EXERCISE PHYSIOLOGY

The Methods and Mechanisms Underlying Performance

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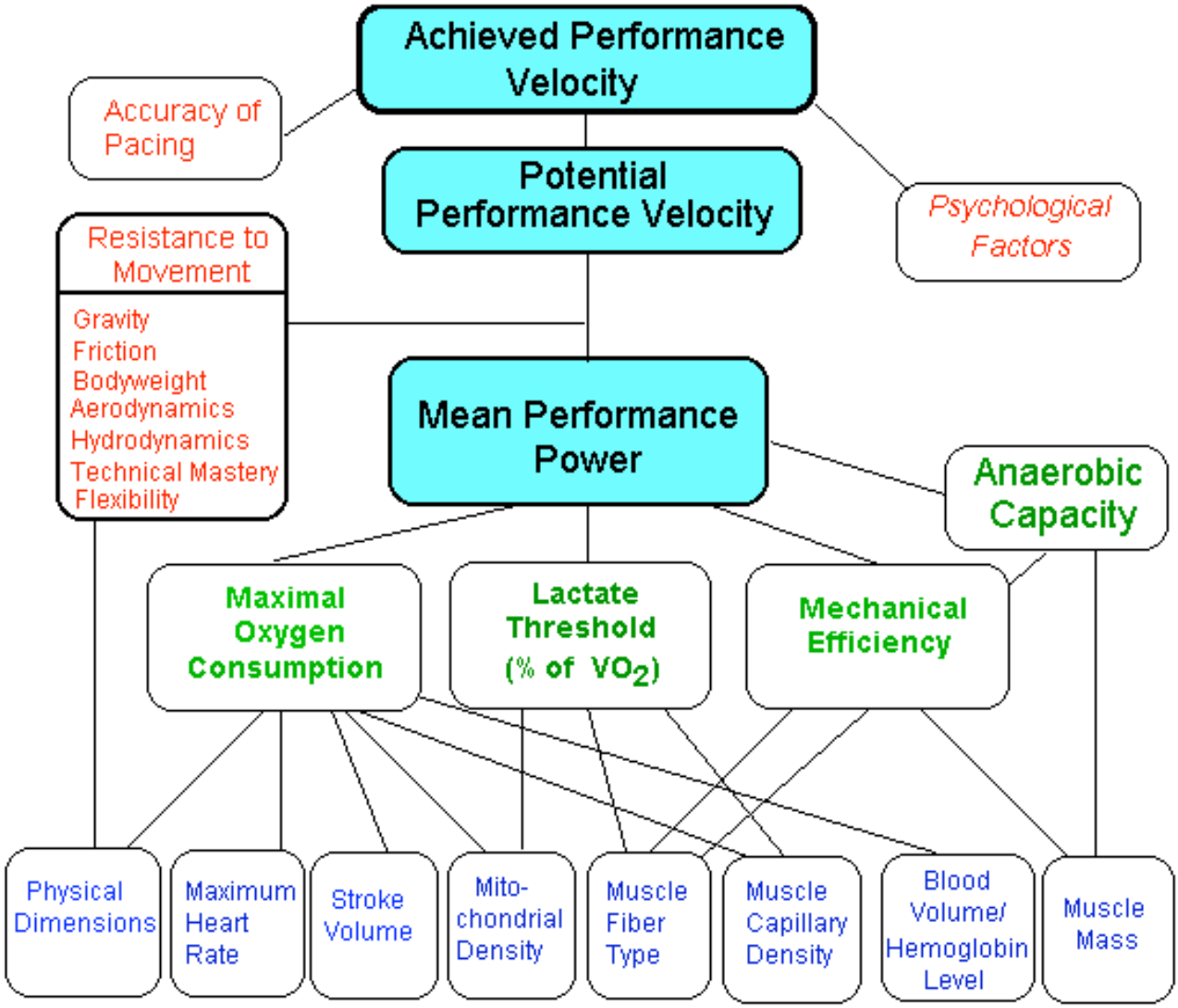
THE ENDURANCE PERFORMANCE MODEL

Whether you run, row, ski or cycle, the goal is always the same; you are attempting to maximize your ACHIEVED PERFORMANCE VELOCITY. All endurance sports demand some combination of three components: 1) High oxygen transport capacity, 2) High fatigue resistance in working muscles, and 3) High efficiency of transfer of physiological work to mechanical movement. Every endurance athlete brings to the starting line some combination of Performance Power (1 and 2). The third variable, Efficiency of Power Transfer (3) links the engine to the specific movement task. These variables combine to determine Potential Performance Velocity. Finally, on a given race day, performance potential is influenced by psychological factors and the accuracy of pacing. The end product is ACHIEVED PERFORMANCE VELOCITY, a personal best, a Masters record etc.

Conservatively, we can list dozens of factors that impact endurance performance. To make things more complex (and interesting), these factors are not independent, but influence each other. Finally, each particular sport discipline puts specific demands and constraints on the system through both the specific resistance to movement that must be overcome, and the race distance or duration.

Given all of this complexity, it is helpful to have a unifying model. So, I am presenting one here for you that you can refer back to when you read other articles. This model summarizes the currently accepted understanding of the physiological limitations to endurance performance. In other words, it is the current paradigm. Perhaps time will show that other factors should be included, or some of these deemphasized. For now, this model seems to fit the data well.

The concept of the figure or this discussion is certainly not original. It summarizes the findings of nearly 100 years of physiological and performance research. An excellent article on this subject based on research he has directed or assisted, and a fairly similar figure, were produced by Edward Coyle PhD (Exercise and Sport Science Reviews, vol.23, p25, 1995. Williams and Wilkens, Publishers.) Michael Joyner M.D. also wrote some excellent synthesizing material on the issue of physiological limitations on performance (running). Journal of Applied Physiology 70:683-687, 1991.



Now, as complicated as the figure above may appear, it is still a simplification. Underneath the physical and anatomical components we could add: 1) genotype, 2) genetically determined responsiveness to training, 3) nutrition, 4) immunological resistance to stress, 5) testosterone level, 6) intensity of training stimulus, 7) frequency of training, 8) years of training load, etc. It is a fantastic puzzle to explore, but remember, the solution is different for each person. Good Luck.

1) HEART FACTS AND TRIVIA

What's in a Name?

The existence of the heart was well known to the Greeks, who gave it the name Kardia, still surviving in modern words such as cardiac and tachycardia. Aristotle believed that the heart was the seat of the soul and the center of man. Romans modified Kardia to Cor, the latter word still surviving in "cordial greetings". The old Teutonic word herton was also derived from Cor and gives us heart via the medieval heorte.

Where is it Located?

Dumb question right? Well if you answered left chest, you're wrong! The heart is situated almost dead center in the middle of the chest nested between the two lungs. However, the apex or tip of the heart is shifted towards the left chest wall and hits against the ribs during contraction. Consequently, the rhythm is best detected on the left side, just below the pectoralis.

How Big is it?

It is generally about the size of your fist. This is not really very big when you think about the job it does. In some animals, such as horses, the heart size to body size ratio is much greater. This helps explain why horses are such great endurance athletes! The heart is also bigger in champion endurance athletes, due to genetics and training. (***see subcategory-(b) below***). The average untrained heart can pump about 15 to 20 liters of blood per minute at max. Large, elite athletes may have a maximal cardiac output of nearly 40 liters / min. This is a huge flow moving through a pump the size of your fist! To get some perspective on these output rates, go to your kitchen sink and turn on the water full blast. Now find a milk jug or something that will give you a measure of volume. I bet you find that your faucet does not flow as fast as the heart can pump.

In a sense, the heart is really two linked pumps, the left heart and the right. Both sides pump the same amount of blood, but to different locations at different pressures. The right side pump (right ventricle) pumps oxygen-depleted blood that has returned from the body to the lungs for reoxygenation. This is a short trip and requires little pressure development, so the right ventricle is rather thin walled, like a fireplace bellows. The left side (left ventricle) is the real workhorse, pumping oxygenated blood that has returned

from the lungs (the right and left side of the heart are thus connected) to the entire body. That means moving blood through an incredible maze of blood vessels from the top of the head to the toes! Consequently it must develop more pressure each beat (about 120mm Hg at rest). The left heart muscle is thicker as a result, just as your bicep would become thicker if you had to lift heavy weights with it all day.

How Does it Pump Blood?

Classically, we have been taught that the heart squeezes blood through the aorta by decreasing the external circumference of the heart. This view is supported by the fact that during heart surgery (with the chest cracked open), the heart does pump in this manner. However, under normal conditions, the heart operates within the thoracic cavity in a closed, fluid-filled volume. There is now growing evidence to indicate that during exercise, the heart performs more like a piston or a vacuum pump, with little change in external circumference. As we learn more about the dynamics of heart function, it is evident that this model is critical to the efficiency of the heart as a pump. More recent models of heart performance indicate that the heart takes advantage of vacuum effects and fluid inertia as heart rate increases during exercise. One reason why artificial hearts have performed so poorly is that they have tried to use a design based on erroneous assumptions about how the human heart pumps. The classical view of heart pumping mechanics will die slowly, due to its pervasiveness. However, it seems reasonable to say that the heart performs more like a vacuum pump than like a hand squeezing the juice out of a lemon. When the heart pumps, the ventricular wall's outer diameter changes little, while the internal diameter dramatically decreases as blood is ejected from the ventricle.

What Controls the Heart Rate?

Now this is a tough question to answer without using a little physiology lingo. Unlike skeletal muscle, which is under voluntary control, the heart is an involuntary muscle. Most of us cannot just tell our heart to slow down or speed up (biofeedback training not withstanding). The beating frequency (heart rate) is controlled by the balance of stimulation coming from the sympathetic and parasympathetic branches of the Autonomic Nervous System. Both nervous inputs to the heart converge on a small area of tissue in the right atrium called the Sino-atrial (SA) node. Parasympathetic (rest and recover) stimulation tends to slow down the rate, while sympathetic (fight or flight) input increases the rate (and the force of contraction). Normally, there is a balance between the two

inputs leaning toward the parasympathetic side. However, even without any nervous input, the heart will beat automatically due to some unique features of its membrane physiology. This intrinsic rate is quite slow however (about 20 bpm). A purely parasympathetic stimulation will result in a heart rate of about 30. So the average untrained person has a resting heart rate of about 70 as a result of some constant sympathetic stimulation. With training, the balance between parasympathetic and sympathetic stimulation tends to shift in favour of the parasympathetic, resulting in a slower resting heart rate. Elite endurance athletes may have resting HRs of 35 to 40. Values of 28 have been reported!

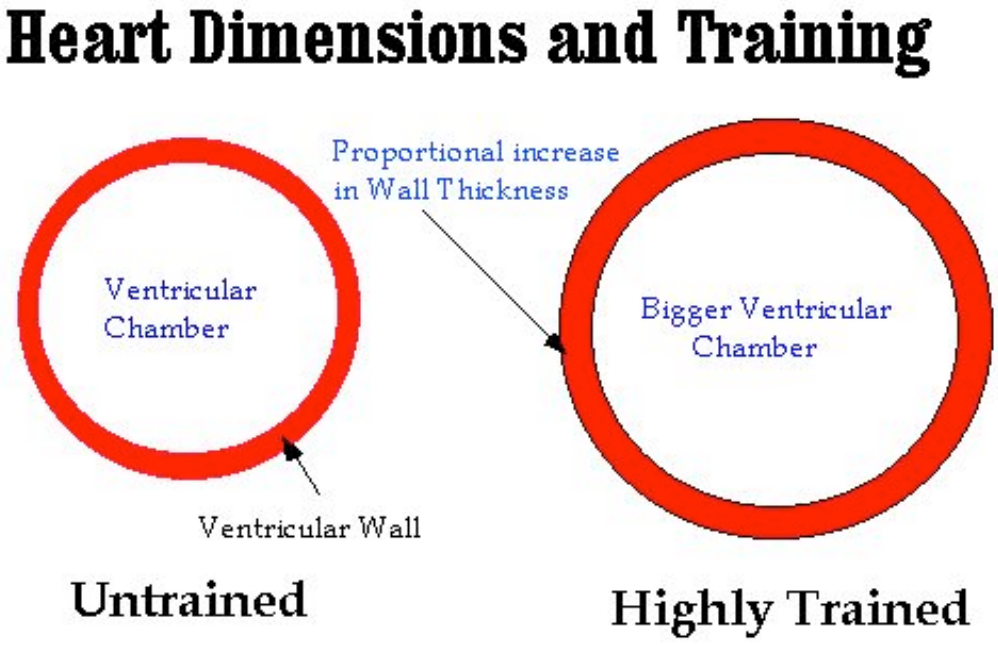
The initiation of activity results first in a withdrawal of the parasympathetic stimulation (up to a heart rate of about 100) followed by an increase in sympathetic stimulation with more intense activity up to the maximum heart rate ***(see subcategory-(d) below).*** A number of studies have demonstrated that maximal heart rate actually tends to DECREASE with high volumes of endurance training. The average of a number of studies is about a 7 beat reduction in maximal heart rate after training compared to the untrained state. Anecdotally, it also appears that even in athletes, periods of very high volume can transiently cause a reduction in the maximal heart rate, or perhaps more correctly a reduction in the capacity of the sympathetic nervous system to maximally mobilize the heart rate. We have tested junior XC skiers before and after a 10 day training camp filled with a lot of training volume. On average, the team showed a slight reduction in VO2 max despite being very fit, and their maximal heart rate during a VO2 max test was perhaps 4 beats per minute lower. The athletes were very fit, but could not fully mobilize; they lacked that last gear. After a few days of relative rest, they were back to normal.

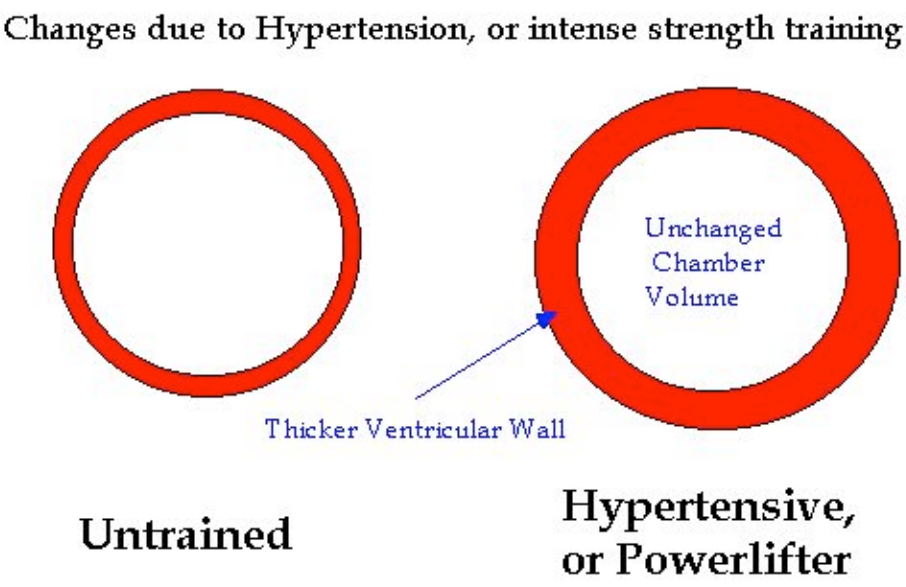
Will Training Make My Maximal Heart Rate Increase?

The answer to this question has just been answered. No, the maximum heart rate is not increased by training! As we get older, our maximum heart rate decreases. The major difference in the endurance trained heart is a bigger stroke volume. The trained heart gets bigger and pumps more blood each beat. So, that small reduction in maximal heart rate is more than compensated for by an increase in stroke volume.

i) More About How the Heart Adapts to Training

More accurately, the End Diastolic Volume (EDV) increases in the trained heart. EDV is the volume of blood in the left ventricle just prior to the initiation of contraction. End Systolic Volume (ESV) is the residual volume remaining immediately after contraction. Ejection fraction is the ratio (EDV-ESV)/EDV. The Frank-Starling Law tells us that if more blood enters the heart, more will be ejected. I will spare you the reasons why that is true. After training, the heart operates on a more efficient portion of the length-tension curve. The Increased End Diastolic Volume and resulting increased stroke volume is accomplished three ways. **First, the decreased heart rate increases ventricular filling time. Second, the ventricle increases in size** through what is termed *eccentric hypertrophy*. The volume of the ventricular chambers (lumen) increases due to longitudinal sarcomere addition. This adaptation is in contrast to the ventricular wall thickening or concentric hypertrophy without increased lumen volume that is observed in hypertensive patients, or in people who train intensely with weights. There is a small increase in ventricular wall thickness in the endurance trained heart. This balances the increased wall tension associated with operating at an increased diameter (**Law of Laplace**). Graphic representation below.





Finally, the EDV increases after training due to an **increase in blood volume**. More blood volume results in greater venous return of blood to the heart at any given peipheral capacitance. Blood volume/kg bodyweight is about 15% higher than untrained. This adaptation is quite rapid, and helps explain why VO2 max is significantly increased after only 1 week of training in previously sedentary subjects. This blood volume expansion is also rapidly lost (3-7 days) with inactivity. The increased blood volume is due to both an increase in blood plasma and an increase in red blood cells. However, the plasma volume change is slightly greater so that blood hematocrit is slightly reduced with training (exercise pseudoanemia).

ii) Maximal Oxygen Consumption - The VO2 max

If you walk into the locker room of a bunch of American Football players, bragging rights are reserved for the man with the heaviest bench press. Similarly, talk to a group of endurance athletes that are "in the know", and conversation will eventually turn to "What is your VO2 max?" A high maximal oxygen consumption is indeed one of the hallmark characteristics of great endurance performers in running, cycling, rowing and cross-country skiing, so it must be pretty important. What is it and how is it measured?

VO2 max defined:

VO2 max is the maximum volume of oxygen that by the body can consume during intense, whole-body exercise, while breathing air at sea

level. This volume is expressed as a rate, either liters per minute (L/min) or millilitres per kg bodyweight per minute (ml/kg/min). Because oxygen consumption is linearly related to energy expenditure, when we measure oxygen consumption, we are indirectly measuring an individual's maximal capacity to do work aerobically.

Why is his bigger than mine?

To rephrase, we might start by asking "what are the physiological determinants of VO2 max?" Every cell consumes oxygen in order to convert food energy to usable ATP for cellular work. However, it is muscle that has the greatest range in oxygen consumption. At rest, muscle uses little energy. However, muscle cells that are contracting have high demands for ATP. So it follows that they will consume more oxygen during exercise. The sum total of billions of cells throughout the body consuming oxygen, and generating carbon dioxide, can be measured at the breath using a combination of ventilation volume-measuring and O2/CO2-sensing equipment. The figure below (fig 2.1), borrowed from Prof. Frank Katch, summarizes this process of moving O2 to the muscle and delivering CO2 back to the lungs.

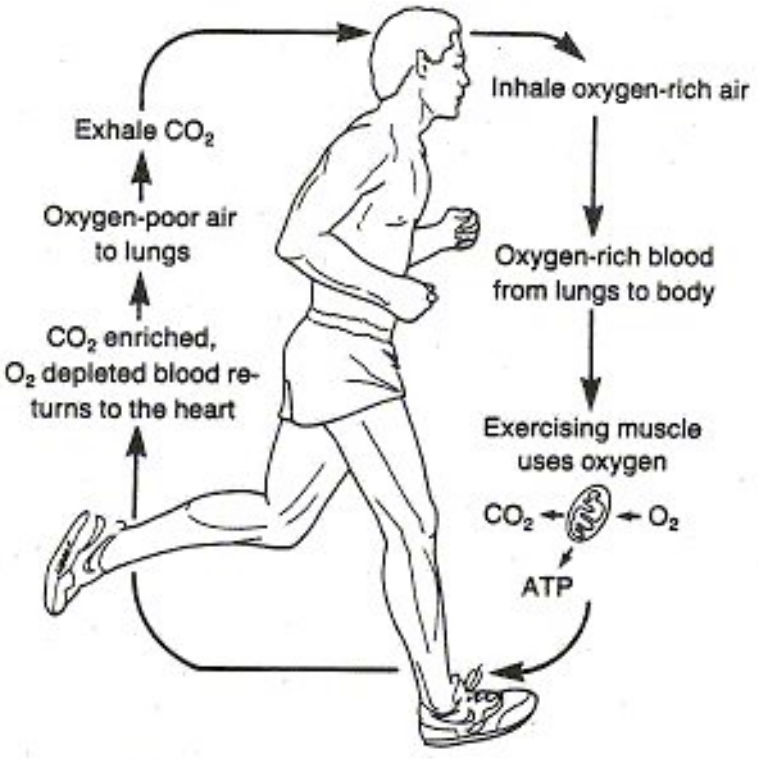


Figure 2.1 The pathways by which oxygen is transported from atmospheric air to the active muscles.

