storage, 122–3
calcidiol, 220
calciferol (vitamin D), 13, 242, 259
  aging athletes, 376–8
  clinical assessment, evaluation and treatment, 256–9
  dietary sources, 219, 247–8, 249
  functions, 252–3
    bone health, 253
    immunity and inflammation, 254–5
    skeletal muscle function, 253–4
  intake, 250–52
  intoxication, 256
  requirements for athletes, 256
  special needs athletes, 400
  status in athletes, 251–2
  status indices, 249–50
  supplementation, 220–21
  synthesis, 249
  young athletes, 364–5
calcitriol, 242
calcium, 242, 259
  absorption, 242–3
  aging athletes, 377
  balance, 12
  clinical assessment, evaluation and treatment, 256–9
  dietary sources, 242–3, 247–8
  functions, 11, 244
    bone health, 244–5
    signaling, 245
  intake, 243–4
  recommended intake, 246
  requirements for athletes, 245–8
  status indices, 243
  toxicity and adverse effects, 248–9
  vegetarian diets, 388, 389
  young athletes, 364
calorimeters, 3–5
capillary diffusion, 21
carbohydrates (CHO), 102
  aging athletes, 374
  carbon dioxide produced from oxidation, 5
  daily requirements for training, 102
    IOC guidelines, 106
    low carbohydrate availability, 107–9
    practicing competition nutrition strategies, 107
    refueling, 102–7
    refueling, updated principles, 104–5
    strategies to reduce carbohydrate availability, 108
  energy yield from oxygenation, 5, 167
  fatigue, influences upon, 210
  future research directions, 110–11
  gastrointestinal (GI) tract, effects upon, 529–30
  glycogen storage supplementation
    amount and frequency, 119
    timing, 118–19
    type and form, 119–20
immunonutrition support, 481–2
inflammation, 471–2
ingestion during exercise, 54–5, 126, 133–4
  brain responses to oral carbohydrate, 127
  high-intensity exercise, 126–8
  implications, 127–8
  intermittent and skill sports, 128–9
  mechanisms, 127
  prolonged exercise, 129
interleukin-6 (IL-6), role in metabolism, 473
as metabolic fuel, 7–9
optimizing delivery, 129
  better oxidation efficiency, 132
  individual differences, 132
  multiple transportable carbohydrates, 130
  multiple transportable carbohydrates, effects on exercise performance, 130–31
  practical implications, 132–3
  training the gut, 131–2
  types of carbohydrates, 129–30
oxidation in aerobic metabolism, 43–5
oxygen consumed by oxidation, 5
promoting glycogen storage, 109–10
proportion of energy intake, 7
respiratory quotient, 5
role in evolution of sports nutrition, 103
special needs athletes, 398
sprinting, 562–4
strength and power events, 552
water of oxidation, 176
water replacement, 202
young athletes, 361–2, 363

carbon dioxide production (VCO_2), 64
carbon dioxide transport, 21, 22–3
  partial pressure, 22
carbon monoxide (CO), 435, 436
cardiac muscle, 24, 25
cardiac output, 23–4
cardiometabolic disease (CMD), 456
cardiorespiratory function, 21–5
cardiovascular disease (CVD), 456, 461
carnitine, 46
  long-chain acyl carnitine (LCAC), 160
carnitine acyltransferase (CAT), 46
carnitine palmitoyltransferase 1 (CPT-1), 162, 170–71
carnosine, 43
  buffering supplement, 327–30
β-carotene, 13, 219
  as an antioxidant, 268
carotenoids, 268
catalase (CAT), 265–6
catechins, 268–9
catecholamines, 50–51
cerebral cortex, 29
cerebral palsy (CP), 395
  energy expenditure, 396
chemical score, 11
chemoreceptors, 25
children, *see* young athletes
cholesterol, 8
chromium
  aging athletes, 377
  dietary sources, 224
  function, 11
  supplementation, 224–5
chronic obstructive pulmonary disease (COPD), 436
circulating angiogenic cells (CACs), 462
cis-fats, 7
citric acid cycle, 43–5
clenbuterol, 555
cobalamin (vitamin B_{12}), 13
  aging athletes, 377
  vegetarian diets, 387
cobalt, function, 11
coenzyme Q, 268
cold stress, 28
commercial weight loss and meal replacement programs, 510
complex carbohydrates, 18
conduction of heat, 28
convection of heat, 28–9
copper, function, 11
Cori cycle, 30
cortisol, 51
C-reactive protein (CRP), 467, 479
creatine (Cr), 301
  buffering supplement, 327
  dietary supplementation for aging athletes, 379
  efficacy of supplementation
    increase in exercise performance, 307–9
    increase in muscle content, 305–7
    other effects, 309–10
  need for supplementation
    creating homeostasis, 301–2
    metabolic role within skeletal muscle, 302–5
  safety considerations, 310–11
  strength and power events, 557–8
  vegetarian diets, 384–5
creatine kinase (CK), 40, 303, 304–5
critical difference (CD), 235
cultural eating styles, 445
curcumin, 279, 282, 485
cycling, 594
  during exercise
    events lasting <60 minutes, 586–7
    events lasting >60 minutes, 587–9
  energy balance and weight control
    muscle building, 593–4
    muscle mass maintenance, 593
    weight reduction, 592–3
  Olympic disciplines, 584
  one-day race nutrition plan, 591
  preparing for competition
    carbohydrate ingestion following last meal, 585–6
    hydration, 586
    muscle glycogen loading, 584–5
    pre-competition meal, 585
  recovery, 589–90
  stage races, 590–92
    nutrition plan, 591
cytokine sickness, 404
cytokines, 50–51
cytokine theory of UUPS, 404–6

dehydration, 176–7, 178–9
  see also water; water replacement
  performance effects, 185–6, 196
    aerobic exercise, 187–93
    fluid intake guidelines, 186–7
    hypohydration, 193–4
    jumping ability and muscle strength, 194–5
delayed onset hypoglycemia (DOH), 492
delayed onset of muscle soreness (DOMS), 486
densitometry, 92–3
diabetes, 490–91
  type 1 (T1DM), 492
    aerobic exercise, 492
    anaerobic exercise, 492–3
  type 1 (T2DM), 499–500
diabetic athletes, 490
  management of T1DM
    challenges in different sports, 498
    during exercise, 495–6
    exercise, 494–5
    after exercise, 496–7
    nutrition and insulin regimens, 493–4
    performance issues, 497–8
    supplements, 497
  management of T2DM
    nutrition and exercise, 500–501
diacylglycerol acyltransferase (DGAT), 171