So, if we measure a greater consumption of oxygen during exercise, we know that the working muscle is working at a higher intensity. To receive this oxygen and use it to make ATP for muscle contraction, our muscle fibers are absolutely dependent on 2 things: 1) an external delivery system to bring oxygen from the atmosphere to the working muscle cells, and 2) mitochondria to carry out the process of aerobic energy transfer. Endurance athletes are characterized by both a very good cardiovascular system, and well developed oxidative capacity in their skeletal muscles. We need a big and efficient pump to deliver oxygen rich blood to the muscles, and we need mitochondria-rich muscles to use the oxygen and support high rates of exercise. Which variable is the limiting factor in VO2 max -- oxygen delivery or oxygen utilization? This is a central question that has created considerable debate among exercise physiologists over the years, but for most. the jury is now out.

In the well-trained, oxygen delivery limits VO2 max

Several experiments of different types support the concept that, **in trained individuals, it is oxygen delivery, not oxygen utilization** that limits VO2 max. By performing exercise with one leg and directly measuring muscle oxygen consumption of a small mass of muscle (using arterial catheterization) it has been shown that the capacity of skeletal muscle to use oxygen exceeds the heart's capacity for delivery. Thus although the average male has about 30 to 35 kg of muscle, only a portion of this muscle can be well perfused with blood at any one time. The heart can't deliver a high blood flow to all skeletal muscle, and still maintain adequate blood pressure. This limitation is analogous to the water pressure in your house. If you turn all the faucets on while trying to take a shower, the shower pressure will be inadequate because there is not enough driving pressure. Without getting in too deep on the hemodynamics, it seems that blood pressure is a centrally controlled variable; the body will not "open the valves" to more muscle than can be perfused without compromising central pressure, and blood flow to the brain. The bigger the pumping capacity of the heart, the more muscle can be perfused while maintaining all-important blood pressure.

As further evidence for a delivery limitation, long-term endurance training can result in a 300% increase in muscle oxidative capacity, but only about a 15 to 25% increase in VO2 max. VO2 max can be altered artificially by changing the oxygen concentration in the air. VO2 max also increases in previously untrained subjects before a change in skeletal muscle aerobic capacity occurs. All of these observations demonstrate that VO2 max can be dissociated from skeletal muscle characteristics.

Stroke volume, in contrast, is linearly related to VO2 max. Training results in an increase in stroke volume and therefore, an increase in maximal cardiac output. Greater capacity for oxygen delivery is the result. More muscle can be supplied with oxygen simultaneously while still maintaining necessary blood pressure levels.

In the untrained, skeletal muscle capacity can be limiting

Now, having convinced you that heart performance dictates VO2 max, it is important to also explain the contributing, or accepting, role of muscle oxidative capacity. Measured directly, Oxygen consumption = Cardiac output x arterial-venous oxygen difference (a-v O2 diff). As the oxygen rich blood passes through the capillary network of a working skeletal muscle, oxygen diffuses out of the capillaries and to the mitochondria (following the concentration gradient). The higher the oxygen consumption rate by the mitochondria, the greater the oxygen **extraction**, and the higher the a-v O2 difference at any given blood flow rate. Delivery is the limiting factor because even the best-trained muscle cannot use oxygen that isn't delivered. But, if the blood is delivered to muscles that are poorly trained for endurance, VO2 max will be lower despite a high delivery capacity. When we perform VO2 max tests on untrained persons, we often see that they stop at a time point in the test when their VO2 max seems to still be on the way up. The problem is that they just do not have the aerobic capacity in their working muscles and become fatigued locally prior to fully exploiting their cardiovascular capacity. In contrast, when we test athletes, they will usually show a nice flattening out of VO2 despite increasing intensity towards the end of the test. Heart rate peaks out, VO2 maxes out, and even though some of the best trained can hold out at VO2 max for several minutes, max is max and they eventually hit a wall due to the accumulation of protons and other changes at the muscular level that inhibit muscular force production and bring on exhaustion.

How is VO2 max measured?

In order to determine an athlete's true maximal aerobic capacity, exercise conditions must be created that maximally stress the blood delivery capacity of the heart.

A physical test that meets this requirement must:

• Employ at least 50% of the total muscle mass. Activities which meet this requirement include running, cycling, and rowing. The most common laboratory method is the treadmill running test. A motorized treadmill with variable speed and variable incline is employed.

• Be independent of strength, speed, body size, and skill. The exception to this rule is specialized tests for swimmers, rowers, skaters, etc.

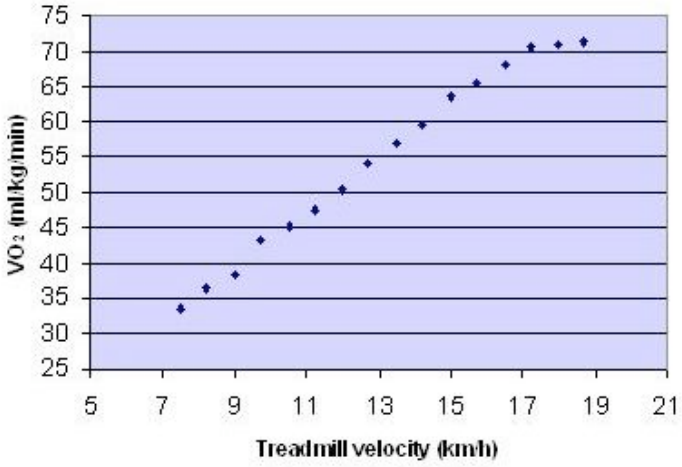
• Be of sufficient duration for cardiovascular responses to be maximized. Generally, maximal tests using continuous exercise protocols are completed in 6 to 12 minutes.

• Be performed by someone who is highly motivated! VO2 max tests are very tough, but they don't last too long.

If we use a treadmill test as an example, here is what will happen. You will go to a good laboratory at a University fitness program, performance testing lab, or hospital wellness center. After a medical exam, and after being hooked up to an ECG machine to monitor cardiac electrical activity, you might start the test by walking on the treadmill at low speed and zero grade. If your fitness level is quite high, the test might be initiated at a running speed. Then, depending on the exact protocol, speed or inclination (or both) of the treadmill will increase at regular intervals (30 sec to 2 minutes). While running, you will be breathing through a 2-way valve system. Air will come in from the room, but will be expired through sensors that measure both volume and oxygen concentration. Using these values and some math, your oxygen uptake will be calculated by a computer at each stage. With each increase in speed or incline, more muscle mass will be employed at a greater intensity. Oxygen consumption will increase linearly with increasing workload. However, at some point, an increase in intensity will not result in an “appropriate” increase in oxygen consumption. Ideally, the oxygen consumption will completely flatten out despite ever-increasing workload. This is the true indication of achieving VO2 max.

In the figure below, we see the results of actual test on a well trained runner performed in our lab with the treadmill incline a constant 5% and velocity increased 0.75km/h each minute. Even well trained athletes cannot stay at their VO2 max very long due to concurrent skeletal muscle fatigue. Other

indications of max VO2 are extreme hyperventilation, and a heart rate of very near 220 minus age that does not increase further with increased workload.



The value you are given by the test administrator will be in one of two forms. The first is called your **absolute VO2 max**. This value will be in liters/min and will probably be between 3.0 and 6.0 liters/min if you're a man and it will be between 2.5 and 4.5 l/min if you're a woman. This absolute value does not take into account differences in body size, so a second way of expressing VO2 max is common. This is called your **relative VO2 max**. It will be expressed in milliliters per min per kg bodyweight (ml/min/kg). So if your absolute VO2 max was 4.0 liters/min and you weighed 75 kg, then your relative VO2 max would be 4000 divided by 75, or 53.3 ml/min/kg. In general, absolute VO2 max favors the large endurance athlete, while relative VO2 tends to be higher in smaller athletes. ***(see subcategory-(iii) below).***

For comparison, the average maximal oxygen consumption of an untrained male in his mid 30's is about 40-45 ml/min/kg, and decreases with age. The same person who undergoes a regular endurance exercise program might increase to 50-55 ml/min/kg. A champion male masters runner age 50 will probably have a value of over 60 ml/min/kg. An Olympic champion 10,000 meter runner will probably have a VO2 max over 80 ml/min/kg! **What about females?** ***(see subcategory-(iv) below).*** The underlying physiology is the same, however specific differences result in lower population values for VO2 max in untrained, trained and champion females when compared to men at a similar relative capacity.

Genetics play a big role

I grew up being told that I could do anything and be anything I set my mind to. I think that was nice of my mother to encourage me that way. However, the

biological reality is that there is a significant genetic component to most of the underlying physical qualities that limit just how “Citius, altius, fortius” we can be with training. VO2 max is no exception. The reality is that if an adult male with a natural, untrained VO2 max of 45 ml/min/kg trains optimally for 5 years, they ***might*** see their VO2 max climb to around 60-65 ml/min/kg. This is a huge improvement. But, alas, the best runners have a VO2 max of 75 to 85 ml/kg. So our hard training normal guy is still going to come up way short against the likes of these aerobic beasts. If they were to stop training for a year, their VO2 max might fall to about where the average guy’s topped out after years of optimal training. How unfair is that? The bottom line is that Olympic champions are born with unique genetic potential that is transformed into performance capacity with years of hard training. Recent studies focusing on the genetics of exercise adaptation have also demonstrated that not only is our starting point genetically determined, but our adaptability to training (how much we improve) is also quite variable and genetically influenced. While the typical person will show a substantial increase in VO2 max with 6 months of exercise, carefully controlled research studies have shown that a small percentage of people will hardly show an increase in VO2 max at all.

One more thing. Just to put things in perspective, the VO2 max of a typical 500kg thoroughbred horse is about 75 liters/min or 150 ml/min/kg! So compared to a horse, even an Olympic endurance champion human comes out looking like a couch potato.

iii) The Impact of Body Dimensions on Endurance Performance

So you want to build a great endurance athlete? Well we know the heart is important, as well as the composition of the working skeletal muscles. What is the effect of the size of the athlete?

Form Follows Function: The "optimal" physical dimensions of an endurance athlete are critically dependent on the specific demands of the sport. What is the resistance that must be overcome? If it is gravity, such as in running or road cycling in the mountains, then a high aerobic capacity relative to bodyweight is most important. If the primary resistance is air (time trial cyclist) or water, then absolute aerobic capacity is most important because bodyweight is supported during the activity.

We will assume for now that skeletal muscle characteristics (i.e. lactate threshold) are identical.

Let's put some numbers to this. Start with an elite road cyclist: 5'7" (1.70m) bodyweight 140 lbs (63.6 kg), absolute VO2 max 5.0 liters/min, (79 ml/kg/min). If we create a **geometrically similar and qualitatively identical athlete** that is 12% taller, he will be 6'3" (1.9 m). His cross sections (bones, muscles, heart) will all be related as the square of 1.7 to the square of 1.9. Because of the increased heart size and, therefore, stroke volume, absolute VO2 max will increase 25% to 6.25 l/min. So, this taller version should be a faster road cyclist right? Wrong. His volume (weight) will increase as a cubed function of height. Therefore our rider will now weigh 195 pounds (88.6 kg). His relative VO2 max will therefore decrease 9% to 71 ml/min/kg. This is still quite high, but not high enough to win major road races. Our big rider will suffer trying to stay with his smaller version in the hills. However, if he learns to row, his absolute increase in aerobic capacity will serve him well because the penalty for carrying around the extra mass will be less severe (there will be some, due to the increased wetted surface area [drag] of his boat).

In fact, these two hypothetical athletes are representative of the physical dimensions and capacities of elite road cyclists and rowers respectively. The men's U.S Olympic rowing team (35 members) averaged 6 feet 4 inches (1.92m) and 194 lbs (89 kg) with an absolute VO2 of 6.25 l/min (data supplied by Fred Hagerman PhD at Ohio University). I do not have exact figures for road cyclists but typical elite class riders have VO2 max values in the mid to upper 70s. And, they are usually smallish, weighing in at 140-150 pounds.

There are some exceptions. Certainly 5 time Tour de France winner Miguel Indirain was one. He was big for a world class climber at ~6 feet tall and 170 lbs, yet he was a great mountain stage performer. Undoubtedly, one explanation for his dominance is that his heart size is disproportionately large relative to his physical dimensions, even for a well trained endurance athlete. That would help explain his resting heart rate of 28 bpm!

And, perhaps the best example of the difference between absolute and relative oxygen consumption comes from the well-known story of 6-time Tour winner Lance Armstrong. His pre-cancer body was more robust and muscular than his post cancer physique. However, he regained his aerobic capacity so that with about 5kg less body mass, his climbing capacity became much better in keeping with his higher weight adjusted maximal oxygen consumption (about 82 ml/kg/min).

iv) Gender Differences in Endurance Performance & Training

This article is long overdue and I apologize to those who were interested in the topic. To bring up the issue of gender differences in physical performance may suggest sexism, but that is not my intention. Historically, there is no doubt that sport has been a center of faulty assumptions and sexism where female athletes are concerned. Social issues, and misunderstanding about female physical and medical limitations (or the presumption of limitations) conspired to slow the development of female performance for many years (the marathon for women was only added to the Olympic schedule in 1984!), but those times are gone, at least among young athletes. Among masters athletes, we still see greatly reduced participation by the older female age groups. This participation difference will no doubt diminish over the next couple of decades. As a result, performances by the oldest females will probably improve more rapidly than those of the oldest males, as this new generation of well trained young female athletes moves into age-group competition, and are joined by more and more talented "late bloomers."

"Old" social norms and habits are still having negative consequences on participation and performance by older (50 +) females. Modern female athletes have repeatedly demonstrated these norms ("women are not built to run long distances" blah- blah-blah) are totally bogus. Currently, teenage daughters are encouraging their formally sedentary mothers and even grandmothers to take up exercise. This transfer of knowledge and norms UPSTREAM is the reverse of what we traditionally see in males (Dad teaching his boy all he knows). However, this is a transitional period for women in sport, so the knowledge transfer across generations is helping to speed the development of women's masters sport.

Having said all that, there ARE some physiological differences between the sexes that impact performance in females independent of age. Some years ago, when the marathon was first becoming a competitive event for women, the rapid improvement in female times led some to predict that female performances would soon equal those of men in the marathon. This has not happened, and it won't. The current world record for women is 2:21, compared to 2:06:50 for the men, a difference in speed of about 10%. This same 10% gap is present across the distance running performance spectrum The reason for the performance gap is not that women don't train as hard as men. There are some important physiological differences between the sexes that can't be overlooked or overcome. I want to point out the most important. Where

relevant, I will try to do so in terms of the BIG THREE Performance adaptations that I have discussed on the MAPP.

The Maximal Oxygen Consumption

The "typical" young untrained male will have an absolute VO2 max of 3.5 liters/min, while the typical same-age female will be about 2 liters/min. This is a 43% difference! Where does it come from? Well first, much of the difference is due to the fact that males are bigger, on average, than females. We humans are all (sort of) geometrically similar, so heart size scales in proportion to lean body size . If we divide VO2 by bodyweight, the difference is diminished (45 ml/min/kg vs 38 ml/min/kg) to 15 to 20%, but not eliminated. What is the source of this remaining difference?

If we compare average bodyfat in males and females, we find part of the answer. Young untrained women average about 25% bodyfat compared to 15% in young men. So, if we factor out body composition differences by dividing VO2 by lean body mass (Bodyweight minus estimated fat weight) the difference in maximal O2 consumption decreases to perhaps 7-10%. Keep in mind though that this is only a meaningful exercise on paper. *A female athlete cannot expect to improve her performance by reducing her bodyfat down to the sub 7% levels that are often observed in elite males. The health consequences for the female are too severe!*

To find an explanation for the remaining 10% difference we must go back to the key limitation on VO2 max, oxygen delivery. On average females have a lower blood hemoglobin content than males, up to 10% lower. Finally, there is some evidence, that the female heart is slightly smaller relative to body size than the male heart. Recent ECG and echocardiographic studies also suggest that the young female heart exhibits less enlargement in response to either endurance or resistance training than the male heart (George et al, 1995) This may be due to differences in androgen receptor density in the female heart. A smaller heart would be expected to be a less effective pump.

Slightly lower oxygen carrying capacity of the blood (lower hemoglobin levels) plus a somewhat smaller or less adaptive heart are sufficient to account for the gender differences in maximal oxygen consumption that are independent of body size and fat percentage.

It is worth noting here the results of a 1993 study by Spina et al. Their data suggested that in previously sedentary older men and women (60 to 65 years

old) who trained for 9 months to a year, both men and women increased their VO2 max by the same amount (an average of 20%). **However, the mechanism of improvement was different.** The men improved primarily by increasing maximal cardiac output due to higher stroke volume. This is just the pattern of response I have previously described ***(see subcategory-(ii) above).*** However, the older women did not demonstrate any increase in cardiac performance, but rather increased oxygen consumption by improving oxygen extraction by the working muscles, due to greater capillarization and more mitochondria. This data supports previous studies in 60+ year old women that show no cardiac hypertrophy in response to endurance training.

To summarize, their is a growing body of data suggesting that females demonstrate a somewhat different pattern of cardiac adaptation to exercise, which may become more dissimilar with age. They also generally have a lower hemoglobin level by several percent. The net effect is a small but significant difference in maximal oxygen consumption, even among similarly trained males and females, and after scaling for differences in size and body composition.

It is important to make note of the fact that these differences are "on average". In reality, there are **many** women with significantly higher VO2 max values than average men. However, if we look at the "best of the best", the differences persist. Using XC skiing as an example from here in Norway, the highest reliable values for VO2 max recorded in national team XC skiers are about 90 ml/min/kg. The very best Norwegian woman has been measured at 77 ml/min/kg, a 17% difference. So, while this woman will outperform 99.9% of all men, she will not out-perform the national team level males.

The Lactate Threshold

Now we come to the second component of endurance performance, the lactate threshold. As a review, this is the exercise intensity at which lactic acid begins to accumulate in the blood stream at levels significantly above "baseline" values. This intensity sets a (slightly fuzzy) boundary between that exercise intensity which can be sustained for long periods (over one hour) versus those which lead to fatigue in minutes. We have already discussed the fact that changes in the lactate threshold are due to adaptations that occur in the exercising muscle. We call these *peripheral* adaptations (Changes in cardiovascular performance are called *central adaptations*).

The question here is, do women demonstrate a different pattern or capacity for peripheral adaptations then men? As best as I can tell, the answer is NO.

First, Female skeletal muscle is not distinguishable from male skeletal muscle. Second, within some margin of error, the fiber type distribution (percentage of slow versus fast fibers) is not different in the male and female population. Third, male and female skeletal muscle responds similarly to endurance exercise. Finally, elite female endurance athletes have similar lactate threshold values compared to men when expressed as a percentage of their VO2 max. Elite women perform at the same high percentage of their maximal oxygen consumption as their male counterparts.

Some years ago it was proposed by some that women would actually perform **better** at ultra-endurance type activities. This theory has been disproved both in the laboratory and in practice as a performance difference persists in the ultramarathon events. Some of you may balk and recall a recent Running Times article that suggested women had an edge in the really long events. They discussed a study in which a group of male and female runners who were matched for marathon time were raced head to head in the Comrades marathon, a 90k race. The women won by 54 minutes, suggesting a female edge in longer events. The problem with this study is that when you match men and women for performance, the women are relatively better runners and probably have a higher slow twitch fiber percentage. This advantage becomes bigger in an ultradistance event.

The fact remains that the performance gap between male and female record holders in the really long running races 50k to 6 days is actually more on the order of 15 to 20%, instead of the 10% difference for the standard distances. Part of this larger gap may be to lower participation, and the fact that the most talented females have not yet tested themselves over the ultradistances. But at elite level, I don't think the gap will disappear.

Efficiency

The third component of endurance performance is **efficiency** which of course has different constraints, depending on the sport. The research information comparing the efficiency of female and male athletes is both sparse and inconclusive. In running, for example females have been found to be more, less, and equally efficient compared to males depending upon the specific study. Some of this confusion comes down to how the differences in bodyweight and bodyfat were accounted for.

After looking over some of the research comparing running economy between genders, I started to go into a couple of studies, but it all starts to become a scaling and factoring game, which I like, but you probably can do without it. So, I decided to just summarize things this way. Currently, I would argue that any inherent economy differences in male and female runners are smaller than the individual variation in running economy that is observed among runners, independent of gender. I would support that argument by suggesting that the differences in VO2 max observed between elite males and females are sufficient to explain the "10% gap" without other factors being involved. If more data comes to my attention to dispute this, I will share it.