dietary periodization, 167
dietary supplementation, 291, 298
  adverse effects, 295–6
  adverse health outcomes, 297
  aging athletes, 379
  buffering agents, 332–3
    β-alanine/carnosine, 327–30
    creatine (Cr)/phosphocreatine (PCr), 327
    summary table, 333
  buffering agents, extracellular
    sodium bicarbonate, 330–31
    sodium citrate, 331–2
  categorization, 297–8
  cost–benefit analysis, 293–5
  doping issues, 296
  effectiveness, 295
  prevalence of use, 291–2
  quality assurance programs, 297
  reasons for use, 292–3
  special needs athletes, 399–400
    vitamin D, 400
  sprinting, 565–6
  strength and power events, 557–8
  young athletes, 364, 365–6
    calcium, 364
    iron, 365
    sodium, 365
    vitamin D, 364–5
diet-induced thermogenesis (DIT), 3
diffusion coefficient, 22
direct calorimetry, 64
  definition, 62
disordered eating (DE) behaviors, 513
  definitions of DE spectrum, 513–14
distance running, 572, 582
  daily nutrition recommendations, 575
  competition phase, 577–8
  preparation phase, 575–7
  ergogenic aids
    caffeine, 582
    fluid and carbohydrate intake, 580
    gastrointestinal tolerance, 580–81
  performance determinants, 572–4
  recovery, optimizing nutrition, 578
    glycogen resynthesis, 578–9
    protein synthesis, 579
    water, 580
diuretic agents, 182–3
  nutrient–drug interactions, 378
divalent metal transporter, 1 (DMT1), 230
dopamine (DA), 207, 209, 211
doping issues, 296, 555–6
doubly-labeled water (DLW), 63, 64
drug–nutrient interactions, 378–9
drug tests, positive, 558–9
dry heat exchange, 29
dual-energy X-ray absorptiometry (DXA), 90–93
duration, definition, 62

eating disorder not otherwise specified (EDNOS), 513, 514
  diagnostic criteria, 515
eating disorders (EDs), 513, 524
  health and performance consequences, 517
  medical consequences, 517–19
  performance consequences, 519–20
  psychological/behavioral characteristics, 518
  management, 520
  primary prevention, 520–21
  recommendations for health professionals, 521–2
  secondary prevention, 521
  tertiary prevention, 522–4
  prevalence among athletes, 514–15
  females, 515–16
  males, 516
  risk factors, 516–17
effective dose (ED) of pollutants, 435, 436–7
electrolytes, 174
  beverage composition
    carbohydrates, 202
    magnesium, 202
    potassium, 201–2
    proteins, 202–3
    sodium, 200–201
  special needs athletes, 399
  sweat concentration, 179
  young athletes, 363–4
ellagitannin, 283
Embden–Meyerhoff pathway, 324–5
endothelial function, 461–2
  emerging directions, 462
endothelial progenitor cells (EPCs), 462
endurance training, metabolic adaptations, 55–6
energy availability (EA), 86
  causes of low EA, 83
  definitions, 72–5
  hormone responses to low EA, 75–6
    acute, 79–81
    chronic, 76–9
  low EA threshold, 81–3
  management
    calculation of EEE, 85–6
    general procedure, 84
    new measurement techniques, 85
energy balance (EB), definitions, 72–5
energy cost assessment, 61
  definitions, 61–2
  methodologies, 62–3
    direct methods, 64
    indirect methods, 64–70
energy expenditure (EE), 61
  daily, 72–3, 74
  definition, 62
energy flux, 455–6
energy intake, 142–4
energy nutrition, 3
  activities, energy cost of, 4
  aging athletes, 373–4
    carbohydrates, 374

energy nutrition *(continued)*
basal metabolic rate (BMR) measurement, 3–6
intake, recommended, 6
metabolic fuels, 7–9
predictive equations, 374, 375
young athletes, 359–61
carbohydrates, 361–2, 363
fats, 362–3
proteins, 362, 363
environment considerations, 425, 437
adaptation to harsh environments
altitude, 427–9
heat, 427
nutritional considerations, 428
air quality issues, 435–7
primary air pollutants, 436
common environmental illness, 429
exercise performance
altitude, 432–5
cold, 431–2
heat, 429–31
homeostasis in harsh environments
altitude, 427
heat and cold, 425–7
recommendations, 437
epigallocatechin gallate (EGCG), 278, 279, 281–2, 283, 485, 486
erythropoietin (EPO), 233
essential amino acids (EAAs), 148, 149
estimated average requirement (EAR), 17
estrogen, 348–9, 350–51
5′-*N*-ethylcarboxamidoadenosine (NECA), 316
eumenorrheic athletes (EuA), 76, 78, 81, 82
eumenorrheic sedentary women (EuS), 76, 78, 81, 82
evaporation of sweat, 29
exercise
definition, 62
fats metabolism during and after, 156
fatty acids as energy sources, 156–8
intensity of exercise, 160–63
postexercise recovery, 165–6
prolonged exercise, 158–60
metabolic adaptations
endurance training, 55–6
strength training, 56–7
metabolic response, 51
high-intensity exercise, 51–2
prolonged exercise, 52–5
water balance, impact upon, 177–9
exercise-associated hyponatremia (EAH), 539–40, 546
diagnostic features, 540
evolution to run with minimal fluid replacement, 540–41
fallacy that humans maladapted to exercise in heat, 541–4
practical considerations, 544–6
exercise-associated hyponatremic encephalopathy (EAHE), 539–40
diagnostic features, 540
exercise energy expenditure (EEE), 85–6
exercise for health enhancement, 463
exercise-induced oxidative stress, 263–4, 274
antioxidants, 265
enzymatic, 265–6
nonenzymatic, 266–9
balancing performance, redox regulation and antioxidants
antioxidant deficiencies, 272
muscle as redox sensing organ, 273–4
muscular contractions, 272–3
ROS-induced muscle fatigue, 273
biomarkers of damage, 269, 270–71
antioxidant capacity, 271
redox changes, 271–2
reactive oxygen species (ROS) production, 264
mitochondria as primary source, 264–5
exercise physiology, 20–21, 34
cardiorespiratory function, 21–5
fatigue, 30
energy depletion, 30–32
metabolic by-products, 32
neuromuscular fatigue, 32–3
neuromuscular function, 25–8
thermoregulatory function, 28–30
training principles, 33–4
exertional heat stroke, 429
extrinsic control of blood flow, 24
extrinsic sugars, 7