Now, if we look at efficiency/economy differences in other sports, things mostly boil down to body shape/anthropometric differences. In situations like running or cycling, these may actually favor females in general, due to narrower upper bodies for a given total body mass, and potentially less wind or water drag. As I have shown for rowing , differences in VO2 max alone are sufficient to explain the gender performance gap in rowing. I am not aware of any research studies to support or dispute this, but it seems that there are no differences in rowing efficiency among male and female rowers of similar relative ability.

Fat metabolism differences?

Back in the 70s, a theory got started that said "Since women have more fat stores, they will be better at utilizing fat during endurance performance when glycogen stores are depleted." One of the supporting pillars of the theory was that it had been noticed by one female runner/author how "fresh" many female runners looked as they crossed the finish line! Well, this shaky theory was crushed under the harsh light of science. Back in 1979, Costill and colleagues compared males and females who were equally trained during a 60 minute treadmill run. There were no differences in any measures of fat metabolism. These guys even took some muscle out of the runners' legs and tested it in a test tube. Still no difference! This is an often repeated finding among similarly trained males and females. There is no gender difference in the ability of men and women to burn fat!

Are Men Sweatier than Women?

On an absolute basis, and per kg bodyweight, women have lower sweat rates than men. However, because of their higher body surface area to volume ratio, they dissipate heat equally well. Men have an advantage in evaporative

cooling, but women have an advantage in radiant cooling, so they come out even.

Summary So Far

Of the three critical components of endurance performance, the only one that is clearly and consistently depressed in females is the maximal oxygen consumption. Even after accounting for differences in bodyweight and body fat percentage, a gap of roughly 10 - 15% remains. Now I want to talk about some other comparisons that don't fit so cleanly into the Performance Model for endurance performance.

Muscle Strength and Power

Although maximal muscular strength and anaerobic power has little to do with pure endurance performance, there are many events which can be classified as "power-endurance" events. These events ranging from 2 to about 8 minutes require some combination of aerobic and anaerobic capacity. For this reason, I think it is important to also consider this "anaerobic" component of the performance package. When we talk about anaerobic capacity, the critical determinant is muscle mass. Females, on average, have less total muscle mass than males. As a result, maximal strength measures as well as maximal power measures (power = force/time) are reduced. Gross measures of upper body strength suggest an average 40-50% difference between the sexes, compared to a 30% difference in lower body strength. What about power? Maud and Schultz compared 52 men and 50 women, all about 21 years old using a maximal power test on a bicycle ergometer. Peak power was about 60% lower for the females when comparing absolute values. But, the men were heavier. Peak power per kg bodyweight was more similar, 9.3 watts/kg vs 7.9 watts/kg for the women, an 18% difference. Finally, when power outputs were adjusted for fat-free mass, the values were 10.4 watts/kg and 9.9 respectively. This 5% difference was not statistically different. Numerous other studies using different techniques have demonstrated that when you just look at muscle **quality**, male and female muscle is not different. Within the accuracy of current comparative techniques, it appears that the strength and power differences between the sexes are a function of muscle **quantity** only. Biomechanical differences probably play a role in some situations, but this will be very sport specific.

Should Men and Women Train Together?

OK, now we move onto something a little different. I think there are two reasons for making this gender comparison. First, I think it is useful to understand that at the elite levels, male and female performance differences are physiological in origin, not a function of differences in training, desire etc. (The one caveat to this is among the oldest athletes. Here, I think the gender performance gap is probably still wider than it will ultimately be, due to differences in participation and training intensity among the oldest age groups.) The second reason is a very practical one. Men and women live together, work together, and often train together, either as husband and wife, as friends, or as part of a training group. So, if we are going to train together, I think it is pretty important that we understand each other as athletes. Athletes are not just bodies. They have brains too! No, really, they do.

Psychological Differences?

I have examined briefly some physiological gender differences. Now I want to move into the psychological realm. Oh boy, now I am really treading on thin ice, but I'm safely and happily married, and living pretty far away from most everyone I know here in Norway, so I am going to proceed. **As a broad generalization**, here are a few things I have noticed, read, experienced etc. that I think are important regarding males and females training together. Again, let me repeat. THESE ARE GENERALIZATIONS. For every point I will make here, I myself have seen just the opposite behavior on occasion.

The Numbers Game

If you walk into a fitness center, teeming with men and women huffing and puffing on all manner of computerized exercise machines, take a look at the men first. As a rule, they will be staring directly at the computer screen, calculating, extrapolating, comparing the numbers with their previous efforts, or with measured glances, to the guy on the next machine. **Males are number guys.**

Now take a look at the women. In my experience, most are using one of several dissociation strategies. They are either listening to music, reading a book or magazine, or simply covering the entire computer screen with a towel. Some use all three methods in combination. The bottom line, is they are NOT paying attention to all the blinking lights! In fact, when I have had the nerve to

inquire about this, most tell me that they hate all the numbers, clocks, bells, and whistles.

Now, most of these folk aren't athletes, but I think the tendency remains among the competitive set. On the sports lists that I lurk on via email, and the messages I get from you guys, it is mostly the men that are getting caught up with heart rate, time, power output etc. Men seem to need to **quantify** their training in as much detail as possible. Have you noticed how men are more likely to keep training logs then the women? Meanwhile, I would argue that women are more sensitive to qualitative, internal, measures of training effectiveness. Which method is better? Neither. We can definitely learn from each other. Sometimes the numbers are helpful for getting us over specific hurdles. They also help us to see small changes in performance and evaluate the effectiveness of our training. On the flip side, a more qualitative approach helps to take some of the internal pressure off sometimes. If the odometer, speedometer, or HR monitor rules our heads, then we men often find ourselves "competing" every workout. This is a sure-fire prescription for become stale and overtrained.

The Sociology of the Training Group

I have not read "Men are from Mars, Women are from Venus", but I have read some similar stuff. Basically, I have to go along with the idea that men and women communicate differently. Men tend to be more hierarchical, while the women develop better horizontal lines of communication. What the heck does this have to do with performance? This is my slant. Men who were active in sports as youth are very familiar with the pecking order mentality. In sports, some get picked first, others get picked last. You have first team and second team etc. "It's not personal. He is just a little faster than you Charlie." Watch a bunch of guys in competitive practice situation. My most recent personal experience is in rowing. On the water, we would do daily battle against each other in our singles. Sweating, grimacing, taunting, yelling, winning, losing. Then we get to the dock, get out of the boat and say "Great workout. See ya tomorrow." On the water each day a hierarchy was established and defended, then dissolved as soon as practice was over. Another example came to me from a coxwain for the men's lightweight national team who was now coaching collegiate rowers. He made the point that the national team athletes had the ability to turn everything on in practice. Then, as soon as practice was over, they forgot about it. No internalization, no dwelling on successes or failures during times when nothing can be done about them!

In my experience, this separation of competition within and outside of the training environment CAN be more difficult among female athletes. The same qualities that often make them more effective communicators and empathizers, also can lead to personalizing the physical battles of daily training. In the extreme it can splinter a team. I have observed it (from a safe distance) in rowing among masters women. Most of those women were not competitive athletes in their youth. Perhaps this made a difference. Battles for seats in the boat waged on the water and coaching decisions that resulted were not forgotten or accepted when practice was over. The women didn't seem to know how to communicate under these new conditions. Disaster!

Competitive training situations are generally good, I think. It helps to train with others who share your goals. However, everyone has their own optimal amount of competitive stimulation. Even among world class athletes, some thrive in an aggressive team environment, and others don't. Here in Norway, I know the coach of last year's top female XC skier in Norway, Marit Mickelsplass. She rose to new levels on the international circuit (Top 3 in the world) this year after leaving the national team, and training on her own. The problem was that the stress of daily training in the aggressive team atmosphere was too much **for her**. Psychological stress led to physical stress and overtraining. Thanks to a good coach who understood the link between psychology and physiology, the problem was solved. I guess my view is that there are often going to be subtle differences in the approach that a coach is going to need to take with female athletes versus male athletes. Failure to understand these differences can impact performance.

Can Women and Men handle the same training Volume?

Here, we return to some physiology. Talking with elite level coaches leads me to believe that there are small but important differences in the recovery capacity of male and females, at least when pushed to the extremes of elite level training. Again I will go to evidence from world class XC skiers here in Norway. It appears that the best women perform optimally at a training volume that is perhaps 10 -15% lower than that observed in the best men. Increasing the volume in the women does not improve results, and often leads to overtraining. The general consensus is that the difference lies in the higher average testosterone levels of males. Remember, testosterone is an anabolic hormone. This means it is critical for tissue growth and repair. Anecdotally, I have been told that only one of the Norwegian female national team skiers has been able to maintain the average yearly training volume (measured in hours) that is maintained by the entire Russian female team. The difference appears

to be steroid use, but of course this is only a rumor. At any rate, I think we should be aware that there is probably a small gender difference in recovery capacity from hard or high volume training, in addition to the individual variation that is observed.

You and your Significant Other as training partners

I married a woman who loves exercise. Heck, that is one of the reasons I was so attracted to her! We focus on different sports, but sometimes we workout together, either running, cycling or XC skiing, depending on the season here in Norway. What we learned pretty quickly was that we couldn't do the same type of workout together effectively. If we run at the same speed, I can be comfortable just under my lactate threshold. Meanwhile she is teetering on the edge of disaster as she runs at or above hers. I have a good run, she is miserable. The same thing happens on a bicycle. What is the solution? Well one is we can just not ever train together. Neither one of us like that idea. So we compromise. Sometimes I train alone. These workouts are usually hard interval sessions or lactate threshold workouts. Then, when we train together, I am running or cycling at a good steady state aerobic pace, and she is doing a tempo run or lactate threshold session on the bike. The bottom line is that we had to understand where we were both at physiologically and make the adjustments necessary to allow us both to profit from our joint training sessions, and not become frustrated with each other.

Even small differences in the performance capacity of you and your partner can be problematic if they are not recognized. The slower partner who always works a little harder to keep up can be at risk of overtraining, or just not achieving the goals of the workout. This may be the man, or the woman. Either way, it can be avoided by taking the time to evaluate the performance difference and make adjustments in the training schedule. One thing is likely. You probably should not train together all the time. Find time for common workouts, but make sure that there remain training sessions were there are no compromises being made. If this means training alone, then do it. *It doesn't mean I don't enjoy your company, dear!*

Final Words

OK, I think that pretty well hits the main points, from my current vantage point. The bottom line is that there IS a physiological explanation for the gender performance gap observed in endurance (and power)sports. Keep in mind that the best women can still beat 99%+ of the men. However, if you ask me "When

will women run as fast as men." I will answer, "just as soon as they have the same VO2 max as men. Grete Waitz probably said it better, *"As long as women are women, I don't think they will surpass men."*

I have read at least one very good and quite popular running book, "The Lore of Running" that has tried to explain the gender performance differences in terms of some unmeasured but imagined difference in muscle quality. He has been forced to assume this angle, despite absolutely contrary data, becasue of his even more unsupported theory that VO2 max is actually not limited at all by cardiac performance. To be honest, this view takes about as much denial of the available data as that of the tobacco industry denying that smoking is bad for you! Enjoy this otherwise wonderful book, but don't read the physiology chapters.

I realize I have skipped over a tremendous area of difference related to the impact of the menstrual cycle and pregnancy on training on performance in females. However, I think there are a lot of excellent resources by much more qualified people available for women athletes with questions on this topic.

As for the presumed psychological differences, I think they are real, but I realize that there are many exceptions. So, please don't flood my mailbox with white hot flame-mail! At least my wife still loves me.

b) Myocardial Adaptations to Training

The heart, in cellular composition, structure, and mechanics, is an absolute marvel of "biological engineering". Even among human couch potatoes, it is an astoundingly well equipped endurance muscle. It has an incredibly dense network of capillaries (over 2000 capillaries per cubic millimeter!) designed to provide reliable delivery of oxygen to the working muscle with a minimum diffusion distance to intracellular mitochondria. The individual heart cells (myocytes) are densely packed with mitochondria. About 25-30% of the human heart cell volume consists of mitochondria. In contrast, mitochondria make up less than 5% of the untrained skeletal muscle cell volume. The specific biochemistry of the muscle cells is designed to minimize lactate production even at very high workloads (H isoform of lactate dehydrogenase for you scientists). The heart can metabolize fat, lactate, and blood glucose with equal effectiveness.

So, how can endurance training improve a muscle that is already superbly designed and equipped to perform constant work? The answer is fairly simple. **IT GETS BIGGER! (OK, it's slightly more complicated than that. *See subcategory-(iii) below***). Endurance trained hearts do not beat faster at maximum. They do not beat more powerfully, gram for gram. They also do not change significantly in terms of mitochondrial or capillary density. The distinction between the athlete's heart and the sendentary heart is the **larger stroke volume of the trained heart**. This improvement is critical to improved endurance performance. Why? The heart is first and foremost a pump. It pumps oxygenated blood to the body to support the production of cellular energy. During exercise, working muscles increase their cellular energy requirements up to 100X. Generating more energy (ATP) requires more oxygen delivery to the mitochondria.

The quantity of work that can be performed by the muscles is critically dependent on the volume of blood that can be delivered by the heart. A body supplied more oxygen by a bigger pump has the potential to sustain work at a greater maximal intensity. Maximal Cardiac Output = Maximal Heart Rate x Stroke Volume. **Stroke volume is the volume of blood ejected from the left ventricle each beat.** Endurance training impacts myocardial function 1) at rest, 2) during submaximal exercise, and 3) during maximal exercise.

Resting Hemodynamics and Exercise

At rest the stroke volume and resting heart rate of the average person can be remembered easily as approximately 70 ml/beat and 70 beats/minute. This gives us 70x70 or about 5 liters/minute resting cardiac output. The resting cardiac output is determined by the oxygen demand at rest, and also by the need for high blood flow to the kidneys for filtration purposes. It doesn't change appreciably with endurance training. However, the manner in which the heart delivers this resting demand does change. After 6 months of endurance training, the resting heart rate may decrease to 55 bpm. At the same time, resting stroke volume increases to about 90 ml (HR x SV stays the ~same before and after training). So a reduced resting heart rate is a hallmark of endurance training. Resting heart rate (RHR) can be much lower. In champion endurance athletes, RHR is often in the 30s and low 40s. Since resting oxygen demand still hasn't changed, this should tip you off that these athletes have extremely high resting stroke volumes! Thus, the resting heart of the athlete is more efficient. It performs the same work with fewer beats and less myocaridal energy demand. However, since some medical symptoms are also marked by

a reduced resting heart rate, your physician may initially raise his/her eyebrow to your low frequency lub-dub during checkups.

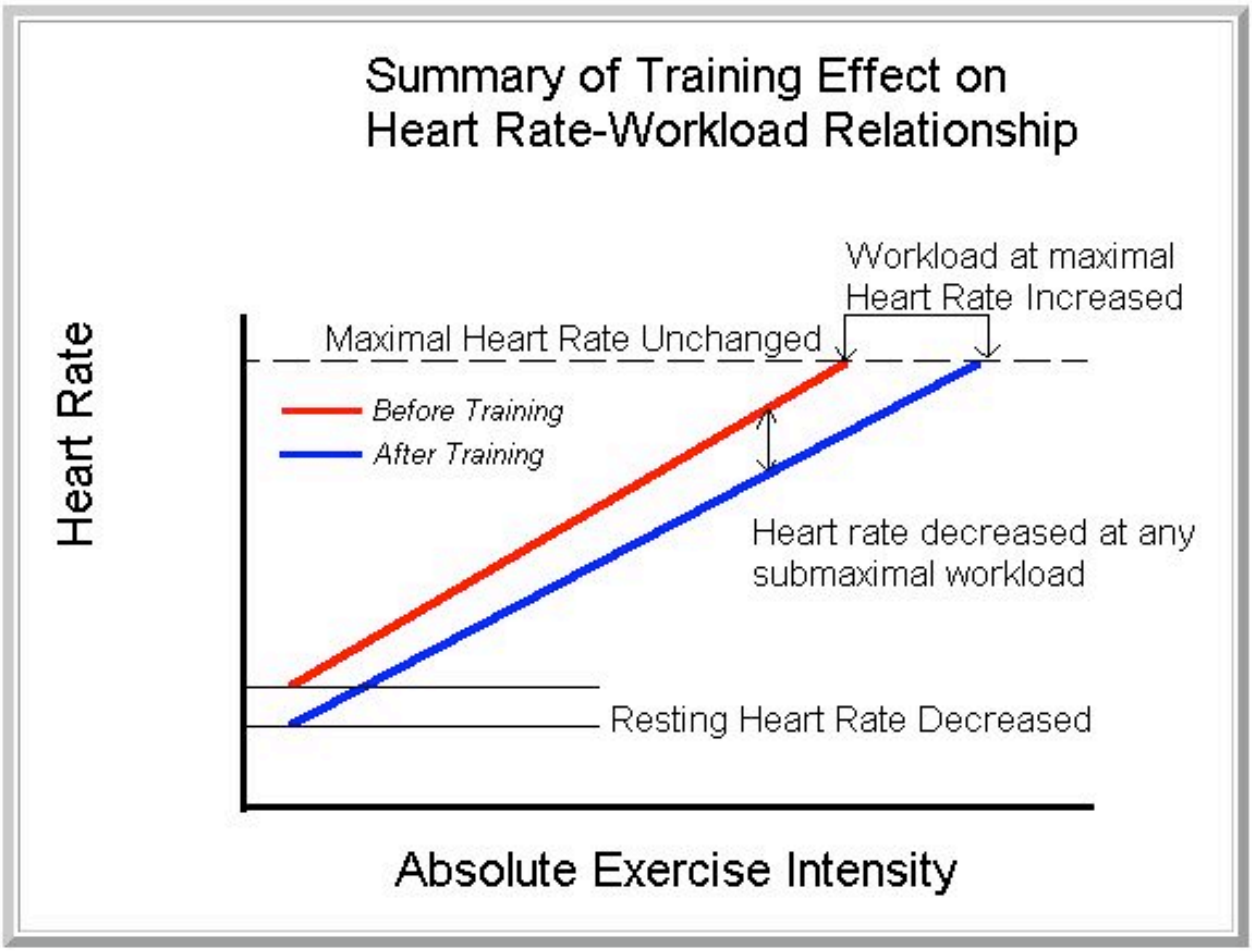
Myocardial Responses to Submaximal Exercise Before & After Training

When we begin to exercise at any given intensity, more oxygen must be delivered to the working muscle. Cardiac output increases in proportion to the increased energy demand. If we measure the responses of an individual to running at 8 min mile pace before and after 3 months of regular exercise, here is what we will see. First, the metabolic cost of working at this intensity will be unchanged (assuming no improvement in running efficiency). Therefore cardiac output will be the same. However, just as during rest, the heart will deliver more blood each beat. Therefore heart rate at this and any submaximal exercise intensity will be reduced. If we use the analogy of a car engine. We have replaced a small motor with a larger one that achieves the same horsepower at lower rpms.

Hemodynamic Response to Maximal Exercise

There is for all of us an exercise intensity that will elicit our maximum cardiac output. Once this limit is achieved, further increases in work intensity will result in no further increase in heart rate. By definition, this is then the maximum heart rate. **The maximum heart rate in humans varies from individual to individual and decreases with age.** ***(see subcategory-(c) below).*** Therefore the only way to know precisely what a specific person's maximal heart rate is would be to do a maximal exercise test. Without such precise knowledge, we often use the formula "220 minus age" to approximate maximal heart rate. This formula will generally give results within plus or minus 10 bpm of reality. True maximal heart rate may not be achieved in some forms of exercise that do not employ a large enough muscle mass, or if the person is unfamiliar with the mode of exercise employed. For example, one person may have a true maximum heart rate of 195 achieved during uphill running, but only 191 during a cycling test, and 187 during swimming. These latter heart rates are termed peak heart rates and should be used as a basis for determining training intensity for a specific exercise mode.

The important thing to remember is: ***Maximal heart rate does not increase after training. It stays the same (or might even decrease just slightly). However, maximal stroke volume increases. Therefore maximal Cardiac Output increases in response to exercise. This is the primary reason for the increase in VO2 max!***



So, in response to endurance exercise the heart adapts by increasing stroke volume at rest, during submaximal exercise, and during maximal exercise. There is some debate regarding whether stroke volume increases BECAUSE heart rate is decreased (increasing diastolic filling time), or because of an increase in ventricular volume due to eccentric hypertrophy of the heart muscle. Both factors probably contribute based on the available data. Both changes also rapidly revert towards normal with the cessation of training. One other important change that takes place is an **increased blood volume**. Increased blood volume helps to take advantage of the increased filling capacity of the heart and facilitates increased stroke volume. This adaption occurs fairly rapidly with training, but is also the first adaptation lost if we stop training for several days!

c) Aging and Cardiovascular Function

World records in endurance sports are not accomplished at age 55. Why? Because one of the unavoidable consequences of aging is a decline in the maximal capacity of the cardiovascular system to pump blood and deliver oxygen while removing metabolic waste products. The components of cardiovascular pump performance are 1) the maximal heart rate that can be achieved, 2) The size and contractility of the heart muscle and 3) The compliance (stiffness) of the arterial tree. We will look briefly at what is known about aging effects on each of these variables.

Maximal Heart Rate

Young children generally have a maximal heart rate approaching 220 beats per minute. This maximal rate falls throughout life. By age 60 maximal heart rate in a group of 100 men will average about 160 beats per minute. This fall in heart rate seems to be a linear process so that maximal heart rate can be estimated by the formula **220 - AGE**. This is an ESTIMATE, however. If we actually measure the maximal heart rates of those same 100 men during a maximal exercise test we would probably see a range of heart rates between 140 and 180. There is no strong evidence to suggest that training influences the decline in maximal heart rate. This reduction appears to be due to alterations in the cardiac electrical conduction system (SA node and Bundle of His), as well as down regulation of beta-1 receptors, which decreases the heart's sensitivity to catecholamine stimulation.

Maximal Stroke Volume

The research picture regarding age effects om maximal stroke volume is far less clear. This is in part due to the technical challenges involved in making these measurements. Studies showing a decline, an increase, and no change can be found in the literature. It appears that if middle-aged and older adults continue to train intensely, stroke volume is well maintained. Heart size in older athletes has been shown to be similar to that of young athletes, and bigger than their sedentary, same-aged peers. Ultimately, maximal stroke volume appears to decrease due to a 1) decrease in training volume and 2) an increase in peripheral resistance.

The Peripheral Resistance

The blood pumped out of the heart enters the systemic arterial system. In our youth, this system of arteries is quite flexible or compliant. This is important for the performance of the heart. Compliant vessel walls stretch when blood is pumped through them, lowering the resistance that the heart must overcome to eject a volume of blood each beat. As we age, these vessels lose their elasticity. Consequently, resting blood pressure and blood pressure during exercise slowly increase as we age. Continued training appears to reduce this aging effect, but does not eliminate it. Increased peripheral resistance results in a decrease in maximal blood flow to working muscles. However, at submaximal exercise intensities, the 10-15% decrease in blood flow is compensated for by increased oxygen extraction (a-v O2 difference). This compensation is probably possible due to the increased transit time of the blood through the capillary tree.

The Big Picture

In the sedentary population, cardiovascular performance declines progressively with age. However, much of this decline is due to 1) physical inactivity and 2) increased body weight (fat). Maximal oxygen consumption declines about 10% per decade after age 25 ***(see subcategory-(ii) above).*** However, if body composition is maintained and physical activity levels are kept constant, the decline in VO2 max due to aging is only about 5% per decade. Prior to age 50, this decline may even be less, perhaps 1-2% per decade in hard training masters athletes. Ultimately, cardiovascular capacity is reduced however, due to the unavoidable decline in maximal heart rate.

d) Understanding Heart Rate and Exercise

If you are reading the MAPP, chances are you already use heart rate as a measure of your exercise intensity. The basics are that 1) heart rate serves as a measure of exercise intensity during steady state activities, and 2) If we estimate maximal heart rate as 220 minus age we can use this value to gauge the intensity of our training. That is not the whole story though. Here are a few details that can be important in your training and racing.

• **The "220 minus Age" formula is Only an Estimate.** Your actual maximal heart rate in a given activity could be 10 to even 20 beats higher or lower than

the estimated value. This has important implications for judging training intensity.

• **Your Maximal Heart Rate Differs in Different Activities.** Cardiac hemodynamics and maximal sympathetic drive are influenced by 1) body position during exercise and 2) muscle mass involvement. So, a triathlete with a max heart rate during running of 180, may only hit 176 on the bike, and 171 during swimming. In this case we call the running heart rate "Maximal Heart Rate" and the highest heart rate observed in cycling and swimming, "Peak" heart rate, for that event. Knowing your peak heart rate for each discipline will help you to more accurately gauge the intensity of your training. If the activity is restricted to upper-body muscle mass, peak heart rate will generally be considerably lower than in whole body activities. Examples include kayaking and double poling during cross-country skiing. Highly trained athletes can achieve a higher percentage of true max heart rate when performing small muscle mass activities.

• **A Better Method for Gauging Exercise Intensity with Heart Rate.** For a given exercise mode, heart rate will increase linearly with exercise intensity, and therefore, oxygen consumption. However, the resting heart rate creates an offset between % of HR max and the associated % of "peak" oxygen consumption for that activity. For example, running at 65% of heart rate max corresponds to approximately 50% of VO2 max. At 87% of HR max, you are at about 77- 83% of VO2 max. Depending on your resting heart rate, heart rate and VO2 percentage finally converge at 100%. I prefer to use HEART RATE RESERVE as my training intensity guide. To do this I need to know 1) my resting heart rate, and 2) my peak heart rate for that specific activity. The first one is easy to determine. The second one may sometimes be a slight estimate. My current resting heart rate is about 36 beats/min. My peak heart rate during rowing is about 181. So my heart rate range is 181-36 or 145 beats. Now, if I want to train at 85% of my peak VO2 for rowing, I will take 85% of my heart rate reserve (0.85 x 145 = 123) and add it to my resting heart rate (123 + 36 = 159). PERCENTAGE HEART RATE RESERVE will give a better approximation of % maximal oxygen consumption than just % max heart rate. And, it is more accurate because you can adjust for changes in your resting heart rate.

• **Body Position on the Bike will Influence Heart Rate.** Let's say I am riding on an indoor bicycle trainer with my upper body parallel to the ground (Hands on the drops) at a heart rate of 145. Raising upright while continuing to cycle at the exact same workload will result in an increase in heart rate of about 5

beats per minute. Trust me I have experimented with this effect on many a winter evening! This is due to decreased venous return in the more upright position. Heart rate increases to compensate for the slightly decreased venous return and stroke volume, keeping cardiac output constant. When I return to the drops, the heart rate drops again.

• **Temperature Will Greatly Influence Heart Rate.** Above about 70 degrees Farenheight (21C), Heart rate at a standard submaximal intensity will be increased about 1 beat/min per degree F increase in temperature. Thus, a steady state run at a heart rate of 150 on a 70 degree Spring day, may have you close to maximal heart rate on a scorching 95 degree day in July, if you try to maintain the same speed. I am from Texas, so I remember these days well. The oxygen demand doesn't increase in the heat, but the thermal stress load does. As a result, your cardiovascular system must divert blood flow to the skin to enhance heat dissapation. Since you only have so much cardiac output, this means a lower maximal steady state speed in the heat, or early exhaustion. You choose. My choice is generally to avoid running in 95 degree heat.

• **Humidity Hurts Too for the Same Reasons**. A higher relative humidity will increase heart rate at a submaximal workload. Increased humidity decreases the evaportation rate of sweat. This means the body has to resort more to heat removal via increased skin blood flow. Data from Wilmore and Costill *"Physiology of Sport and Exercise"* shows a 10 beat increase in heart rate from 165 to 175 when running in 90% humidity compared to 50%. This is the difference between a morning and afternoon workout in many parts of the country.

• **What about the Time of Day?** Our bodies show diurnal (time of day) variations in many physiological responses. Within the normal range of times that you might be training, this can result in a 3-8 bpm difference in heart rate at rest, during moderate exercise, and during recovery. The differences during maximal exercise are probably smaller. Data demonstrating this effect is in the literature. However, I suspect the exact pattern of these changes can be altered by your specific exercise pattern. For example, after several years of rowing before sunrise, I am sure my diurnal response pattern was modified. I have no data to support this assumption, but I do know that I was transformed from an afternoon exerciser, to a morning guy! So, my best guess is that you should not be too surprised by small differences in heart rate response if you do your training at an unusual (for you) time of day.

• **What is Cardiovascular Drift?** If you begin a 90 minute steady state ride on your bicycle trainer at a controlled intensity, your heart rate may be 145 after 10 minutes. However, as you ride and check your heart rate every 10 minutes, you will notice a slight upward "drift". By 90 minutes, your heart rate may be 160. Why is this happening if intensity is held constant? There are two explanations. As you exercise, you sweat (dah). A portion of this lost fluid volume comes from the plasma volume. This decrease in plasma volume will diminish venous return and stroke volume. Heart rate again increases to compensate and maintain constant cardiac output. Maintaining high fluid consumption before and during the ride will help to minimize this cardiovascular drift, by replacing fluid volume.

There is also a second reason for the drift during an exhaustive exercise session. Your heart rate is controlled in large part by the "Relative" intensity of work by the muscles. So in a long hard ride, some of your motor units fatigue due to glycogen depletion. Your brain compensates by recruiting more motor units to perform the same absolute workload. There is a parallel increase in heart rate. Consequently, a ride that began at heart rate 150, can end up with you exhausted and at a heart rate of 175, 2 hours later, even if speed never changed!

2) SKELETAL MUSCLES

a) Basic Skeletal Muscle Physiology

This is intended to be a bare-bones review of physiology of muscle function. There are numerous sources on the internet for those who are interested in a more in-depth exploration of skeletal muscle physiology. The concepts here have direct application to understanding how specific training improves (or decreases) endurance performance capacity.

Basic Architecture

A single muscle fiber is a cyclindrical, elongated cell. Muscle cells can be extremely short, or long. The sartorious muscle contains single fibers that are at least 30 cm long. Each fiber is surrounded by a thin layer of connective tissue called endomysium. Organizationally, thousands of muscle fibers are wrapped by a thin layer of connective tissue called the perimysium to form a muscle bundle. Groups of muscle bundles that join into a tendon at each end are called muscle groups, or simply muscles. The biceps muscle is an example. The entire muscle is surrounded by a protective sheath called the epimysium. Between and within the muscle cells is a complex latticework of connective tissue, resembling struts and crossbeams that help to maintain the integrity of the muscle during contraction and strain. It is an amazing cellular system even before it contracts!

Interior Components

Every muscle cell contains a series of common components that are directly associated with contraction in some way, and influenced by training. I will briefly describe these. For now we will not worry about the rest (like the nucleus, ribosomes etc.).

1. **The Cell Membrane:** - Controls what enters and leaves the cell. Contains regulatory proteins that are influenced by hormones like epinephrine (adrenalin) and insulin. The blood concentration of these hormones greatly influences fuel utilization by the muscle cell.

2. **Contractile Proteins:** - The contractile machinery of a muscle fiber is organized into structural units called sarcomeres. Muscle length is determined by how many sarcomeres are lined up in series, one next to the other. Muscle thickness ultimately depends on how many sarcomeres line up in parallel (one

on top of the other). The sarcomere structures consist of two important proteins, actin and myosin (about 85% by volume). Several other important proteins called troponin and tropomyosin, and proteins with cool names like titin, nebulin, and desmin help to hold these units together. The sarcomeres are organized as many thin myofibrils. A single muscle fiber will contain 5 to 10,000 myofibrils. Each myofibril in turn contains about 4500 sarcomeres. Multiply the number of muscles in the body by the number of muscle fibers per muscle by the number of myofibrils per fiber by the number of sarcomeres per myofibril and well, the numbers become pretty staggering. It is the individual myofibrils, long chains of sarcomeres, which actually produce force in the muscle cell. All of the rest of the machinery plays a supporting or repair function.

3. The Cytosol: This is the aqueous fluid of the cell. It provides a medium for diffusion and movement of oxygen, new proteins, and ATP within the cell's interior. The cytoplasm also contains glycogen, lipid droplets, phosphocreatine, various chemical ions like magnesium, potassium and chloride, and numerous enzymes.

4. Mitochondria: - The organelles in each muscle cell that contain oxidative enzymes consume oxygen during exercise. Recent research suggests that mitochondria may look more like an interconnected network than little isolated oval "powerhouses" shown in most old textbooks. Mitochondria convert the chemical energy contained in fat and carbohydrate to ATP, the only energy source that can be used directly by the cell to support contraction. Ultimately, glucose and fat molecules (and certain amino acids) break down and combine with oxygen to form ATP, carbon dioxide, water, and heat energy. This occurs via enzymatic processes occurring first in the cytosol and then the mitochondria. The carbon dioxide and excess water leave the body through our breath. The ATP generated provides a usable energy source for muscle contraction and other cell functions. Heat removal occurs by sweating and as radiant heat transfer from the skin to the surrounding air. Clearly, each by-product of energy metabolism has significance to the exercising athlete.

5. Capillaries - These microscopic size blood vessels are not actually part of the muscle cell. Instead, capillaries physically link the muscle with the cardiovascular system. Each muscle cell may have from 3 to as many as 8 capillaries directly in contact with it, depending on fiber-type and training. One square inch of muscle cross-section contains 125,000 to 250,000 capillaries! The volume of blood forced through the heart's aorta (about the diameter of a heavy duty garden hose) is spread so thin among the billions of capillaries that

red blood cells must squeeze through in single file like soldiers marching along a path. Distributing the blood flow through such an immense network of vessels is critical so every individual cell maintains a supply line and waste removal system. This and other “infrastructural challenges” are the price multi-celled organisms (we humans) pay for our complex organization. Endurance exercise increases the demands on nutrient supply and waste removal, but also stimulates the growth of more capillaries. Endurance training improves the delivery and removal function of this fantastic network of vessels. The total number of capillaries per muscle in endurance-trained athletes is about 40% higher than in untrained persons. Interestingly, this is about the same as the difference in VO2 max between well-trained and untrained people. In contrast, strength training tends to decrease the capillary to muscle fiber diameter ratio. This occurs because muscle fibers grow in diameter, but the number of capillaries essentially remains unaltered.

The Motor Unit

A motor unit is the name given to a single alpha motor neuron and all the muscle fibers it activates (neurophysiologists use the term innervates). With 250 million skeletal muscle fibers in the body (give or take a few million), and about 420,000 motor neurons, the average motor neuron branches out to stimulate about 600 muscle fibers. Interestingly, large muscles may have as many as 2000 fibers per motor unit, while the tiny eye muscles may have only 10 or so fibers per motor unit. The size of a motor unit varies considerably according to the muscle’s function. Muscles with high force demands but low fine control demands (like a quadriceps muscle) are organized into larger motor units. Muscles controlling high precision movements like those required in the fingers or the eyes are organized into smaller motor units. The motor neuron branches into many terminals, and each terminal innervates a specific muscle fiber. The motor unit is the brain’s smallest functional unit of force development control; if a motor unit comprising 600 muscle fibers in the left biceps is stimulated, then all 600 of those fibers will contract simultaneously and contribute to the total force produced by the biceps. The brain cannot stimulate individual fibers one at a time. Even for our sophisticated nervous system, that would require far too much wiring.

Regulation of Muscular Force

The brain combines two control mechanisms to regulate the force a single muscle produces. The first is **RECRUITMENT**. The motor units that make up a

muscle are not recruited in a random fashion. Motor units are recruited according to the *Size Principle*. Smaller motor units (fewer muscle fibers) have a small motor neuron and a low threshold for activation. These units are recruited first. As more force is demanded by an activity, progressively larger motor units are recruited. This has great functional significance. When requirements for force are low, but control demands are high (writing, playing the piano) the ability to recruit only a few muscle fibers gives the possibility of fine control. As more force is needed the impact of each new motor unit on total force production becomes greater. It is also important to know that the smaller motor units are generally slow units, while the larger motor units are composed of fast twitch fibers.

The second method of force regulation is called **RATE CODING**. Within a given motor unit there is a range of firing frequencies. Slow units operate at a lower frequency range than faster units. Within that range, the force generated by a motor unit increases with increasing firing frequency. If an action potential reaches a muscle fiber before it has completely relaxed from a previous impulse, then force summation will occur. By this method, firing frequency affects muscular force generated by each motor unit.

Firing Pattern

If we try and relate firing pattern to exercise intensity, we see this pattern. At low exercise intensities, like walking or slow running, slow twitch fibers are selectively utilized because they have the lowest threshold for recruitment. If we suddenly increase the pace to a sprint, the larger fast units will be recruited. In general, as the intensity of exercise increases in any muscle, the contribution of the fast fibers will increase.

For the muscle, intensity translates to force per contraction and contraction frequency/minute. Motor unit recruitment is regulated by required force. In the unfatigued muscle, a sufficient number of motor units will be recruited to supply the desired force. Initially desired force may be accomplished with little or no involvement of fast motor units. However, as slow units become fatigued and fail to produce force, fast units will be recruited as the brain attempts to maintain desired force production by recruiting more motor units. Consequently, the same force production in fatigued muscle will require a greater number of motor units. This additional recruitment brings in fast, fatiguable motor units. Consequently, fatigue will be accelerated toward the end of long or severe bouts due to the increased lactate produced by the late recruitment of fast units.

Specific athletic groups may differ in the control of the motor units. Top athletes in the explosive sports like Olympic weightlifting or the high jump appear to have the ability to recruit nearly all of their motor units in a simultaneous or *synchronous* fashion. In contrast, the firing pattern of endurance athletes becomes more *asynchronous*. During continuous contractions, some units are firing while others recover, providing a built in recovery period. Inital gains in strength associated with a weight training program are due to improved recruitment, not muscle hypertrophy.

b) Training Adaptations in Skeletal Muscle

Introduction

Adaptability is a fundamental characteristic of skeletal muscle (and the body in general). The nature of this adaptation can be summarized using the following principle: cells will adapt in a manner that tends to minimize any movement away from **homeostasis**, or resting conditions. In exercise physiology we refer to the acute changes that occur in a sytem, organ, or cell during exercise as **responses**. An example is the increase in heart rate that occurs when we jump up from our chair and start jogging. The long-term changes that occur as a result of repeated bouts of exercise are called **adaptations**. Cellular adaptations generally involve an increase or decrease in the rate of synthesis of a specific cellular protein. All muscle cells are in a constant state of synthesis and degradation. If synthesis rate exceeds degradation rate, an increase in the cellular component occurs. A change in protein synthesis requires a cellular signal. Biologists and physiologists continue to explore the communiction process by which different forms of muscular work induce cellular changes. At the cellular level, there are some theories, but no complete understanding. However, we do know quite a bit about **what** adaptations do occur, even if all the details regarding **how** remain unclear just yet.

Contrast Between Maximal Strength and Maximal Endurance

If we could build a skeletal muscle for the purpose of endurance, what would the recipe be? Since the heart is the supreme endurance muscle, let's cheat by taking a look at it first.

Characteristics of Fatigue Resistant Muscle Cells:

• Heart cells are **smaller** in diameter than skeletal muscle cells. This results in very short diffusion distance between oxygen molecules coming from capillaries and the mitochondria where they are used.

• The surrounding network of capillaries is extremely well developed. This characteristic also facilitates even and rapid oxygen distribution to all myocardial cells.

• The mitochondrial density of heart cells is extremely high, 20-25% of cell volume in adults. Mitochondria use oxygen to metabolise carbohydrate and fat and produce ATP.

• The cytoplasmic enzymes responsible for breaking down fatty acid molecules into 2 carbon fragments that can enter the mitochondria are present in high concentrations.

• Contractile protein makes up about 60% of cell volume. The ATPase subtype found in heart is slower than that seen in skeletal muscle. Consequently, the rate of force development is slower, although absolute tension/cell diameter is the same.

• Heart lactate dehydrogenase, the enzyme that converts pyruvate to lactic acid competes poorly with pyruvate dehydrogenase. This contributes to the very low lactate production in heart cells despite high metabolic flux. So, heart cells display almost zero fatiguability due to the tremendous capacity they have to receive and consume oxygen. Fatigue resistance is traded for anaerobic capacity. This is why the heart has little tolerance for oxygen deprivation, the dreaded heart attack. If we want to build a skeletal muscle that is highly fatigue resistant, it must resemble heart muscle in its basic features.

Now let's build a muscle that is optimized for brief efforts and maximum force production. Here are the characteristics needed.

Characteristics of Maximal Strength Muscle Cells:

o Each muscle cell should contain a high volume of contractile protein. Since oxygen diffusion is not a concern, making the cell diameter larger will help it hold more contractile protein (actin and myosin).

o To make more room for actin and myosin, mitochondrial density should be minimized to that necessary to maintain resting cell function.

o Since fat can only be metabolized aerobically, high levels of fat- cleaving enzymes in the cytosol are also unnecessary.

o The capacity for anaerobic glycolysis should be high to allow brief but high capacity energy production without oxygen. The capacity for lactic acid production should be high.