fast-twitch muscle cells, 27
fat-free mass (FFM), 82, 89
fatigue, 30, 324
carbohydrate ingestion during exercise, 54–5
definitions, 206–7
energy depletion, 30–32
'fatiguogens' 406
metabolic by-products, 32
metabolic response to high-intensity exercise, 52
neuromuscular fatigue, 32–3
nutritional influences on central fatigue, 212
branched chain amino acids (BCAAs), 207–8
caffeine, 211
carbohydrates, 210
summary, 211
tyrosine, 208–9
fat mass (FM), 89
fats
carbon dioxide produced from oxidation, 5
energy yield from oxygenation, 5, 167
as a fuel store, 47–8
metabolic adaptation to high-fat diets, 166
fat adaptation followed by carbohydrate restoration, 167–71

intramuscular TG storage and breakdown, 171
performance endurance, 171–2
protocols and terminology, 166–7
as metabolic fuel, 7–9
metabolism during and after exercise, 156
fatty acids as energy sources, 156–8
intensity of exercise, 160–63
postexercise recovery, 165–6
prolonged exercise, 158–60
oxidation in aerobic metabolism
fatty acid (FA) uptake by muscle, 46–7
lipolysis, 45–6
oxygen consumed by oxidation, 5
proportion of energy intake, 7–8
respiratory quotient, 5
special needs athletes, 399
strength and power events, 553–4
water of oxidation, 176
young athletes, 362–3
fatty acid-binding protein (FABPc), 158
fatty acids (FAs), 7
as energy sources, 156
mitochondrial metabolism, 162–3, 170–71
oxidation, 161–2
sources of different FAs, 160
transport from plasma into muscle, 157–8
triacylglycerols in skeletal muscle cells, 158
very low density lipoprotein triacylglycerol (VLDL-TG), 156–7
lipolysis, 46
nomenclature, 8
uptake by muscle, 46–7
fatty acid transport proteins (FATPs), 158
females, 347, 356–7
energy intake, average, 6
exercise effects upon menstruation, 353–4
cognitive dietary restraint, 354–6
energy availability, 354
polycystic ovarian syndrome (PCOS), 356
subclinical disturbances, 356
gymnastics
female athlete triad, 601
growth retardation, 602
iron deficiency, 233
menstrual cycle
hormonal fluctuations, 348
implications for nutrition, 350
normal physiology, 347–9
ovulatory disturbances, 349–50
premenstrual syndrome (PMS), 349–50
menstruation, 12
amenorrheic athletes (AmA), 76, 78, 81, 82
eumenorrheic athletes (EuA), 76, 78, 81, 82
eumenorrheic sedentary women (EuS), 76, 78, 81, 82
performance effects of menstruation, 350–53
prevalence of eating disorders (EDs), 515–16
sweat production, 178
vitamin D levels, 220–21
fermentable oligosaccharides, disaccharides, monosaccharides and polyols (FODMAPs), 533
ferritin, 232, 237
ferroportin-mediated iron, 231–2
flavin adenine dinucleotide (FAD), 222–3
flavin adenine dinucleotide, reduced ($FADH_2$), 43–5
flavin adenine mononucleotide (FMN), 222–3
flavonoids, 268, 278, 279, 485–7
FLEX HR, 65–7
flexitarian diet, 383
flow-mediated dilation (FMD), 462
fluoride, function, 11
folic acid, 13
dietary sources, 219
supplementation, 223–4
follicle-stimulating hormone (FSH), 348
food and nutrition considerations at major competitions, 439–40
factors influencing food intake, 444
age and gender, 447
cultural and religious influences, 444–6
special dietary needs, 447–8
sports performance, 446–7
taste and sensory factors, 444
food provision
consumption patterns, 442–4
food safety, 441–2
gross volumes of food, 443
menu design, 440–41
objectives, 440
venues, 442
future directions, 451
nutritional support, 448
labeling, 448–9
menu review and assessment, 448
nutrition service, 449–51
quality assurance (QA), 451
Frank–Starling mechanism, 24
free fatty acids (FFAs), 31
frequency, definition, 62
fructose-1,6-diphosphate, 49–50
fructose-6-phosphate, 49–50
fuel stores in the body, 47–8

gallic acid, 279
gastric emptying, 526–8
gastrointestinal (GI) function, 526, 535–6
distance running, 580–81
dysfunction during exercise, 531
GI symptoms, 532
mechanisms, 532–5