What you should notice is that these two lists are exactly opposite. The optimal muscle for endurance CAN NEVER be maximally strong or powerful. And the muscle fiber that produces the most force CANNOT be optimally developed for endurance as well. The two conditions are mutually exclusive. This is one of the most important concepts to understand when designing a training program.

Three Points to Remember:

o There are identifiable proteins in the muscle that contribute to its ability to produce high force at high rates (strength and power respectively).

o There are also identifiable proteins and structural characteristics that confer high fatigue resistance (endurance).

o There is no identifiable specific protein or structure that confers the quality "Strength-Endurance". When we train for strength-endurance, what we are really doing is training in a way that fails to stimulate either strength or endurance adaptations optimally. An example of this "best of neither worlds" approach is circuit training.

As a coach / athlete, your sucess begins with your ability to accurately understand the muscular demands of your sport. Then, a training program can be designed that will result in muscular development suited to the combination of strength and endurance that your sport requires. Here are two real world examples.

Strength and Endurance in the Proper Dose

Example Number One:

In 1994, I joined three other rowers in Austin, Texas to train for the Masters National Championships in the 4x. One of my teammates was a fellow named Jason. Then 29, he is a computer engineer, former varsity lightweight sweep rower at Cornell University, turned sculler. Now several years out of college, Jason was no longer a lightweight. He carried 190 pounds of muscle on his frame, and little fat. Jason had been training on his own for several years. His routine was demanding. He did a lot of weight training in the gym. On the water 6 days a week in his 1x, he almost always performed intervals. These usually consisted of 1 to 4 minutes race pace intervals with lengthy recovery between. Both the volume (total number of intervals), and intensity of rowing training were quite high. This training program had generated an athlete with excellent anaerobic capacity, and excellent speed through 500 meters of a rowing race. Unfortunately, Masters rowing races are 1000 meters and the open events are 2000! At the Masters that year, Jason showed two weaknesses that were a direct result of his training. He began to bog down over the last 200 meters, and his overall performance deteriorated over the course of 4 days of heats and finals. He was fast enough to reach three finals in 1x, 2x, and 4x competition, but left with only a silver in the 4x to show for it.

He (and we) wanted more. Each of us had specific work to do for 95. As the exercise physiologist on the team, I made a few suggestions. Jason had to put away the barbells and go back to the basics of endurance training. He had emphasized strength training too much. Despite training with great intensity, He had lost much of the aerobic capacity he had as a lightweight in college. He had no aerobic base!

The weight training volume was reduced. Almost all interval training was eliminated from his training for 6 months and replaced by long, steady state rows of 60 to 90 minutes, and road cycling. After 6 months, interval training was re-introduced, but the interval length was extended to 4, 8, or 20 minutes depending on the specific workout. Steady state training remained a major component of the total training volume. Intensity was monitored with heart rate. Only in the final month before the competition were short (500) meter, supra-race pace intervals included in the training program.

The anaecdotal results observed in the final months of training were a slightly less muscular athlete who retained his 500 meter speed, but now was difficult to hold off in a 20 minute interval as well. The hard (metallic) evidence was even more obvious: Three gold medals at the 1995 Masters Nationals (4x, Mx 8+, and 8+). Two finals were held within one hour of each other, with the second being the all important 4x final we had lost in 94. A 0.7 sec loss became a 2.5 sec victory in the 3 minute race. Recovery was never a problem, and after 500 meters the race was never in doubt. Of course, Jason wasn't the only guy in the boat. But, we all followed the same training program.

Take Home Message: Even for races that only last 3 minutes, endurance capacity is critical! Too much emphasis on strength training results in a sub-optimal muscular system for the competitive task.

Example Number Two:

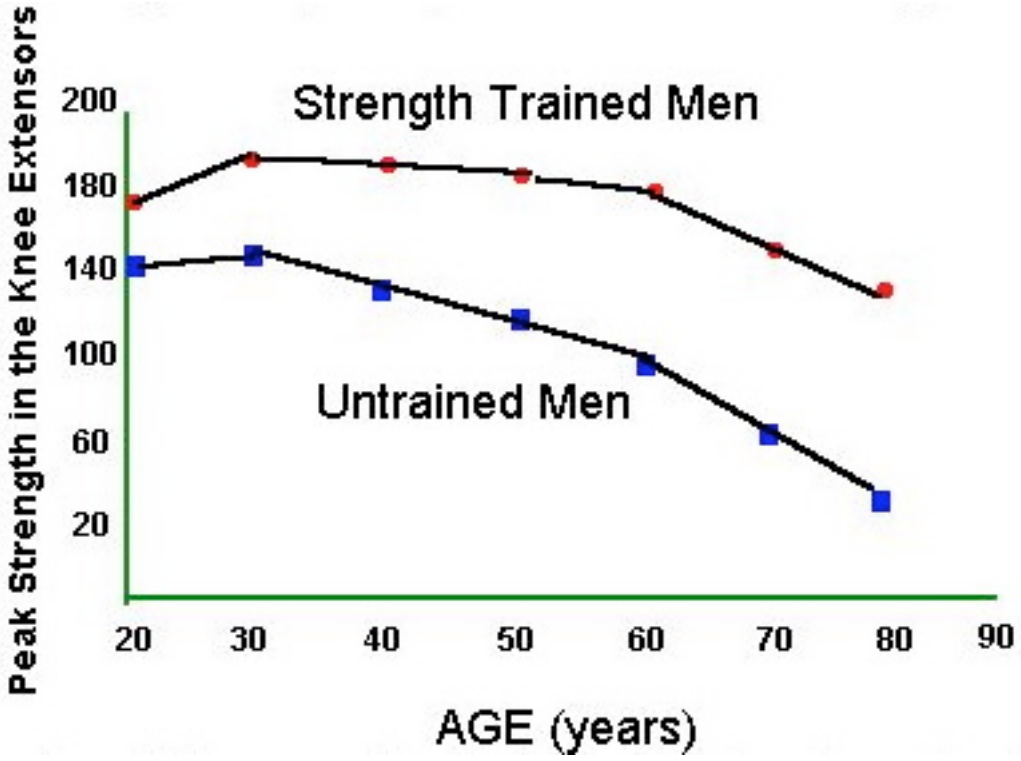
In my second example, I will use someone even closer to home, my wife. Hilde is a natural endurance athlete. She has competed in X-country skiing, cycling, and triathlon races. In general, the longer the distance, the stronger she becomes. However, when I met her, she complained of painful hips that made running more than 40 minutes difficult. She had undergone physical therapy and employed all manner of flexibility training to relieve the apparent immobility. After watching her run, I could see she was very weak in her hip extensors. Sadly, all her physical therapy had never included appropriate strength training!

I started her with lunges, then eventually progressed to 1 legged squats from atop a 3 foot bench. Initially, she did not have sufficient strength to even approach a true parallel, 1 legged squat. But she grimaced and persisted. Now, 5 months later, her extensor strength is much improved. The payoff has been obvious in her hill climbing. She runs from her hips, instead of from her knees. She has also reported improved strength on the skies. In Hilde's case, a specific strength training program was added to an endurance regime, and the results were very positive. Her total strength training investment (including upper body and back extension exercises) is two, 30 minute sessions per week.

c) Aging Effects on Skeletal Muscle

Changes in Muscular Strength

It is well documented that a person's maximal strength decreases with increasing age. Is this due to an unavoidable effect of aging or the typical decrease in physical activity that often accompanies getting older? The answer appears to be BOTH.



From the figure above, it is apparent that strength training remains highly effective in maintaining muscular strength throughout life. However, after about age 60, strength levels fall more rapidly, independent of training. This is probably influenced by marked changes in the hormonal mileau. Both testosterone and growth hormone appear to decline more dramatically after about age 60. Reduction in the circulating concentration of these hormones will result in a shift in the balance between muscle protein synthesis (anabolism) and protein breakdown (catabolism). The decreased strength is due to atrophy of muscle fibers. It is important to notice that with strength training, the maximal strength of a 60 year old can exceed that of his untrained sons! And, several studies have demonstrated that strength gains are possible even at 90 years old. So it is never too late to begin a strength training program!

Fiber Type and Aging

There have been conflicting reports and myths developed regarding fiber type changes with aging. Cross-sectional studies of post-mortem bodies between age 15 and 83 have suggested that fiber type composition is unchanged throughout life. This is also supported by comparing muscle biopsy results of younger and older endurance athletes. In contrast, one longitudinal study of a group of runners examined in 1974 and again 1992, suggested that training could play a role in fiber distribution. Those athletes who continued training showed unchanged fiber composition. Those who stopped training appeared to have greater slow-twitch fiber percentage. This was primarily due to selective atrophy of the fast fibers. This is not difficult to explain since they are seldom recruited. There is also some evidence that the actual number of fast motor units decreases slightly with aging after age 50, about 10% per decade. The reasons or mechanisms for such a change are unclear. So, the net effect of aging for the endurance athlete is unchanged fiber composition or a slight *relative* increase in Slow fiber type due to selective Fast fiber loss. The Fast motor units **do not** become Slow motor Units.

Muscle Endurance Capacity and Aging

The good news for the endurance athlete is that there appears to be little change in skeletal muscle oxidative capacity with age, as long as training is maintained. The number of capillaries per unit area of muscle is the same in young and old endurance athletes. Oxidative enzyme levels are similar or slightly lower in older athletes. This small decrease is probably attributable to decreased training volume in the older athletes. Furthermore, it appears that the older individual who starts endurance training retains the potential to improve muscle enduranace capacity.

Summary

It appears that the Masters athlete who continues endurance training at high intensities and mantains a maintenance strength training program experiences few changes in skeletal muscle through age 50. After age 50, declines in the quantity, but not the quality of muscle occur. These declines are also deminished by continued training. In general the changes that occur diminish maximal strength and power more than endurance capacity. This helps to explain the tendency for older athletes to move toward longer events within their sports discipline.

d) Skeletal Muscle Fiber Type

Have you ever sat down for Thanksgiving dinner and found yourself wondering why turkeys have some dark meat and some white meat? Well, you were not the first. A scientist named Ranvier reported differences in muscle color within and among animal species back in 1873. The explanation for the color differences is pretty simple and has a basis in physiology. The dark meat of the turkey, or chicken, is "red" or slow-twitch muscle. The white meat is "white" or fast-twitch muscle. Most animals have some combination of these two fiber types, though the destinctions may be less obvious. Why are they differently colored? The slow muscles have more mitochondria (full of red pigmented cyctochrome complexes), and more myoglobin packed within the muscle cells. This gives them a darker, reddish color. Humans also have dark and white meat. Some of our muscles, like the soleus in the lower leg are almost all slow twitch fibers. Others such as those controlling eye movements are made up of only fast twitch fibers. Function dictates form in these highly specialized muscles. The majority of human muscles contain a mixture of both slow and fast fiber types. From an evolutionary standpoint this makes sense. Our not so very distant ancestors' daily survival sometimes dictated a long walk or jog in search of food. Other times, a fast sprint or jump may have kept one out of harm's way. The exact composition of each muscle is genetically determined. On average, we have about 50% slow and 50% fast fibers in most locomotory muscles, with substantial intra-individual (and muscle to muscle) variations. This variation helps make sports interesting!

Olympic Champions are Oddballs

If you want to win an Olympic medal in the 100 meter dash, you had better be born with about 80% fast twitch fibers! Want to win the Olympic marathon? Put in an order for 80% slow twitch fibers in your quads. The fast twitch fibers benefit the absolute sprinter because they reach peak tension much faster than their slow twitch counterparts. Gram for gram, the two types are **not different in the amount of force they produce**, **only their rate of force production.** So, having a lot of fast twitch fibers only makes a positive difference when the time available for force production is very limited (milliseconds), like the 100ms or so the foot is in contact with the ground during a sprint or long jump. It makes no difference to the powerlifter who may use 3-4 seconds to execute a slow, smooth lift.. In cycling, the only event that they are decidedly advantageous for is the match sprint, analogous to the track 100 meter dash, but with more anticipatory tactics and theatrics.

For the pure endurance athlete, more slow twitch fibers are advantageous. These fibers give up lightning contraction and relaxation velocity for fatigue resistance. Lots of mitochondria and more capillaries surrounding each fiber make them more adept at using oxygen to generate ATP without lactate accumulation and fuel repeated contractions, like the 240 or so in a 2000 meter rowing race, or the 15,000 plus in a marathon.

Does Fiber Type Change with Training?

This has been one of the 10,000 dollar questions in exercise physiology. It has been documented that elite endurance athletes possess a higher percentage of slow twitch fibers in the muscles they use in their sport, compared to untrained individuals. Is this due to genetic endowment or years of rigorous training? The answer is difficult to get at directly because we don't have comparative muscle biopsies ***(see subcategory-(i) below)*** of great athletes before and after they started training and excelling in their sport. However, good basic investigation using experimental models has helped generate some answers. The critical knowledge to remember is that fiber type is controlled by the motor nerve that innervates a fiber. Unless you change the nerve, you won't change fiber types from fast to slow or vice versa. Just this type of experiment has been performed in animals (generally rats). **So, remember, there is no compelling evidence to show that human skeletal muscle switches fiber types from "fast" to "slow" due to training.**

Then Why Am I Training So Hard?

Two reasons; first, skeletal muscles respond to chronic overload (training), by trying to minimize the cellular disturbance caused by the training. With intense endurance training, fast fiber types can develop more mitochondria and surrounding capillaries. So can the slow fibers. So training improves your existing fiber distribution's ability to cope with the exercise stress you create for it.

Second, even among a group of elite endurance athletes, fiber type alone is a poor predictor of performance. This is especially true in the intermediate duration events. There are many other factors that go into determining success! In fact, there is also evidence to suggest that a mixed fiber composition is ideal for success in an event like the mile run, or if good performances are to be possible in a range of events. More about Fiber type and training. **(*see subcategory-(ii) below*)**

i) The Muscle Biopsy

In humans, samples of muscle are extracted using a biopsy needle or bioptome. This is 3-piece instrument about the diameter and length of a pencil. The outside is a hollow, stainless steel cylinder with a point on the end and a small oval "window" cut out about 1 inch above the tip. Inside the cylinder is a circular blade attached to a long plunger or handle. Finally, inside that is a plunger to push the extracted piece of muscle out of the biopsy needle. They look like this:

An athlete is given a local anaesthetic like xylocaine, by injection, above the site to be biopsied. A small ~1cm long incision is then made through the numbed skin, fat and fascia with a regular surgical scalpel. The biopsy needle is then introduced into the muscle, about 3 inches deep. The athlete has been riding on a cycle ergometer and at some point is asked to lay back as quickly as possible for a biopsy in order to capture the cellular conditions in the muscle corresponding to the exercise situation as closely as possible. Since the preliminary incisions have already been made, if all goes well, the muscle sample will be extracted and frozen within 15 seconds of exercise cessation.

Since there are no sensory nerves within the muscle, this will only be detected as pressure. Well, that is true most of the time. I had 8 biopsies during my student days. On a couple of those I got a jolt when one or another nerve got a good tickle, but it was not really pain I felt, at least not the kind you would feel if something cut through your skin. When the needle is in the leg, a small portion of muscle will push inside the window of the barrel. Then the blade is passed

through the barrel, cutting off the small piece (about the size of a green pea, or smaller), which remains inside the barrel as it is extracted from the muscle. This small piece is quickly frozen in liquid nitrogen or pentane for later analysis.

In sports science, the principle value of the muscle biopsy has been to generate histological cross-sections for fiber type staining, and for the determination of muscle glycogen concentration. Because of the heterogeneity of fiber distribution within most muscles, fiber type determination from a single biopsy should be considered only a reasonable estimate.

I sometimes receive emails from athletes enquiring where they might have biopsies performed to determine their fiber type percentage. My answer is that this is not a normal type of athlete testing offered by performance testing facilities, or even elite training centers. There really is no value to the athlete in knowing his/her fiber type distribution, unless it is to use as an excuse for performances that do not meet expectations. You aren't going to learn anything that will guide you to train differently for your chosen sport. Muscle biopsies, when performed on healthy athletes, are almost always done in the interest of some physiological research project.

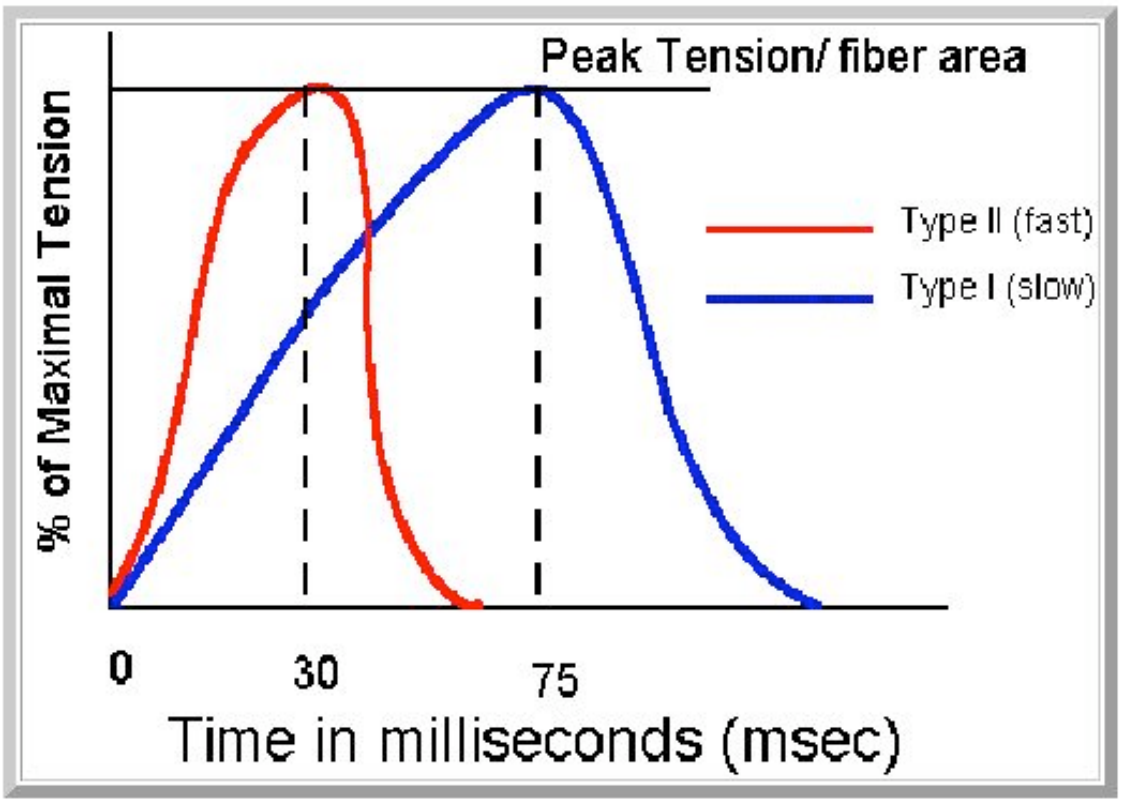
ii) Skeletal Muscle Fiber Type - Part Two

Like most things, there is the simple story, and the real story. Physiological investigations in the late 60s and early 70s have done a great deal to shape our knowledge of skeletal muscle function and fiber type. The biopsy technique, enzyme histochemistry, and physiological studies all advanced this issue. From this work, we now know the fiber types differ: 1) in contractile speed, 2) in myosin ATPase enzyme characteristics, and 3) in metabolic enzyme profile. From these three differences, three different fiber type classification schemes have emerged.

Dr. Phil Gollnick and colleagues studied differences in contractile speed in different muscles. They found that the fiber types were distinguishable based on the time it took them to reach peak tension when stimulated. That difference is graphically demonstrated here.

Skeletal Muscle Fiber Type and Contractile Velocity

When a muscle contraction occurs, it is initiated by an electrical impulse originating in the brain. All of the fibers in one motor unit are activated by a single motor nerve. The two fiber types differ in the time it takes for the fiber to fully contract, or reach peak contractile tension. This difference is depicted graphically below. Notice that there is a big difference in the rate of both force development, and relaxation. The peak force attained is not different when measured per unit cross sectional area of the fiber. This is one of the critical concepts to be grasped in order to understand the impact of fiber type on different sports activities.



They proposed the distinction *slow*, and *fast*. This turned out to be an oversimplification. Meanwhile, even before this study, Brooke and Engle distinguished the fiber types based on differences in Myosin ATPase enzyme activity. They arbitrarily divided the muscles into two groups and called them *Type I* and *Type II.*

Around the same time Gollnick and colleagues were classifying muscles based on contractile speed, Dr. J.B. Peter and his group investigated the properties of the two categories of fibers established by Brooke and Engle. They proposed another set of terminology created by combining tension generating and metabolic properties. Type I cells were termed *Slow Oxidative*. That was

simple. The slow fibers had a lot of mitochondria (containing oxidative enzymes) and capillaries. However the *Type II* or *Fast fibers* had to be further divided into two sub-categories. Type II cells were either *Fast Glycolytic (FG)* or *Fast Oxidative Glycolytic (FOG).* The FG fibers stored lots of glycogen and had high levels of enzymes necessary for producing energy without oxygen, but contained few mitochondria. The FOG fibers had the best of both worlds, high speed and glycolytic capacity, plus high levels of oxidative enzymes. These INTERMEDIATE fibers were termed type *II*A fibers by a fourth research group (Brooke & Kaiser, 1970). The pure fast fibers (FG) were termed Type *IIb*. This last lingo system seems to have stuck within the physiological research community.

For the athletic community, the important information is this. It does appear that pure fast (Type IIb) fibers can transition to "hybrid" (Type IIa) fibers with chronic endurance training. Biopsies of elite endurance athletes reveal that after years of training, they have almost no IIb fibers, but often have a significant percentage of the intermediate, IIa fibers. BUT, the majority of the available research suggests that Type IIa fibers do not transition to Type I. This is the more accurate way of saying what I said at the end of Part I of the Fiber type discussion.

	TYPE of FIBER		
Characteristic	**Slow Oxidative (I)**	**Fast Oxidative (IIa)**	**FastGlycolytic (IIb)**
Myosin ATPase activity	LOW	HIGH	HIGH
Speed of Contraction	SLOW	FAST	FAST
Fatigue Resistance	HIGH	Intermediate	LOW
Oxidative Capacity	HIGH	HIGH	LOW
Anaerobic Enzyme Content	LOW	Intermediate	HIGH
Mitochondria	MANY	MANY	FEW
Capillaries	MANY	MANY	FEW
Myoglobin Content	HIGH	HIGH	LOW
Color of Fiber	RED	RED	WHITE
Glycogen Content	LOW	Intermediate	HIGH
Myoglobin Content	HIGH	HIGH	LOW
Fiber Diameter	SMALL	Intermediate	LARGE

3) PUTTING THE PIECES TOGETHER

a) The Lactate Threshold

In exercise physiology, there have been few topics more frequently investigated, or more vigorously debated than the lactate threshold. It is the details, not the basics that create the big research problems. However, it is the basics that have great application to training and performance. So, we'll stick to those.

What is Lactic Acid and Where Does it Come From?

When you consume carbohydrate, it consists of several different sugar molecules; sucrose, fructose, glucose to name a few. However, by the time the liver does its job, all of this sugar is converted to glucose which can be taken up by all cells. Muscle fibers take up glucose and either use it immediately, or store it in the form of long glucose chains called glycogen. During exercise, glycogen is broken down to glucose which then goes through a sequence of enzymatic reactions that do not require oxygen to proceed. All of these reactions occur out in the cell fluid, or cytosol. They can occur very rapidly and yield some ATP in the process. This pathway is called the anaerobic (no oxygen) glycolysis (glucose breakdown) pathway. Every single glucose molecule must go through this sequence of reactions for useful energy to be withdrawn and converted to ATP, the energy molecule, that fuels muscle contraction, and all other cellular energy dependant functions.

The Metabolic Fork in the Road

There is a critical metabolic fork in the road at the end of this chemical pathway. At this fork, glucose has been converted from one 6 carbon molecule to two, 3 carbon molecules called pyruvic acid, or pyruvate. This pyruvate can either be shuttled into the mitochondria via the enzyme pyruvate dehydrogenase, or be converted to lactic acid via the enzyme lactate dehydrogenase. Entry into the mitochondria exposes the pyruvate to further enzymatic breakdown, oxidation, and a high ATP yield per glucose. Conversion to lactate means a temporary dead end in the energy yielding process, and the potential for contractile fatigue due to decreasing cellular pH if lactic acid accumulation proceeds unchecked. Like a leaf floating in a river, the pyruvate molecule has no "say" in which metabolic direction is taken.

Which Way will MY pyruvate go during exercise?

I am sure you have surmised that that is a critical question with big implications for performance. I will try to answer the question at three levels: a single muscle fiber, an entire muscle that is active during exercise, and the entire exercising body.

The Muscle Cell at Work

In a single contracting muscle fiber, the frequency and duration of contractions will determine ATP demand. ATP demand will be met by metabolizing a combination of two energy sources: fatty acids and glucose molecules (ignoring the small contribution of protein for now). As ATP demand increases, the rate of glucose flux through glycolytic pathway increases. Therefore at high workloads within the single fiber, the rate of pyruvic acid production will be very high. If the muscle fiber has a lot of mitochondria (and therefore more Pyruvate Dehydrogenase), pyruvate will tend to be converted to Acetyl CoA and move into the mitochondria, with relatively little lactate production. Additionally, fatty acid metabolism will account for a higher percentage of the ATP need. Fat metabolism does not produce lactate, ever! If lactate is produced from glucose breakdown, it will tend to diffuse from the area of high concentration inside the muscle cell to lower concentration out of the muscle fiber and into extracellular fluid, then into the capillaries.

The Whole Muscle at Work

Now let's look at an entire muscle, say the vastus lateralis of the quadriceps group during cycling. At a low workload, glycolytic flux is low and the pyruvate produced is primarily shuttled into the mitochondria for oxidative breakdown. Since the workload is low, primarily slow twitch fibers are active. These fibers have high mitochondrial volume. As workload increases, more fibers are recruited and recruited fibers have higher duty cycles. Now ATP demand has increased in the previously active fibers, resulting in higher rates of pyruvic acid production. A greater proportion of this now is converted to lactic acid rather than entering the mitochondria, due to competition between LDH and PDH. Meanwhile, some Fast twitch motor units are starting to be recruited. This will add to the lactate efflux from the muscle due to the lower mitochondrial volume of these fibers. The rate of lactate appearance in the blood stream increases.

The Body at Work

The vastus is just one of several muscles that are very active in cycling. With increasing intensity, increased muscle mass is called on to meet the force production requirements. All of these muscles are contributing more or less lactic acid to the extracellular space and blood volume, depending on their fiber type composition, training status and activity level. However, the body is not just producing lactate, but also consuming it. The heart, the liver, the kidneys and inactive muscles are all locations where lactic acid can be taken up from the blood and either converted back to pyruvic acid and metabolized in the mitochondria or used as a building block to resynthesize glucose (the liver). These sites have low intracellular lactate concentration, so lactic acid diffuses INTO these cells from the circulatory system. If the rate of uptake or dissappearance of lactate equals the rate of production or appearance in the blood, then blood lactate concentration stays constant (or nearly so). When the rate of lactate production exceeds the rate of disappearance, lactic acid accumulates in the blood volume, then we see the ONSET of BLOOD LACTATE ACCUMULATION (OBLA). This is the "Lactate Threshold" (LT).

Performance Implications

Lactic Acid production is not all bad. If we could not produce lactate, our ability to perform brief high intensity exercise would be almost eliminated. However, As I am sure you are aware, lactic acid is the demon of the endurance athlete. Cellular accumulation of the protons (increased acidity) that dissociate from lactate results in inhibition of muscle contraction. Blame those heavy legs on the protons! The bottom line is that exercise intensities above the OBLA point can only be sustained for a few minutes to perhaps one hour depending on how high the workload is above the intensity at OBLA. Exercise at or below this intensity may be sustainable for hours. The causes of fatigue at these sub-LT intensities include carbohydrate depletion and dehydration.

Factors that Influence the Rate of Lactate Accumulation in the body

- **Absolute Exercise Intensity** - for reasons mentioned above.

- **Training Status of Active Muscles** - Higher mitochondrial volume improves capacity for oxidative metabolism at high glycolytic flux rates. Additionally, improved fatty acid oxidation capacity results in decreased glucose utilization at submaximal exercise intensities. Fat metabolism proceeds via a different pathway than glucose, and lactic acid is not produced. High capillary density

improves both oxygen delivery to the mitochondria and washout of waste products from the active muscles.

• **Fiber Type Composition** - Slow twitch fibers produce less lactate at a given workload than fast twitch fibers, independent of training status.

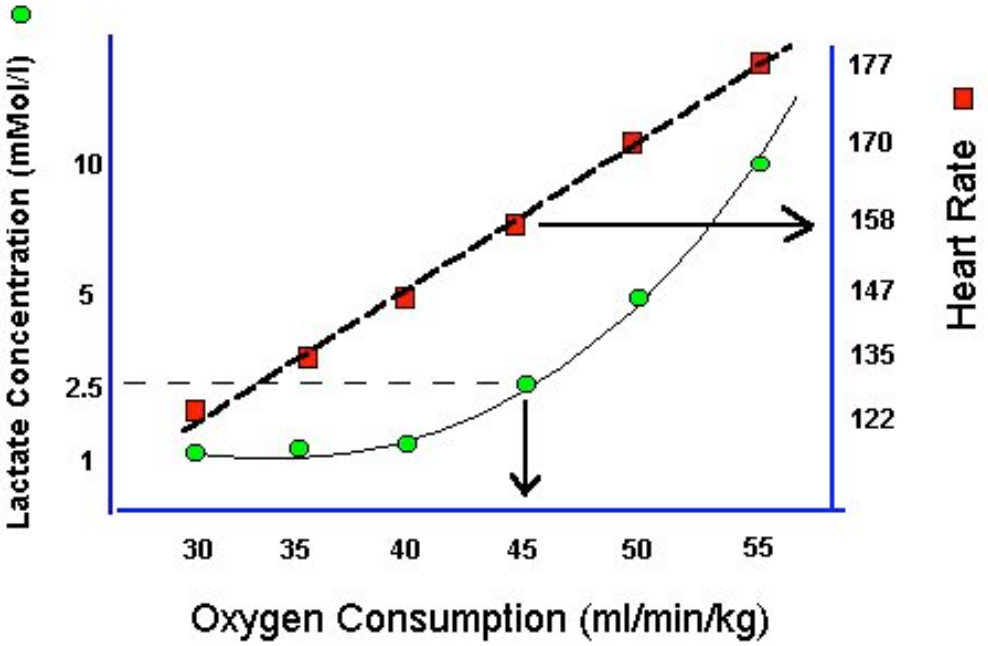
• **Distribution of Workload** - A large muscle mass working at a moderate intensity will develop less lactate than a small muscle mass working at a high intensity. For example, the rower must learn to effectively distribute force development among the muscles of the legs back and arms, rather than focusing all of the load on the legs, or the upper body.

• **Rate of Blood Lactate Clearance** - With training, blood flow to organs such as the liver and kidneys decreases less at any given exercise workload, due to decreased sympathetic stimulation. This results in increaed lactate removal from the circulatory sytem by these organs.

Measuring the Lactate Threshold

We have previously discussed the value of a high maximal oxygen consumption for the endurance athlete (***see subcategory-(1.a.v) below)***. A big VO2 max sets the ceiling for our sustainable work rate. It is a measure of the size of our performance engine. However, the Lactate Threshold greatly influences the actual percentage of that engine power that can be used continuously.

Most of you will never have this measured in a laboratory, but a brief description of a lactate threshold test is still useful, because it will lead us into some specific applications for your racing and training. The test consists of sucessive stages of exercise on a treadmill, bicycle ergometer, swimming flume, rowing machine etc. Initially the exercise intensity is about 50- 60% of the VO2 max. Each stage generally lasts about 5 minutes. Near the end of each stage, heart rate is recorded, oxygen consumption is measured, and a sample of blood is withdrawn, using a needle prick of the finger or earlobe. Using special instrumentation, blood lactate concentration can be determined during the test. After these measurements, the workload is increased and the steps repeated. Through a 6 stage test, we would expect to achieve a distribution of intensities that are below, at , and above the intensity of OBLA or the lactate threshold. The data from a test would generally look simililar to the example below.



Interpreting the Data

For purposes of interpretation, let's say that the athlete above had a maximal heart rate of 182, and a VO2 max or 61 ml/min/kg. These were also determined using a bicycle test. So they are good values for comparison. Looking at the green dots, we see that blood lactate concentration does not begins to increase until during the 4th workload, from a concentration of about 1 mM to 2.5 mM. This is the break point. The subject's VO2 was 45 ml/min/kg at this point. So we determine that his LT occurs at 45/61 or about 74% of VO2 max. If we look at the heart rate at this point, it is 158. Now we have a heart rate at lactate threshold. 158 = about 85% of his max heart rate. This is useful for the athlete. When he is cycling, he can judge his training intensities based on this important value. If he is a time trialist, this would approximate his racing heart rate for the hour long event.

So, Do I Race at My LT Intensity?

This depends on your race duration. If you are rowing 2000 meters, running a 5k race etc, your exercise intensity will be well above the AT. Consequently, the blood lactate measured after these events is extremely high in elite athletes, on the order of 15mM (resting levels are below 1 mM). In races

lasting from 30 minutes to 1 hour, well trained athletes also perform at an intensity above LT, but by a smaller margin. It appears that in these events, top performers achieve what might be termed a **"maximal lactate steady state"**. Blood lactate may increase to 8 to 10 mM within minutes, and then stabilize for the race duration. A high but stable lactate concentration may seem to contradict the idea of the LT. But, remember that blood lactate concentration is the consequence of both production and clearance. It seems likely that at these higher lactate concentrations, uptake by non-working muscles is optimized. At any rate, measurements in cyclists, runners and skiers demonstrate the fact that elite performers can sustain work levels substantially above the LT for up to one hour.

Specificity of the Lactate Threshold

It is important to know that the lactate threshold is highly specific to the exercise task. So if this cyclist tries to get on his brand new, previously unused, rowing machine and row at a heart rate of 158, he will quickly become fatigued. Rowing employs different muscles and neuromuscular patterns. Since these muscles are less trained, the cyclist's rowing LT will be considerably lower. This specificity is an important concept to understand when using heart rate as a guide in "cross training activities", as well as for the multi-event athlete.

Effect of Training

For reasons mentioned above, training results in a decrease in lactate production at any given exercise intensity. Untrained individuals usually reach the LT at about 60% of VO2 max. With training, LT can increase from 60% to above 70% or even higher. Elite endurance athletes and top masters athletes typically have LTs at or above 80% of VO2 max. Values approaching 90% have been reported. The lactate threshold is both responsive to training and influenced by genetics.

b) Efficiency, Economy and Endurance Performance

So far, if you read the two previous articles regarding "The System", you know that high level endurance performance depends on 1) a high maximal oxygen consumption, or VO2 max, and 2) a high lactate threshold, or point of OBLA. Your VO2 max sets the upper limit for your sustainable work potential. For the elite endurance athlete, a high VO2 max is like the invitation to the big dance.

Having an invitation to the dance does not ensure you will dance with the prettiest girl. But, not having one ensures you won't! The lactate threshold tells us something about how much of the cardiovascular capacity you can take advantage of in a sustained effort. It is determined by skeletal muscle characteristics and training adaptations. Multiplying VO2 max x LT (Oxygen Consumption at Lactate Threshold) gives us a measure of the effective size of your endurance engine. Now we come to efficiency. What does efficiency have to do with endurance performance? Victory goes to the person with the biggest endurance engine right? Well, let's use a racecar analogy.

If I build a powerful, well-tuned engine that can run at redline RPMs for hours, and then drop it into a Ford truck chassis, the truck might go 120 mph. But if I drop it into a streamlined Ferrari chassis, I might hit 200 mph (in theory, personally I am afraid I would soil my pants and hit the brakes long before I reached 200 mph). That is a big difference. Engine performance didn't change, but performance velocity did. To some extent, the same efficiency effect is observed in every endurance sport. Efficiency is critical to maximizing performance velocity!

Physiological Efficiency Defined

In an exercise setting, efficiency is defined as the percentage of energy expended by the body that is converted to mechnical work (another form of energy).

Work Efficiency = Mechanical work / Chemical energy expended

We can measure the mechanical work performed using an ergometer, like a bicycle ergometer, or rowing machine. We can measure the energy expended by the body indirectly via its oxygen consumption at sub maximal workloads. With some basic biochemistry we can convert the oxygen consumption we measure during exercise to a standard measure of energy like **kJoules, or Calories.** And, we can do the same for the work we measure on the ergometer. Work/time = power. Power is measured in watts and is a measure of the intensity of work. Intensity (watts) x exercise duration (minutes) gives **us total work, again measured in kJoules or Calories.**

If we take a group of cyclists, or a group of rowers and perform sub maximal testing on them to determine how much energy they consume when performing a standard sub maximal workload, we find that overall work efficiency will range between about 17 and 26%, with an average somewhere

in the middle of that range. In other words for every 100 Calories of energy burned, we manage to convert 20 Calories of that energy to useful work on the pedals of the ergometer, or as pulling power on the rowing machine. Now, if your goal is to lose body fat during exercise, then I suppose it pays to be inefficient, since it is Calories burned that matter. However, if your goal is to move your body faster than the other guy, than being 25% efficient is way better than 18%! So, what are the sources of inefficiency and what, if anything can we do about them?

Sources of Inefficiency in the Performance Machine

Let's use a 40 km time-trial in cycling as an example. The goal is to propel your body on a bicycle over land, and through air at the fastest sustained speed. So where do the energy losses occur in the path from chemical energy stored in pasta to velocity sustained on the bicycle over a 40km distance?

1. Chemical energy conversion losses - Your body must generate ATP for muscle contraction by chemically converting food energy, using a process that ultimately requires oxygen (hence the need for a big oxygen delivery capacity), while minimizing the production of lactic acid (high LT). All of the chemical energy in food is not transferred to ATP. About 60% is lost as heat energy. This is why you get hot during exercise. This source of inefficiency is the same in everyone.

2. Fiber type differences in converting ATP energy to contraction force - The next source of energy loss is in the step in which the chemical energy trapped within the ATP molecule is converted to mechanical energy via muscle contraction. There is some recent data from one laboratory in the United States suggesting that fiber composition of the muscle influences efficiency of muscle contraction (Coyle et al., Medicine and Science in Sports and Exercise. 24:782-788, 1992). Cyclists with a high percentage of slow twitch fibers appear MORE efficient. This was observed as a smaller increase in oxygen consumption for a given increase in cycling power output, in a group of cyclists whose fiber composition varied between 35 and 76% slow twitch. The higher efficiency was also observed when performing repetitive leg extensions, suggesting the source of efficiency was in the muscle, not the riding technique. The way they get at this is to measure the DELTA efficiency which is just the change in energy demand for a given change in power. By measuring the change in oxygen consumption an athlete requires to increase his work rate from say 150 to 200 watts, while keeping his cadence and body position the same, this specific efficiency of the muscles can be determined. The

differences are small. Perhaps a cyclist with 80% slow twitch fibers would have a **Delta efficiency** of 25%. A person with only 50% slow twitch fibers might be 21% efficient. So within the endurance community, the numerical difference in muscular efficiency seems small, but the impact on power output in a 40km time trial can be 8-10% independent of other variables. For example, data from Horowitz et al. (Int. J. Sports Medicine, 15:152-157, 1993) compared two groups of seven cyclists. The average VO2 maintained during 1 hour of cycling (a function of VO2 max and Lactate threshold) was the same in the two groups (4.48 vs. 4.46 l/min). However, the group with the higher average % slow twitch fibers (73 vs 48) achieved higher power output during the hour of cycling at voluntary maximal power (342 watts vs 315). They achieved 8% higher power output for the same physiological cost. How does this translate to velocity on a bike that actually moves?

3. The energy cost of moving the limbs - If you sit on a bicycle ergometer with the load set at zero and pedal at 80 rpms, you will discover that even though you are not doing any measurable mechanical work, your are still WORKING. It costs energy to just move your limbs, support your body, hold your balance, etc. The same is of course true for ANY movement, like running or skiing, or rowing. When this “unloaded cost of movement” is included in our measure of the mechanical work to energy expenditure ratio, then we get the GROSS Efficiency. Here, the word “gross” means “overall”, not “icky”.

One factor that impacts gross efficiency is movement frequency. That can be cycling cadence, or rowing stroke rate, or stride frequency in XC skiing. Higher cadences tend to cost more energy in general. And heavier limbs have been shown to be less efficient to move. However, there is a balance such that trained athletes tend to zero in on an optimal cadence for their body type and anatomy. When they are pushed away from that cadence, they use more energy to do the same work. Therefore, it is important to realize that the ideal movement frequency is not a universal, but varies from individual to individual. So, you should not try automatically to mimic your training partner’s cadence if they are much taller or shorter, or more or less muscular than you.