gastrointestinal (GI) function *(continued)*
symptom prevalence and type, 531–2
nutritional provision during exercise
effect of carbohydrate, 529–30
effect of exercise, 526–9
effect of other nutrients/beverages, 530–31
genomics, 20
ghrelin, 84
Ginkgo biloba, 285
ginseng, 285
glomerular filtration rate (GFR), 378
glucagion, 50
glucagon-like peptide, 1, 84
β-glucan immunonutrition support, 483–5
gluconeogenesis, 7
non-carbohydrate substrates, 77–8
glucose, 7
glucose uptake by muscles, 40–41
muscle uptake from blood, 40–41
normal regulation during exercise, 491
glucose-1-phosphate, 41
glucose-6-phosphate (G6P), 38–9, 40–41
glycolytic pathway to pyruvate, 41–3
glutamine immunonutrition support, 482–3
glutathione (GSH), 266–7
oxidized (GSSH), 266–7
glutathione peroxidase (GPx), 265–6
glycemic index (GI), 374, 461
low-GI diets, 509
glycerol in gluconeogenesis, 7
glycerol-3-phosphate acyltransferase (GPAT), 171
glycogen, 31
alcohol, effect on metabolism, 338–9
breakdown in muscle, 41
daily replenishment of muscles stores, 117–18
formation, 113–15
as a fuel store, 47
inflammation, 471
liver, 76
promoting storage through carbohydrate intake, 109–10
recommendations for storage strategies, 123
regulation by nutrient intervention, 113
sedentary stores, 54
short-term storage, 118
amount and frequency of carbohydrate supplementation, 119
caffeine addition, 122–3
protein addition, 120–22
timing of carbohydrate supplementation, 118–19
type and form of carbohydrate supplementation, 119–20
structure, 113–14
supercompensation, 116–17
glycogenin, 113–14
glycogen phosphorylase (PHOS), 166
glycogen synthase regulation, 115
after exercise, 115–16
glycolysis, 40, 41–3
goiter, 12
gonadotropin-releasing hormone (GnRH), 81, 348
granulocyte colony-stimulating factor (G-CSF), 479
grapefruit, 378
green tea extract (GTE), 281–2
growth hormone (GH), 51
fatty acid regulation, 78–9
growth hormone promoters, 379
gut-associated lymphoid tissue (GALT), 484
gymnastics, 596
background, 596–7
energy intake, 597
comparison between different age populations, 599
comparison between different national populations, 598
substrate distribution, 597–9
nutrient intake, 599
calcium, 600
iron, 600–601
niacin, 600
vitamin A, 599
vitamin B_1, 600
vitamin B_2, 600
vitamin C, 599–600
vitamin D, 599
nutritionally related problems
attaining ideal weight and body composition, 602
female athlete triad, 601
growth retardation, 602
gymnastics injuries, 601–2
recommendations, 604
eating during competition/practice, 604–5
post-competition/post-practice eating, 605
pre-composition/pretraining eating (PCPTE), 604

hangovers, 341
haptoglobin (Hp), 233, 237
Harris–Benedict equation, 396
health enhancement, exercise for, 463
heart, 21
cardiac muscle, 25
hypertrophy of the left ventricle, 24
heart rate (HR), 62
measurement for indirect calorimetry, 65–7
heat balance equation, 426
heat exhaustion, 29, 429
heat stress, 28
heat stroke, 29
hematocrit, 25
hemoglobin (Hb), 22, 23, 233
blood concentration, 23
free, 237–8
Henry's law, 22
hepcidin, 231–2
herpes simplex virus (HSV), 484

high-fat, low-carbohydrate (HFLC) diets, 166, 167, 508–9
fat adaptation followed by carbohydrate restoration, 167–9
membrane transport, 169–70
mitochondrial FA transport, 170–71
high-fiber diets, 509
high responders, 33
histidine, 9, 10
'hitting the wall' in marathons, 31
homeostasis in harsh environments, 20
altitude, 427
heat and cold, 425–7
hormesis, 457–8
hormones
energy metabolism regulation, 50–51
response to low energy availability (EA), 75–6
acute, 79–81
chronic, 76–9
hormone-sensitive lipase (HSL), 171
human growth hormone, 555
hunger, 83–4
hydrometry, 93
hyperinsulinemia, 460
hyperthermia, 28
hypertrophy of the left ventricle, 24
hypohaptoglobinemia, 233
hypohydration, 193–4
hyponatremia, 176
hypothalamic–pituitary–adrenal (HPA) axis, 469
hypothalamus, 29
hypothermia, 30, 429

I-band of muscle fibers, 26
immune function, 478
exercise-induced dysfunction, 478–9
future research, 487
nutritional support, 479–80
antioxidants, 482
carbohydrates, 481–2
flavonoids, 485–7
β-glucan, 483–5
glutamine, 482–3
other supplements, 485
summary, 480
indirect calorimetry, 65
definition, 62
individuality, principle of, 33
inflammation, 466, 475
acute inflammatory response, 466–7
chronic low-grade inflammation, 467–8
adipose tissue, 468
chronic low-grade inflammation, reduction by regular exercise, 468
anti-inflammatory environment, 469–70
TLR4 reduction through exercise, 469
visceral fat mass reduction, 468–9
glycogen and carbohydrate ingestion, 471
interleukin-6 (IL-6), 470
role in mediating anti-inflammatory environment, 472
role in metabolism, 473
sources, 470–71
other myokines, 474–5
signaling requirements for IL-6, 472
training status, 471–2
insulation, 29
insulin, 46, 50
fatty acid regulation, 78–9
physical activity, effect of, 460
secretion and action, 460
insulin resistance, 373, 460
interleukin-6 (IL-6), 473–4
intensity, definition, 62
interleukin-6 (IL-6), 50, 51
hypothesis to UUPS, 405–6
inflammation, 470
role in mediating anti-inflammatory environment, 472
role in metabolism, 473
signaling requirements, 472
sources, 470–71
insulin resistance, 473–4
interleukin-8 (IL-8), 474–5
interleukin-15 (IL-15), 475
interleukins, 466–7
International Society for the Advancement of Kinanthropometry (ISAK), 95
intracellular factors, 48–50
intramuscular triacylglycerols (IMTGs), 158, 160
storage and breakdown, 171
intrinsic control of blood flow, 24
intrinsic sugars, 7
involuntary muscle, 25
iodine
deficiency, 12
function, 11, 12
iron, 239–40
aging athletes, 377
chelation by tannins, 12–13
dietary sources, 224
function, 11
overload, 12
physiology, 229–30
hepcidin, 231–2
intestinal absorption, recycling and transport, 230–31
regulation of body stores, 232
recommended dietary intake (RDI), 238
sports use, 232–3
response to exercise, 233–4
role of infammation in deficiency, 234–5
sports anemia, 233
supplementation, 225
athletes, 239
bioavailability, 238–9
menstruation, 350
vegetarian diets, 387, 389
young athletes, 365
iron deficiency, 12, 229–30, 232–3
see also anemia
diagnosis

iron deficiency *(continued)*
biological variability and critical difference (CD), 235
diagnostic indices, 235–8
ferritin, 237
free hemoglobin, 237–8
haptoglobins, 237
serum iron, 236
total iron binding capacity (TIBC), 236
transferrin, 236
transferrin saturation, 236–7
transferrin soluble receptor, 237
role of inflammation, 234–5
sports anemia, 233
iron-refractory iron-deficiency anemia (IRIDA), 231
iron-regulation protein (IRP), 232
islets of Langerhans, 50
isoleucine, 9, 10

ketogenesis, 78
ketone bodies, 31
kidneys, 176
kilocalorie (kcal), definition, 62
kilojoule (kJ), definition, 62
Krebs cycle, 43–5

labeled bicarbonate, 64
lactate shuttle, 30
lactic acid role in fatigue, 30, 32
lacto-ovo vegetarian diet, 383
lactose, 7
lacto vegetarian diet, 383
leucine, 9, 10, 148, 150–51
leukemia inhibitory factor (LIF), 474
lignin, 18
linoleic acid (C18:3 ω6), 7
α-linolenic acid (C18:3 ω3), 7
lipase, 46
lipid hydroperoxides (LOOH), 271
α-lipoic acid, 268
lipolysis, 45–6
lipopolysaccharides (LPS), 472
lipoprotein lipase (LPL), 156–7
lithium
function, 11
pharmacological effects, 12
liver glycogen storage, 76
long-chain acyl carnitine (LCAC), 160
Loughborough Intermittent Shuttle Test (LIST), 128, 629
low-density lipoproteins (LDLs), 8
reduction by β-glucans, 483–4
lower reference nutrient intake (LNRI), 17
low-fat, high-carbohydrate diets, 507–8
low-GI diets, 509
low responders, 33
lumisterol, 256
luteinizing hormone (LH), 81, 348
pulsatility, 354
lysine, 9, 10

macrobiotic diet, 383
macrophage inflammaotry protein, 1 beta (MIP-1β), 479
macrophage migration inhibitory factor, 1 (MIF-1), 479
macrophages, 467
magnesium
dietary sources, 247–8
function, 11
water replacement, 202
major histocompatibility complex (MHC), 478
males
energy intake, average, 6
prevalence of eating disorders (EDs), 516
malondialdehydes (MDAs), 271
malonyl-CoA, 162
manganese function, 11
marathons
dehydration, effects of, 191–2
'hitting the wall' 31
metabolic response to, 53–4
maximal voluntary contraction (MVC), 329–30
mechanoreceptors, 25
men, *see* males
menaquinones, 13
metabolic adaptation, 147, 154
acute versus long-term exercise response, 152–3
high-fat diets, 166
fat adaptation followed by carbohydrate restoration, 167–71
intramuscular TG storage and breakdown, 171
performance endurance, 171–2
protocols and terminology, 166–7
postexercise muscle protein reconditioning, 147–9
protein ingestion following exercise, 149
amount, 149–50
carbohydrate co-ingestion, 151
sources, 150–51
timing, 152
metabolic equivalent (MET)
definition, 62
typical values, 63
metabolic fuels, 7–9
metabolic rate, definition, 62
methionine, 9, 10
methylxanthines, 315
microcytic anemia, 412–13
micronutrients
aging athletes, 375–8
minerals, 11–13
other compounds, 18
special needs athletes, 399–400
vitamin D, 400
vitamins, 13–14
milk proteins, 150–51
minerals, 11–13, 217
dietary reference values, 14–16
dietary sources, 224
supplementation for athletes, 217–18, 226
performance effects, 218–19
mitochondria, structure and function, 456–7
mitogen activated protein kinase (MAPK), 472
mixed amino acids (MAA), 149
molybdenum function, 11

monocyte chemotactic protein, 1 (MCP, 1), 479
monounsaturated fats, 7
  lipoprotein levels, 8
motor units, 27
mountain sickness, 429, 534
movement monitors, 67
muscle fibers, 25
muscle protein breakdown (MPB), 138
muscle protein synthesis (MPS), 138, 139
muscle pump, 22
muscles, 25
  cardiac muscle, 25
  contraction, 36–7
  daily replenishment of glycogen stores, 117–18
  energy for muscle force generation
    amino acid oxidation in aerobic metabolism, 47
    ATP hydrolysis, 38
    ATP resynthesis, 38–9, 302–5
    buffering excessive pH falls, 43
    carbohydrate oxidation in aerobic metabolism, 43–5
    fat oxidation in aerobic metabolism, 45–7
    glucose uptake from blood, 40–41
    glycogen breakdown, 41
    glycolysis, 40
    glycolytic pathway, 41–3
    phosphocreatine breakdown, 39–40
  hypertrophy, 27
  postexercise protein reconditioning, 147–9
  skeletal muscle, 26
    biochemistry, 36–8
  smooth muscle, 25
  type I cells, 27, 37–8
  type II cells, 27, 37–8
myofibrils, 26, 27
  muscle contraction, 36–7
  types, 37–8
myoglobin, 37–8
myokines, 470
myosin, 27

natural killer cells, 479
net protein balance, 138, 139
net protein utilization (NPU), 11
neuromuscular function, 25–8
nickel function, 11
nicotinamide, 13
nicotinamide adenine dinucleotide (NAD^+), 40
  reduction to NADH, 41–3
nicotinic acid, 13
nitrates, 45
nitrogen dioxide (NO_2), 435, 436
nitrogen (N) balance, 9
non-rapid eye movement sleep (NREM), 411
nonsteroidal anti-inflammatory drugs (NSAIDs), 282
  nutrient–drug interactions, 378
noradrenaline (NA), 50–51, 207, 209
nuclear factor of activated T-cells (NFAT), 472
nutrient–drug interactions, 378–9
nutritarian diet, 383
nutrition, general, 3
  energy, 3
    activities, energy cost of, 4
    basal metabolic rate (BMR) measurement, 3–6
    intake, recommended, 6
    metabolic fuels, 7–9
  micronutrients
    minerals, 11–13
    other compounds, 18
    vitamins, 13–14
  protein requirements, 9
    quality and amino acid requirements, 9–11