Having said that, in sports like rowing and cross-country skiing, there is a general tendency that the best athletes with the big engines use it by pulling or pushing harder each stroke, not revving up their movement frequency. This makes sense. Pushing harder each stroke means that more of the total energy goes to propelling the body and less to moving the limbs back and forth. And, you have no doubt seen the truth of this in watching how smoothly the great rowers, or skiers, or runners generate speed. Their technique looks

controlled and powerful, not frantic or hurried. There is a fine balance though. If you try to work with huge powers at too low frequencies, then the muscles become overloaded, blood flow gets compromised and fatigue results. So, the endurance athlete seeks a balance between the efficiency of lower movement frequencies and the decreased muscle tension and blood vessel compression of higher frequencies for a given workload. Exactly where this balance point lies varies from athlete to athlete.

In sports that are very technique intensive, like cycling, XC skiing, or rowing, there is much to be gained in perfecting the biomechanics of the movement. However, in cycling, with its relatively basic movement pattern, there does not seem to be any difference in overall cycling efficiency between elite cyclists and cyclists that have not been training very long. Sure, efficiency is an advantage, but the research indicates that cycling efficiency does not get better and better with years of training.

Efficiency vs Economy

The difference between efficiency and economy in an exercise setting is that **economy is measured as movement velocity for a given energy consumption**, while **efficiency is mechanical power output for a given energy consumption**. When we measure economy we connect the power produced to the movement intended, like cycling as fast as possible over 40km.

This brings us to the Truck vs. Ferrari analogy. Having the biggest engine doesn't guarantee the fastest performance in car racing or bicycle racing (or rowing, running, and swimming). The Ferrari goes faster because it is lighter and slices very cleanly through the air, reducing aerodynamic drag. So does the cyclist who perfects an aerodynamic riding position. (***See discussion of Cycling Aerodynamics from expert Jim Martin in separate article***). The best distance runners display high running economy. This means that they can run at a given speed with less oxygen demand. A high economy can make up for a relatively lower VO2 max. For example, Derek Clayton ran an incredible 2:08 marathon in 1969. His VO2 max was "only" 69 ml/min/kg (well it was probably a bit higher than that, but this was data from one non-peak season test). Thanks to his high running economy, that time stood for 12 years and was not matched by talented runners such as Craig Virgin, Gary Tuttle, and Bill Rodgers, whose VO2 max values ranged from 78 to 82 ml/min/kg! In rowing, both the hydrodynamics of the racing shell and the technical mastery of the rower contribute to rowing economy. However, even on a stationary

ergometer, elite rowers are more efficient than well-trained but non-elite oarsman. This is not due to a difference in fiber composition. So, it appears that subtle changes in rowing technique can continue to contribute to improve rowing efficiency and performance with additional years of training.

In no sport is efficiency more important than in swimming. The best swimmers in the world do not stand out in physiological tests of raw endurance capacity when compared to other endurance athletes. This suggests that high efficiency, achieved through a combination of ideal anatomical structure and technical perfection of the stroke is critical.

The Big Picture - Going back to the performance model, I identified several anatomical and cellular characteristics that contribute to 1) maximal oxygen consumption, 2) relative work intensity at lactic acid threshold, and 3) efficiency of transfer of physiological work to movement velocity. The details differ with each sports discipline and the event duration. But these are the BIG THREE variables. Maximal oxygen consumption is limited by central cardiovascular function, but also dependent on the peripheral adaptations that occur in the trained muscles. A high lactate threshold is due to peripheral adaptations improving the muscle's ability to generate energy aerobically. And, a high efficiency/economy creates the link between the physiological engine and the actual performance goal, to maximize average velocity.

c) The Brain - Body Link and Adaptation to Training

Because most exercise physiologists focus our training on muscle and systemic physiology, we tend to treat the brain as a mysterious black box, but that is changing. I for one, am recognizing that I must learn more about the impact of exercise on the brain, and the impact of brain activity on physical function. The MIND-BODY link is becoming more than just mysticism.

Here I will try to present some material that comes from the 25 years of work by Dr. Heinz Liesen. He served as National Team Doctor for the German soccer (football) team that reached the WorldCup final in 86 and 90, despite only modest talent. He also served as team doctor for the very successful national Field Hockey team and the Nordic Combined (cross country skiing plus ski jumping) team. Today his focus has returned to preventive medicine. The knowledge that he derives from folllowing specific athletes (and non-athlete exercisers) for several years, with repeated measures of cellular immune reactivity, training pattern, performance, and even regionalized brain

wave activity, is unique in the field. Some of the material also is based on a growing body of research generated in the United States and in Germany.

The Brain is the Center of Sports Performance

After all this talk about heart and muscles, this must sound pretty crazy. It is true though. The brain both initiates all of our voluntary movements and reacts to the stress that exercise creates. And, to some extent, stress seems to be a universal quality. The brain responds to the stress of job, driving, training, competition. The measurable impact of this stress is revealed in several ways:

• **Resting Catecholamine Levels.** Appropriate training tends to produce parasympathetic (rest and recover) dominance in the endurance trained athlete. However, if training stress becomes too severe, sympathetic (fight or flight) hormone levels remain elevated even at rest, an indication of incomplete recovery. An outward manifestation of this change is an elevated resting heart rate, although this may not be as sensitive as other measures. Another characteristic of the heart rhythm at rest is a certain degree of irregularity. There is considerable beat to beat variability in the heart rate, measured as minute changes in the interval between successive beats. This variability actually decreases in anticipation of a mental task, as sympathetic stimulation increases.

• **Testosterone / Cortisol Ratio.** Testosterone is an *anabolic* hormone that plays a role in regeneration and repair of muscle and tissue. Cortisol is a catabolic hormone that stimulates tissue breakdown. For example, cortisol levels are elevated in starvation situations when muscle tissue is catabolized for energy. Testosterone levels tend to be higher in individuals with a high capacity for hard training and recovery (and in those who receive it via a needle). Testosterone levels are naturally lower in women compared to men (about 10x lower). The testosterone/cortisol ratio has been shown in some (but not all) studies to be indicative of overtraining and staleness in elite athletes.

• **Immune System Function.** The immune system is a simple term for an intrinsic, adaptable cellular system that responds to invading foreign substances and eliminates them, or minimizes their capacity to replicate. The immune sytem is modifiable both in the rapidity and magnitude of its response to a foreign invader. Exercise creates both acute and chronic changes in immune system function. Acute exercise has been shown to cause a transient depression of certain components of the immune system, creating a window of susceptibility to infection of several hours after an exercise bout. The stress of

chronic exercise has a biphasic effect on immune function. This can be demonstrated several ways. First, the incidence of upper respiratory tract infections (URTI) **decreases** with moderate exercise, but **increases in hard training athletes** (J-shaped Incidence curve). Second, the magnitude of immune response to an applied antigen is decreased in overstressed athletes. Diagnostic kits are available which allow the controlled application of 7 allergens to the skin of the forearm. The total area of resulting skin reactions represent a quantitiative measure of the vigor of the immune system (within a given individual). This measurement is routinely made by many national teams in Germany and Scandinavia.

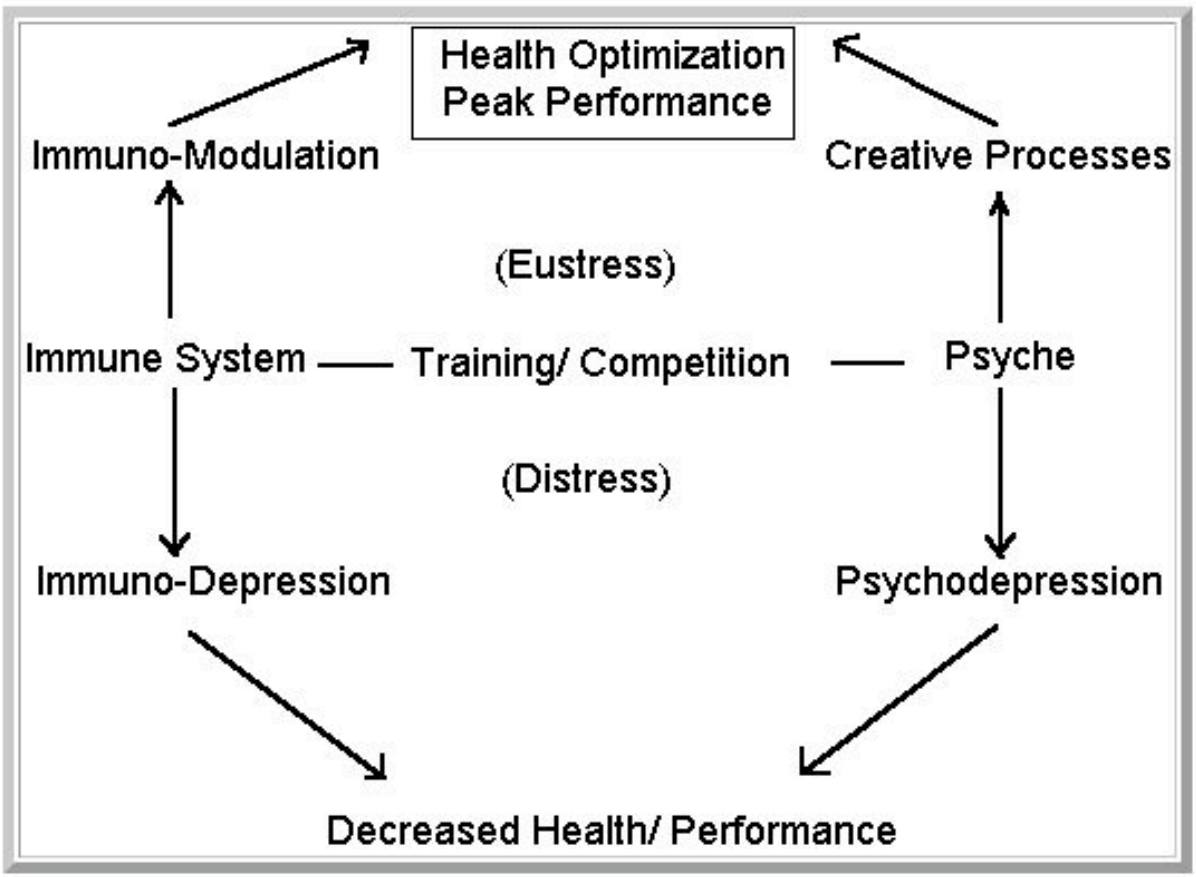
Perhaps the most interesting information I can give you is also the most difficult for me to understand with my miniscule knowledge of brain chemistry. It appears that the brain interacts with the immune system and modulates immune responses. This has been poignantly demonstrated by Dr. Liesen. By comparing blood withdrawn immediately before, and 1 hour after an unanticipated, but stress inducing medical diagnosis, he observed dramatic changes in the antigenic responsiveness of blood leukocytes. This brain modulation of immune function appears to involve the release of specific immuno modulating chemicals by the brain in response to emotional stimuli.

• **Psychological Profiles.** Several survey instruments have been developed which appear to be sensitive to emotional changes that accompany **or precede** the physiological and performance changes associated with an overtrained state. The instruments ask questions about current mood, anxiety, sleep quality, desire to train, etc.

The Big Picture

In today's world of elite sport, the real limitation to continued improvement has moved from the quantity of training to the capacity of the mind and body for restititution. Many elite athletes are training 50 weeks per year, sometimes 3-4 hours/day. When this extreme physical stress is combined with the stress of more frequent competitions to satisfy sponsors, media pressure and a tendency to lose time or interest in mentally diverting creative activities, the results are often disastrous. What we often see if we observe closely, is the sudden appearance of extremely talented performers, followed one or two years later by a decline in their performance or a complete dissappearance from the scene. Behind these early burnouts is usually a coach or performance team that is pushing too hard.

Below is a model presented by Dr. Liesen based on his experience and research, depicting the potential for both positive and negative effects of physical exercise on health and peak performance.



The success of the teams and individual athletes managed by Dr. Liesen was not due to an intensification of their training. On the contrary, more careful application of low intensity, "recuperative" training and even, complete rest days was the key. Keep in mind that complete days without training for world class endurance athletes are bitter pills to swallow. Here in Norway, World and Olympic Champion cross country skier, Bjorn Dahlie was recently quoted in the paper, "A day without training is a day without value". Three weeks later he had to withdraw from the National championships due to illness. Rest is important

How we rest is also important. For example, Dr. Liesen observed that the football (soccer) players tended to do nothing more than lie around and watch TV between training sessions, their mind assuming an almost vegetative state.

To increase their mental creativity, he took his team members to museums, helped them study new languages, started them doing hand-crafts, all during the heat of training and World Cup competition. The results were outstanding. Modestly talented German teams advanced to the World Cup final in 1986 and 1990 (running out of talent both times in the Final). They were successful in large measure because they stayed healthy and strong throughout the tournament.

When you look at the model above, you see both exercise level and creative mental activity as potential modulators of health and performance. When we build a training program, we have to consider the brain as well as the body.

The typical masters athlete does not train at the same volume as elite athletes. So, you might think, "overtraining is not an issue for me since I only train 12 hours per week." But, do you have a career, children, an hour of rush hour traffic each day. Has every training session become intense? Does each training ride/run become a race? Have other hobbies dissappeared from your routine. When you aren't training are you thinking about training? If you answered yes to most of these questions, you have to reevaluate your training program and your approach to exercise.

The Long Haul

In college and at the world class level, the clock is always ticking. Athletes feel pressure to reach their peak "this year". In many cases, this leads to yearly cycles that do not consider the ongoing development of the athlete "the next year". As a masters athlete, remember that you are in this game for the long haul. Training is a long process of learning and physical and technical growth. Medals go to those who combine talent with patience, and intensity with intelligence. Ultimately, no matter what your level of performance, the satisfaction of athleticism is sweetest when it enhances your life, not just your VO2 max!

4) AGING, EXERCISE AND SHORT TERM POWER
The effects of age and exercise on short term maximal performance: A model based on physiological systems.

by Jim Martin Ph.D University of Utah, Salt Lake City

Maximal exercise of short duration is most often associated with sport performance, but is also a factor in many daily activities. Climbing stairs, hurrying across a busy street, or carrying a heavy grocery bag may represent short term maximal efforts, and the ability to perform these types of activities can have a major influence on the independence of older adults, and may serve as an index of an individual's ability to live independently. This type of performance has rarely been studied in older adults, primarily due to safety concerns. Therefore, we are limited to examining short term maximal performance in the elderly by first determining the physiological systems that limit such performance in general, and then examining the effect of aging on each individual system. Since it is often difficult to separate the effects of aging from those of disuse, we will assume that aging means sedentary aging. Subsequently, we will consider the effects of exercise on these systems.

Among the few studies of maximal short term performance in older adults, Makrides et al., (1985) showed that maximal power output for 30 seconds decreased with age. However, they also showed that lean thigh volume was highly correlated ($r = 0.84$) with power. Therefore, it may be that power would not have shown a decrease with age, if lean thigh volume had been controlled for statistically. In a subsequent investigation, Makrides et al., (1990) showed that 12 weeks of high-intensity training produced greater increases in total work accomplished in 30 seconds in old (60-70 year old, 12.5%) than young (20-30 year old, 8%) subjects. One interpretation of this greater increase in work after training is that the older subjects were more detrained at the beginning of the study, due to the effects of sedentary lifestyle. Also, the fact that the older subjects adapted at least as well as the younger subjects serves to refute the claim that aging alone reduces short term performance, because these older subjects retained the ability to adapt to a training stimulus.

General model of short term maximal performance

Maximal performance of short duration (i.e. 30 - 120 sec) is often thought of as "anaerobic", or not relying on aerobic energy production. However, Medbo and Tabuta (1989) have shown that aerobic processes accounted for 40% of the energy produced in a 30 second maximal exercise bout, and 65% of the energy produced in a 2 minute maximal exercise bout. Therefore, the

physiological mechanisms of both aerobic and anaerobic energy production must be accounted for in order to properly model short term maximal performance. Additionally, the ability to recruit muscular activity of high intensity may be limited by central neural drive and neural signal transmission.

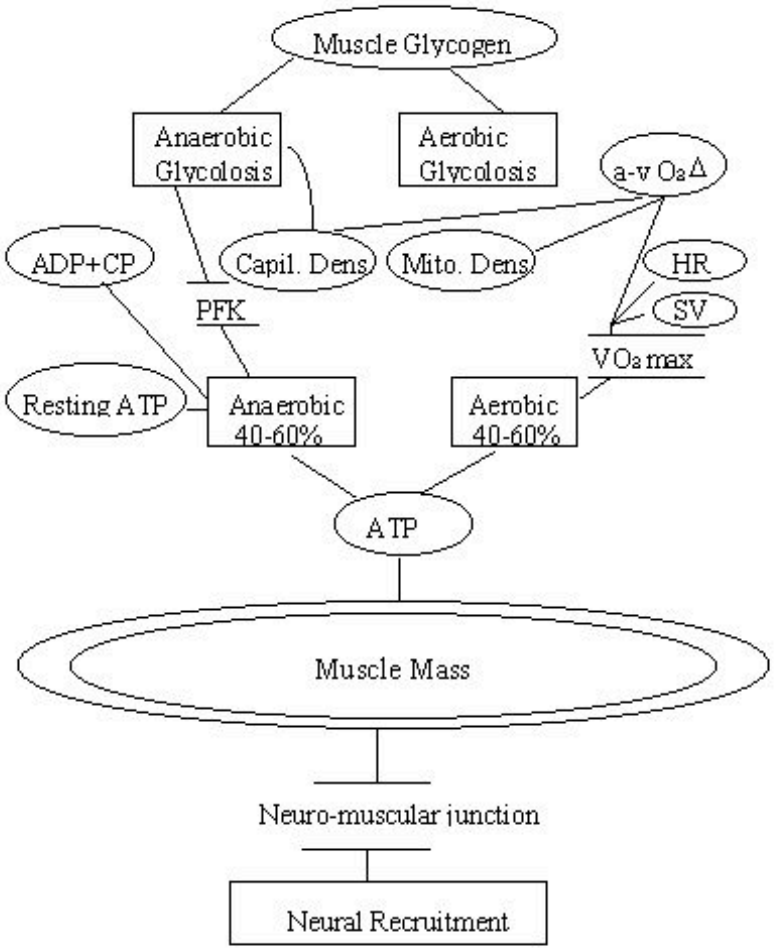
Aerobic energy production is limited by VO2 max, the maximum ability of an individual to take in and use oxygen. VO2 max is limited by both the amount of oxygenated blood the cardiopulmonary system can provide to the working muscles and by the amount of oxygen the working muscles can extract from the blood. The delivery of oxygenated blood to the working muscles is limited by cardiac output, which is the product of heart rate and stroke volume. The ability of the working muscles to extract oxygen is characterized by the arterial-venous oxygen difference (a-v O2 difference) and is limited by muscle mass, capillary density, and mitochondrial density.

Anaerobic work capacity is defined as the maximum amount of work that can be produced from anaerobic energy systems (Green, 1994). These systems include provision and phosphorylation of ATP from 3 sources: (1) stored ATP, (2) ATP that is resynthesized via the ADP+CP to ATP reaction, and (3) ATP produced by anaerobic glycolosis. The limiting factors for anaerobic work capacity are muscle mass, and muscle metabolic characteristics. There are several critical or rate limiting metabolic characteristics for anaerobic energy production, including catalyzing enzymes and substrates:

1. Resting levels of ATP
2. ATPase, which is rate limiting for hydrolysis of ATP,
3. Resting creatine phosphate (CP) levels, which limits the total energy available via the ADP+CP to ATP pathway,
4. Creatine kinase (CK) which catalyzes and is thought to be rate limiting for ADP + CP to ATP reaction,
5. Muscle glycogen as a substrate for glycolosis,
6. Phosphorylase (PHOS) and phosfofructokinase (PFK), which are thought to be rate limiting for anaerobic glycolosis, and
7. Anaerobic work capacity may be indirectly dependent upon capillary density which facilitates diffusion of metabolic byproducts out of the muscle.

Figure 1 below presents a general model for maximal short term performance which is based on these physiological systems. At the center of the model is the active muscle mass, which is supplied with ATP from both aerobic and

anaerobic systems, and which is recruited by neuromuscular signals. Next, we will examine the effects of aging on physiological systems.