occupations, PAR classification, 5
oleic acid (C18:1 ω9), 7
oligomenorrhea, 349
orexigenic hormones, 84
osteoporosis, 220–21
  vegetarian diets, 388
ovarian function, 81
overreaching, 34, 404, 413
  recovery through nutrition, 406–8
overtraining, 34
overweight athletes, 503
  challenges manipulating body weight and composition
    environment, 505
    genetics, 504–5
    growth and puberty, 505
    training, 505–6
  clinical and practical aspects of weight management, 511
  strategies for weight and fat loss
    commercial weight loss and meal replacement programs, 510
    dairy consumption, 509–10
    energy density manipulation, 509
    energy intake, 506–7
    exercise-related strategies, 510–11
    high-fat, low-carbohydrate (HFLC) diets, 508–9
    low-fat, high-carbohydrate diets, 507–8
    low-GI, high-fiber and gut microbia, 509
  weight, physique and athletic performance, 503–4
ovo vegetarian diet, 383
ovulatory disturbances, 349
oxidation efficiency of carbohydrates, 132
oxidative stress, 263–4, 274, 458–9
  alcohol, 339
  antioxidants, 265
    enzymatic, 265–6
    nonenzymatic, 266–9
  balancing performance, redox regulation and antioxidants
    antioxidant deficiencies, 272
    muscle as redox sensing organ, 273–4
    muscular contractions, 272–3
    ROS-induced muscle fatigue, 273
  biomarkers of damage, 269, 270–71

oxidative stress *(continued)*
antioxidant capacity, 271
redox changes, 271–2
exercise-induced reactive oxygen species (ROS) production, 264
mitochondria as primary source, 264–5
oxidized glutathione (GSSH), 266–7
oxygen consumption in food component oxygenation, 5
oxygen intake (VO_2), 62
oxygen transport, 21, 22
blood concentration, 23
partial pressure, 22
ozone (O_3), 436

pancreas, insulin production, 50
pancreatic polypeptide, 84
pantothenic acid, 13
parathyroid hormone (PTH), 242
particulate matter (PM), airborne, 436
pedometers, 67
peptide YY, 84
performance-enhancing substances, 554–5
banned substances, 555–6
dietary supplements, 557–8
permitted substances, 556
periodization, principle of, 33
permitted substances, 556
peroxisome proliferator-activated receptors (PPTARs), 56
phenolic acids, 279
phenyalanine, 9, 10
phosphate function, 11
phosphate, inorganic (P_i), 30–31
phosphofructokinase (PFK), 49–50, 52, 166
phosphorylase, 41
phosphorylation, 38–9
phospho(ryl)creatine (PCr), 30–31, 301, 303–5
ATP resynthesis, 38–9
breakdown, 39–40
buffering supplement, 327
metabolic response to exercise
high-intensity exercise, 51, 52
prolonged exercise, 52–3
vegetarian diets, 384–5
phylloquinone (vitamin K), 13
physical activity, 455
acute exercise and nutrition, 461
definition, 62
dietary energy deficit, 460–61
emerging directions in disease prevention, 459–60
endothelial function, 461–2
energy flux, 455–6
hormesis, 457–8
insulin action, effect upon, 460
mitochondrial structure and function, 456–7
nutrition and oxidative stress, 458–9
redox balance, 458
single nutrients, 461
physical activity level (PAL), 3, 69
aging athletes, 374
physical activity ratio (PAR), 3
energy cost of activities, 4
ratio (PAR)
work classification, 5
Physical Working Capacity at Fatigue Threshold (PWC(FT)), 293
phytochemicals, 277, 288
central nervous sysem (CNS) and performance, 283–5
definition, 277–9
exercise performance, 279
green tea extract (GTE), 281–2
quercetin, 279–81
resveratrol, 281
future directions, 285–8
inflammation and performance, 282
anthocyanin, 283
curcumin, 282
ellagitannin, 283
quercetin, 282–3
plasmalemma, 26
plasma osmolality, 176, 177
p3 mitogen-activated protein kinase (p38 MAPK), 55–6
Poisson's law, 92
polycystic ovarian syndrome (PCOS), 356
polyphenols, 278
polysaccharides, 18
polyunsaturated fats, 7
lipoprotein levels, 8
polyunsaturated fatty acids (PUFAs), 485
pomegranate, 283
P/O ratio, 45
potassium
function, 11
water replacement, 201–2
pre-composition/pretraining eating (PCPTE), 604
premenstrual syndrome (PMS), 349–50
preoptic anterior hypothalamus, 29
probiotics, 485
progesterone, 350–51
progressive overload, principle of, 33
prohormone supplements, 555
protein digestibility-corrected amino acid score (PDCAAS), 11
protein efficiency ratio (PER), 11
protein leverage hypothesis (PLH), 508–9
proteins
aging athletes, 375
carbon dioxide produced from oxidation, 5
energy yield from oxygenation, 5
excessive intake, 141–2
as a fuel store, 48
intake, safe, 9
metabolic adaptation trigger, 147, 154
acute versus long-term exercise response, 152–3
postexercise muscle reconditioning, 147–9
protein ingestion following exercise, 149–52
as metabolic fuel, 7–9
milk proteins, 150–51
muscle protein, 27

optimum intake for athletes, 136, 138–41
current recommendations, 136–8
energy intake, 142–4
practical recommendations, 144–5
timing of protein consumption, 144
oxygen consumed by oxidation, 5
proportion of energy intake, 8–9
quality, 9–11
requirement, 9
respiratory quotient, 5
safe intake, 9
special needs athletes, 398–9
sprinting, 564
strength and power events, 553
supplementation for glycogen storage, 120–22
vegetarian diets
protein quality, 386
protein requirements, 386–7
water of oxidation, 176
water replacement, 202–3
young athletes, 362, 363
protein score, 11
pulmonary diffusion, 21
pulmonary ventilation, 21–2
pyridoxal, 13
pyridoxamine, 13
pyridoxine (vitamin B_6), 13
aging athletes, 377
dietary sources, 219
supplementation, 223
pyruvate dehydrogenase (PDH), 166
pyruvate (pyruvic acid), 31, 39
carbohydrate oxidation in aerobic metabolism, 43–5
glycolytic pathway from G6P, 41–3

quercetin, 279–81, 282–3, 485, 486
Quetelet's index, 99

radiation of heat, 29
ratings of perceived exertion (RPE), 130
reactive oxygen and nitrogen species (RONS), 56
reactive oxygen species (ROS), 229, 263–4, 274
alcohol, 339
antioxidants, 265
enzymatic, 265–6
nonenzymatic, 266–9
balancing performance, redox regulation and antioxidants
antioxidant deficiencies, 272
muscle as redox sensing organ, 273–4
muscular contractions, 272–3
ROS-induced muscle fatigue, 273
exercise-induced, 264
mitochondria as primary source, 264–5
hormesis, 458
identification, 270
mitochondria, 457
oxidative stress, 459
redox balance, 458
recommended dietary allowance (RDA), 17
recommended energy intake, 6
red blood cells (RBCs), 21, 22, 23
blood doping, 25
redox balance, 265, 266
reference nutrient uptake (RNI), 17
rehydration, *see* water replacement
relative protein value (RPV), 11
religious dietary restrictions, 446
respiratory exchange ratio (R), 64
respiratory membrane, 22
respiratory pump, 22
respiratory quotient (RQ), 5, 64
resting energy expenditure (REE), 61
resting metabolic rate (RMR), 3
resveratrol, 279, 281, 485
retinol (vitamin A), 13
dietary sources, 219
supplementation, 219
reversibility, principle of, 33
rhabdomolysis, 378
riboflavin (vitamin B_2), 13
dietary sources, 219
supplementation, 222–3
room calorimeters, 64

sarcolemma, 26
sarcomeres, 26
sarcoplasm, 27
sarcoplasmic reticulum (SR), 26, 27, 36
satellite cells, 27
saturated fats, 7
selective estrogen receptor modifiers (SERMS), 379
selenium
function, 11
intake, 12
semi-vegetarian diet, 383
serotonin (5-hydroxytryptamine; 5-HT), 207–8
serum iron, 236
shivering, 30
silicon, function, 11
skeletal muscle, 26
biochemistry, 36
contraction, 36–7
fiber types, 37–8
postexercise protein reconditioning, 147–9
protein turnover rate, 147
sleeping metabolic rate (SMR), 81
sliding-filament theory of muscle contraction, 27
slow-twitch muscle cells, 27
smooth muscle, 25
sodium, 176
function, 11
water replacement, 200–201
young athletes, 363–4
sodium bicarbonate, 23, 330–31
strength and power events, 558
sodium citrate, 23, 331–2
special needs athletes, 392–3
energy and macronutrients
body composition assessment, 397–8
carbohydrates, 398

special needs athletes *(continued)*
energy expenditure, 395–7
fats, 399
proteins, 398–9
water and electrolytes, 399
health and clinical sports nutrition, 401–2
micronutrients and supplements
vitamins and trace minerals, 399–400
practical issues
food and nutrition at major competitions, 401
traveling, 400–401
underpinning science
amputees, 395
cerebral palsy (CP), 395
spinal cord injuries (SCI), 393–5
specificity, principle of, 33, 34
spinal cord injuries (SCI), 393–5
energy expenditure, 396
sport, definition, 62
sports anemia, 233
sprinting, 561–2, 570
competition nutrition strategies, 566–8
dietary practices and recommendations, 562
carbohydrates, 562–4
proteins, 564
water, 564–5
physique traits, 568–70
recovery, 565
supplementation, 565–6
starvation, 54
statins, nutrient–drug interactions, 378
stearic acid (C18:0), 7
cholesterol, effect on, 8
stilbenes, 278, 279
strength and power events, 551, 559
energy intake, 551–2
macronutrient intake
carbohydrates, 552
fats, 553–4
proteins, 553
water, 554
micronutrients, 554
performance-enhancing substances, 554–5
banned substances, 555–6
dietary supplements, 557–8
permitted substances, 556
positive drug tests, 558–9
strength training, metabolic adaptations, 56–7
subcutaneous adipose tissue (SAT), 90, 93
sugars, 7
sulfur dioxide (SO_2), 435–6
superoxide dismutase (SOD), 265–6
supplementation, *see* dietary supplementation
sweat, 174, 177–8
cooling effect, 29
electrolyte concentrations, 179
evaporation, 29
rate, 29
volume produced, 29
swimming, 607, 616–17
achieving ideal body composition, 609–10
carbohydrate requirements, 610–12
competition nutrition, 615
multiday meets, 615–16
open water swimming, 616
energy requirements, 607–9
micronutrient requirements, 613–15
protein requirements, 612–13
supplements and sports foods, 616
sympathetic nervous system, 469
syndrome of inappropriate ADH secretion (SIADH), 543
systems biology, 20

tachysterol, 256
tamoxifen, 379
team sports, 629–30
characteristics, 630
cultural issues, 636–7
dietary planning, 633–4
hydration, 634–5
modifying size and body composition, 632–3
nutrient intake of team athletes, 631
nutrient timing, 633
specifics, 630
supplementation, 635–6
terminal cisternae, 26
testosterone precursors, 555
therapeutic use exemption (TUE), 379
thermic effect of food (TEF), 61
thermogenesis, 30
thermoreceptors, 29
thermoregulatory function, 28–30
thiamin (vitamin B_1), 13
dietary sources, 219
supplementation, 222
thiobarbituric acid reactive substances (TBARS), 271
thiokinase, 46
thirst, 177, 189–90
threonine, 9, 10
thyroxine, 12
tin, function, 11
tiredness, 30
titin, 27
tocilizumab, 474
tocopherol (vitamin E), 13
as an antioxidant, 267–8
dietary sources, 219
supplementation, 221–2
tocotrienols, 13
toll-like receptor (TLR), 466
TLR4 reduction through exercise, 469
tonic vasoconstriction, 24
total energy expenditure (TEE), 61, 73
total iron binding capacity (TIBC), 236
training
carbohydrate needs, 102
daily requirements, 102–9
future research directions, 110–11
IOC guidelines, 106
promoting glycogen storage, 109–10
goals, 72
maximal rates of ATP resynthesis, 40

overtraining, 34
principle, 33–4
trans-fats, 7
recommended intake, 8
transferrin saturation, 236–7
transferrin soluble receptor, 237
transferrin (Tf), 236
transverse tubules, 26
traveling athletes, 415
backup plan, 420
food safety, 420–22
nutrition guidelines
pretravel, 415–18
upon return, 420
traveling, 418–20
special issues, 422–4
triacylglycerol droplets, 36
triacylglycerols (TGs), 47–8, 156
adipose tissue triacylglycerol (ATGL), 158
intramuscular triacylglycerols (IMTGs), 158, 160
storage and breakdown, 171
very low density lipoprotein triacylglycerol (VLDL-TG), 156–7, 160
tricarboxylic acid (TCA), 38–9, 43–5
tri-iodothyronine, 12
tropomyosin, 27, 37
troponin, 27, 37
tryptophan (TRP), 9, 10, 207–8
tumor necrosis factor-α (TNF-α), 466–7, 468, 479
type I muscle cells, 27, 37–8
type II muscle cells, 27, 37–8
tyrosine, 208–9

ubiquinone, 268
ultrasound, 93–4
underwater weighing (UWW), 92–3
unexplained underperformance syndrome (UUPS), 404, 413
cytokine theory, 404–6
interleukin-6 (IL-6) hypothesis, 405–6
nutritional strategies
anorexia, 412
daytime fatigue and poor sleep quality, 410–11
frequent infectious episodes, 408–10
microcytic anemia, 412–13
slow wound healing, 411–12
signs and symptoms, 406
triggers, 406
upper respiratory tract infections (URTIs), 478, 479
uracil, 329
urea, 5, 31
uric acid, 267
urinary tract infections (UTIs), 400, 401
urine, 179–80

valine, 9, 10
vanadium, function, 11
variation, principle of, 33
vasoconstriction, 24, 30
vasodilation, 24, 30
vegan diet, 383
vegetarian athletes, 382, 389–90
advantages, 382
amenorrhea, 388–9
antioxidants, 384
classification of diets, 382–3
creatine (Cr), 384–5
nutritional adequacy
calcium, 388, 389
iron, 387, 389
protein quality, 386
protein requirements, 386–7
vitamin B_{12}, 387, 389
zinc, 387–8, 389
nutritional strategies for micronutrients, 389
performance, 383–4
very low density lipoprotein triacylglycerol (VLDL-TG), 156–7, 160
vitamin D receptor (VDR), 253
vitamins, 13–14, 217
A (retinol), 13
dietary sources, 219
supplementation, 219
B_1 (thiamin), 13
dietary sources, 219
supplementation, 222
B_2 (riboflavin), 13
dietary sources, 219
supplementation, 222–3
B_6 (pyridoxine), 13
aging athletes, 377
dietary sources, 219
supplementation, 223
B_{12} (cobalamin), 13
aging athletes, 377
vegetarian diets, 387, 389
C (ascorbic acid), 13
as an antioxidant, 267
dietary sources, 219
supplementation, 224
D (calciferol), 13, 242, 259
aging athletes, 376–8
clinical assessment, evaluation and treatment, 256–9
dietary sources, 219, 247–8, 249
functions, 252–5
intake, 250–52
intoxication, 256
requirements for athletes, 256
special needs athletes, 400
status in athletes, 251–2
status indices, 249–50
supplementation, 220–21
synthesis, 249
young athletes, 364–5
deficiency, 14
dietary reference values, 14–17
dietary sources, 219
E (tocopherol), 13
as an antioxidant, 267–8
dietary sources, 219
supplementation, 221–2
fat-soluble, 7, 14
folic acid, 13
dietary sources, 219
supplementation, 223–4
H (biotin), 13
K (phylloquinone), 13
niacin (nicotinic acid), 13
pantothenic acid, 13
supplementation for athletes, 217–18, 226
performance effects, 218–19
water-soluble, 14

volume of expired air (VE), 62
VO_2 max, 23, 24

water
see also dehydration
aging athletes, 378
basal requirements, 174–6
control of water balance, 176–7
impact of exercise, 177–9
hydration status assessment, 179–80
intake guidelines, 186–7, 195
before exercise, 195–6
during exercise, 196
isotopically labeled for respirometry, 6
special needs athletes, 399
sprinting, 564–5
strength and power events, 554
young athletes, 363–4
water replacement, 174, 180–81, 183, 199
see also dehydration
diuretic agents, 182–3
factors, 199–200
beverage composition, 200–203
beverage palatability and voluntary intake, 203
food and fluid consumption, 203
volume and rate of drink ingestion, 200
post-exercise rehydration, 203–4
practical considerations, 204
water turnover measurement, 181–2
weight-category sports, 639, 647–8
health concerns, 646
nutritional concerns, 643–4
performance concerns, 644–6
practical considerations
before weigh-in, 646
recovery strategies, 646–7
recommendations, 648–9
weigh-in procedure, 642
weight-loss strategies and techniques, 639–41
boxers and taekwondo athletes, 641–2
lightweight rowers, 641
wrestlers and judokas, 641
Weir equation, 65
wet bulb globe temperature (WBGT), 426
wind chill index (WCI), 426
winter sports, 619
alpine skiing, 622
nutrition issues, 622–3
common nutrition issues, 626–7
freestyle skiing and snowboarding, 623
nutrition issues, 623–4
nordic skiing, 619–21
nutrition issues, 621–22
skating sports, 624–5
nutrition issues, 625–6
sliding sports, 626
nutrition issues, 626
women, *see* females
work, PAR classification, 5
World Anti-Doping Agency (WADA), 555–6
positive drug tests, 558–9

young athletes, 359
dietary supplementation, 365–6
energy needs, 359–61
carbohydrates, 361–2, 363
fats, 362–3
proteins, 362, 363
micronutrients, 364
calcium, 364
iron, 365
sodium, 365
vitamin D, 364–5
practical considerations, 366
water and sodium, 363–4

zinc
dietary sources, 224
function, 11
supplementation, 225–6
vegetarian diets, 387–8, 389
Z-line of muscle fibers, 26