Age related changes to the determinants of short term maximal performance

Many of the determinants for aerobic and anaerobic capacity change with age. Muscle mass, which is central to both aerobic and anaerobic capacity, decreases with sedentary aging, as shown by several investigations (Lexell et al, 1988, Coggan et al, 1992a, Gollnick et al, 1972, Larsson and Karlsson 1978, 1984, Grimby et al., 1983, Grimby et al., 1984, Lexell et al., 1983, Lexell et al, 1986). These decreases appear to occur primarily due to the loss of muscle fibers (Lexell et al., 1988), but are also due to atrophy of individual fibers, particularly fast twitch muscle fibers (Lexell et al, 1988, Coggan et al, 1992a). Some early research has suggested that there is a selective loss of fast twitch muscle fibers with aging (Gollnick et al, 1972, Larsson and Karlsson

1978). If true, this selective loss of fiber could result in a relative decrease in anaerobic work capacity, greater than the amount of muscle atrophy. However, more recent work has not confirmed this specificity of fiber loss (Sato et al., 1984, Grimby et al, 1983, Grimby et al, 1984, Lexell et al, 1983, Lexell et al, 1986), and indicates a preservation of muscle fiber type distribution with aging.

In healthy sedentary individuals, VO2 max decreases about 1% per year (Heath et al, 1981). This decrease is related to decreased capacity in all of the systems that determine VO2 max: maximum heart rate, stroke volume, and a-v O2 difference. Maximum heart rate declines about one beat per year with age (Shephard, 1987). Although results regarding stroke volume are mixed, most investigations have shown that during exercise, stroke volume decreases with sedentary aging (Hagberg et al, 1985, Ogawa et al,1992). However, other investigations show that stroke volume is maintained or even increased (Rodeheffer et al, 1984). Similarly, a-v O2 difference decreases with age in sedentary people (Hagberg et al, 1985, Ogawa et al, 1992). This decrease is related to decreased capillary density, mitochondrial density, and muscle mass.

Early studies of mitochondrial density in skeletal muscle did not indicate any age-related decrease (Oralander et al, 1978, Larsson et al, 1978a, Larsson et al, 1978b, Grimby et al, 1982). These findings may have been influenced by the effects of an active older subject population. More recently, however, investigations of subjects that were truly sedentary showed a significant (25-40%) age-related decrease in three mitochondrial enzymes (SDH, CS, and b-HAD, Coggan et al, 1992a), and in in-vitro muscle oxygen uptake (Meredith et al, 1989).

Parizcova et al, (1971), demonstrated that the number of capillaries per unit of cross-sectional area did not change with age, but that, due to muscle fiber atrophy, there was a significantly lower capillary to fiber ratio in the older subjects. These findings were supported by those of Grimby et al, (1982). More recently, however, Coggan et al, (1992a) has shown that in truly sedentary subjects, both capillary density and capillary to fiber ratio decrease with aging (Coggan et al, 1992a).

Early studies of age-related effects on the anaerobic enzymes in skeletal muscle showed that the maximal activity of PFK, MK, and ATPase did not change with age, but that LDH decreased significantly (Oralander et al, 1978, Oralander et al, 1980, Larsson et al, 1978a, Larsson et al, 1978b). These findings were supported by Aniansson et al., (1981) except that they did not

find an age related change in LDH. Grimby et al, (1982) found no age related decrease in the activity of HK and LDH. More recently, Coggan et al, (1992a) found no age related difference in phosphorylase, LDH, or PFK. Additionally, resting levels of ATP are not affected by age (Moller et al, 1980). Perhaps the most thorough investigation of glycolytic enzymes and aging was performed by Essen-Gustavsson and Borges (1986), who found no age-related change in HK, LDH, CK, or MK. Aside from early findings that LDH was reduced with aging, no age-related decrease in glycolitic enzyme activity or high energy phosphates has been demonstrated.

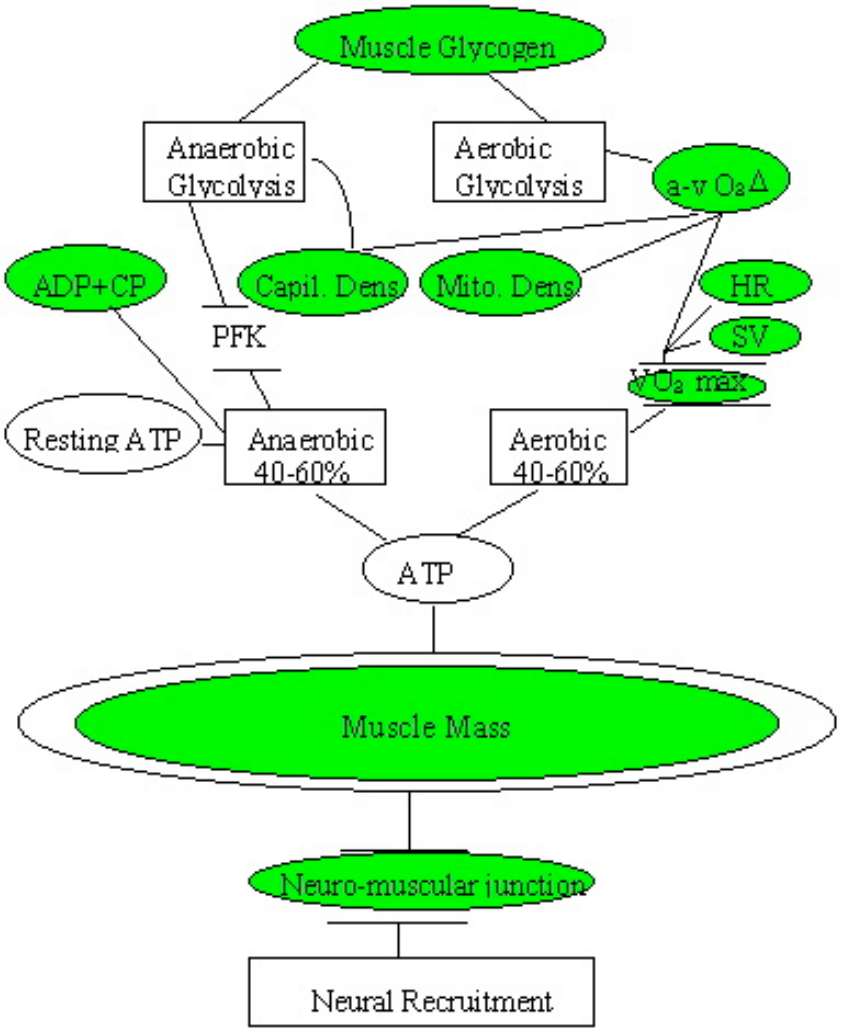
The findings regarding metabolism of glycogen might indicate that older individuals can metabolize muscle glycogen as well as younger individuals. Unfortunately, Meredith et al, (1989) have shown that older people have less muscle glycogen to metabolize. They showed that muscle glycogen in vastus lateralis was 61% higher in sedentary 24 year old men compared with sedentary 65 year old men.

Creatine phosphate concentrations have been shown to decrease slightly with respect to aging. Moller et al (1980) found a slight (5%) but significant decrease in creatine phosphate in skeletal muscle of men and women 52-79 years of age, compared with younger adults. McCully et al (1991) also found a small decrease in the creatine phosphate to inorganic phosphate ratio using magnetic resonance spectroscopy.

Neural activation of skeletal muscles may change with aging. Evidence to support this hypothesis stems from the finding that specific tension (i.e. force per unit cross sectional area) is maintained with aging in muscle fibers in-vitro (McCarter 1978), but not in whole muscles in-vivo (Phillips et al, 1992). Since neural enervation is maintained (Phillips et al, 1992 and Walters et al, 1990) with respect to aging, it is possible that "age-related deficiencies of motor function are probably related to other factors, such as those associated with neuromuscular transmission or propagation of nerve impulses" (McCarter and McGee, 1987). Smith and Rosenheimer (1984) investigated neural transmission velocity and found no age related effects in rats. They concluded that "smotor deficits during aging must be associated primarily with changes in central pathways or at the muscular apparatus." To test that hypothesis, they examined architectural changes to the neuromuscular junctions (Rosenheimer and Smith 1985), and found age-related changes in nerve terminal branch number; the rate of changes in end-plate morphology of all muscles studied increased after 25 months of age. In rats, 25 months is equal to the mean lifespan, so it is unclear whether these changes occur as a linear function of

aging, or as an end lifespan event. In summary, the neural signal to fire muscle contraction is maintained with age, but the ability to communicate that signal to the contractile machinery of the muscle may be compromised by changes at the motorneuron endplate.

Figure 2 below presents the effects of aging on the mechanisms of maximal short term performance. The shaded portions represent those systems in which maximal performance or function has been shown to decrease with age.



Next we will examine the effects of exercise on these systems in an attempt to determine if exercise can help maintain short term maximal performance with increasing age.

The age-related loss of muscle mass can be ameliorated with strength training (Brown et al, 1990, Charette et al, 1991, Fiatarone et al, 1990, Frontera et al,

1988). The subjects in the study of Frontera et al, (1988) increased muscle cross sectional area by 11% after 12 weeks of intense training, which is similar to the increases experienced by young adults for similar training (Hurley et al, 1991). The subjects in the study of Brown et al, (1990) trained only one arm for twelve weeks, and increased CSA in the trained arm by 17%. Charette et al, (1991) trained older women for 12 weeks. These women increased CSA by 20%. In all these studies, cross sectional area was increased due to hypertrophy of individual fibers, not by restoration of lost fibers. In an animal model study, Klitgarrd et al, (1989) found that weight training reduced the age-related decline in rat muscle.

In cross-sectional studies of VO2 max, older individuals who maintain vigorous endurance training, experience a decrease of about one-half of one percent per year, compared to younger adults (Heath et al., 1981, Ragers et al., 1990), or about half the decline experienced by sedentary adults. Some of this difference may be related to training between older and younger subjects. To control for training differences, Hagberg et al, (1985) matched older (56 year old) runners for training volume, intensity and 10k performance, with younger runners (25 years old) and still found a 9% difference, or 0.3% / year decrease in VO2 max. However, in a longitudinal investigation, runners who maintained their training over a ten year period did not exhibit any decrease in VO2 max (Pollock et al, 1987). Several of the runners in this study stopped training and they experienced a 14% decrease in VO2 max. This is interesting in that it is similar to the decrease in VO2 max experienced after only 56 days of detraining (Coyle et al, 1984). Taken together, these findings suggest that the effects attributed to aging in sedentary individuals and detraining in previously trained individuals may be difficult to separate.

With regard to the components of cardiac output, training has no effect on the age-related decrease in maximum heart rate (Rodeheffer et al, 1984, Hagberg et al, 1985, Shephard 1987). However, stroke volume is improved significantly with endurance training (Levy et al, 1993, Stratton et al, 1994). Specifically, training appears to enhance diastolic filling (Levy et al, 1993). In one of the best conceived investigations of training and hemodynamic function, Hagberg et al, (1985) studied older and younger runners who were matched for performance, training intensity and training volume. Their findings indicate that the decreased VO2 max of the older runners was solely a function of decreased maximum heart rate, and that stroke volume and a-v O2 difference were similar to those of the younger runners.

The age related decline in mitochondrial density may be ameliorated with exercise training. Early studies found little or no change in markers of mitochondrial density (Aniansson and Gustafsson, 1981, Oralander and Aniansson, 1980, Souminen et al, 1977). However, these studies used exercise protocols that were relatively low intensity, and were maintained for only a brief time. Also, the subjects in these studies may have been moderately active rather than truly sedentary. More recently, Coggan et al, (1992b), screened carefully to obtain a truly sedentary subject population, and employed a vigorous exercise regime for 9 to 12 months. They found that this exercise significantly increased citrate synthase, the best predictor of mitochondrial density (29% in men and 17% in women). Furthermore, masters runners who were matched for training volume and intensity with young runners had similar CS activity (Coggan et al, 1993). Also, the primary cause of loss of mitochondrial function is thought to be the prolonged effects of oxidative free radical stress (Ames et al., 1995 review), therefore it is possible that chronic intake of antioxidants may preserve mitochondrial density independent of exercise.

The results of investigations of capillary density are similar to those of mitochondrial density. Exercise regimes that involve low intensity exercise for a short period, and those that trained active older people did not increase muscle capillary density (Aniansson and Gustafsson, 1981, Oralander and Aniansson, 1980, Denis et al, 1986). However, Coggan et al, (1992b) found significant increases in both capillaries per unit cross-sectional area (16% for men and 25% for women) and in capillaries per fiber (26% for men and 38% for women) when truly sedentary individuals performed more intense exercise for a longer period. Furthermore, masters runners who were matched for training volume and intensity with young runners had similar capillary density in capillaries per mm2 and a greater number of capillaries per fiber (Coggan et al, 1993). Interestingly, the strength training performed by the subjects of Frontera et al, (1988) elicited an unexpected increase in VO2 max, capillary to fiber ratio, and mitochondrial enzyme activity.

Muscle glycogen is increased in young people after exercise training, but has apparently only been investigated once, by Meredith et al, (1989). They showed that a relatively short (12 week), but fairly intense (70% of VO2 peak) training program produced significant (27%) increase in muscle glycogen content. Even with this increase, the older subjects still had less muscle glycogen than either the trained or sedentary younger men. It remains to be determined if higher intensity or longer duration training would restore muscle glycogen content to levels equal to those of younger people.

The small age related decrease in creatine phosphate may be eliminated with training. Moller and Brandt (1982) showed that six weeks of cycle ergometer training increased creatine phosphate levels of 61 - 80 year old men to those of younger adults.

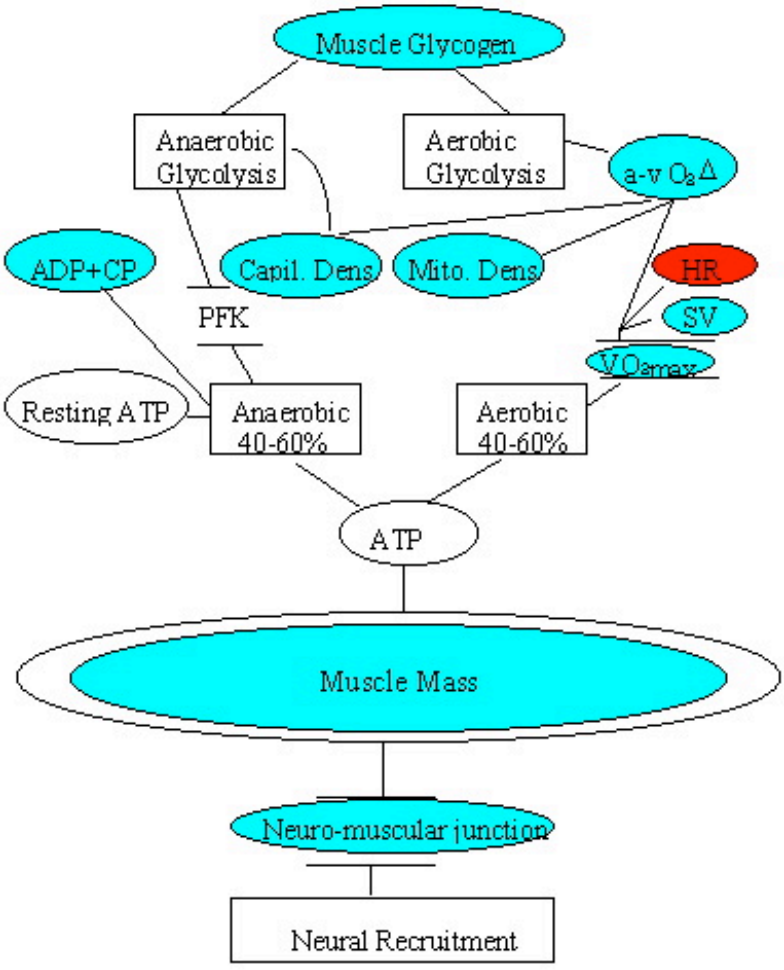
Although the joint effects of aging and exercise on motorneuron endplate structure has not been specifically investigated, we may be able to hypothesize on the effects based on other findings. Specifically, in the strength training studies cited, the increases in strength are greater than the increase in muscle cross sectional area, indicating an increase in specific tension. It is known that specific tension in muscle fibers in-vitro is maintained with aging (McCarter, 1978) but decreased in whole muscle in-vivo (Phillips, et al, 1992), probably due to changes in motorneuron endplate structure (Rosenheimer and Smith, 1985). Therefore, we can surmise that the apparent increase in specific tension with strength training is due, at least in some measure, to increased performance of the motorneuron. It should also be mentioned that improvements in expressed strength after training may be in part due to improved coordination and learning.

Summary

Many, but not all of the effects of sedentary aging can be altered with exercise training. Figure 3 presents the combined effects of aging and exercise on the mechanisms of maximal short term performance. The blue shaded portions represent those systems in which age related decreases in maximal performance or function has been shown to be minimized or eliminated with exercise. The red shaded portions represent those systems in which age related decreases in maximal function is not minimized by exercise.

Muscle mass can be at least partially maintained with strength or endurance training. Mitochondrial density and capillary density can be maintained with vigorous exercise training. Stroke volume can be increased with exercise training, but heart rate declines irrespective of activity level, resulting in an age related decrease in cardiac output and VO2 max. Muscle glycogen content can be increased in older individuals, but the studies that have been conducted to date have used low intensity or short term training programs and have not increased muscle glycogen levels up to those of younger people. Exercise probably enhances the function of the neuromuscular junction, and allows better recruitment of skeletal muscle. Based on the findings presented, our model predicts that sedentary older adults should have dramatically lower

short term maximal performance than younger adults. However, an older adult who is trained at the same volume and intensity as a younger adult should be capable of performances similar to his/her younger counterpart. The unalterable decrease in heart rate and consequential reduction in VO2 max may prevent the older individual from training at the same volume and intensity as a younger athlete, and consequently, performance of the older athlete will be limited by the reduction in absolute training volume and intensity. However, the trained older adult should almost certainly have performances superior to those of younger and older sedentary adults, and therefore may have a better ability to cope with the physiological demands of free living.



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5) PRINCIPLES OF TRAINING - REVISITED

I think if you have been in the exercise game for any time at all, you know a lot about the "principles of training". But, I still want to explore this topic a little bit as a prelude to additional training articles that will follow. I will discuss four training principles. Depending on what your read, there are others. This is especially true if you happen to read "Muscle and Fitness". But, I think these main concepts are fundamental to understanding exercise induced adaptation, and encompass most everything else.

1. The Overload Principle

The Cells are Sensitive

We are biological organisms composed of an interdependent assortment of billions of individual cells. It has been said that "every cell in our body is psychological". This may sound crazy, but in a sense it is true. Every cell is in some form or another sensitive to certain forms of stress, and capable of initiating a specific **response**.

Training is a cyclical process of tearing down and building up

Part of understanding this overload principle is knowing that the adaptations we are trying to stimulate require synthesis of new biological material. This process takes time! Even as you sit reading, your body is constantly in a state of deterioration and repair. Some cells, like red blood cells are dying out completely at the rate of 2-3 milllion every second, and being replaced just as fast! Others, like muscle cells, hang around much longer, but are constantly repairing themselves from within. When we train, we do additional, specific damage to some cells, and use up cellular resources (fuel, water, and salts are 3 examples). When you walk off the track or get out of the pool after a workout, you are WEAKER, not stronger. How much weaker depends on the severity of the exercise stress. The cells always seek to maintain *homeostasis*, or the status quo, so the cellular and systemic stress of exercise elicits not just a repair to former levels, but an adjustment, or build-up, of the stressed system that serves to minimize the future impact of the stressor. For example, the depletion of muscle glycogen to low levels by a lengthy exercise session triggers a rebound increase in glycogen storage level. Another example, getting hot and bothered during a run on the first hot summer day initiates a process of adaptation whereby we, within 10 days or so of repeated heat exposures, turn on sweating faster, more intensely, and over a bigger skin

surface area, but lose less salt (which means our eyes stop burning when we get that more dilute sweat in them. This **GENERAL ADAPTATION SYNDROME** was described by Hans Selye, and expanded by Yakovlev. If the stress is too small in either intensity or duration, little or no adaptation growth is stimulated. On the other hand, if the stress is too severe, "growth" is delayed or even prevented.

Maintaining homeostasis in the face of chronic stress means increasing the synthesis of specific proteins (mitochondrial enzymes for example) that enable the cell to respond to future demands with less disruption. The optimal training program would be one that maximally stimulated these positive adaptations, while minimizing the cellular and systemic stress thrown at the body in order to trigger the changes. Very hard training does damage and sometimes threatens our health by transiently lowering our resistance to infection. Not to mention the fact that it can stress our time schedules and relationships. Put in real world training terms, the doubled edged sword nature of the body's response to training suggests that we should try to organize training (frequency, intensity and duration) in such a way that we minimize the negative stress effects while still achieving the physiological adaptations desired. This program would then incorporate the appropriate **recovery time**; 1) long enough to allow the synthetic processes time to occur, while 2) not so long that reversion back towards the previous cellular state could begin. Finally. our overall training program would have to recognize that some cellular adaptations have a faster response time than others. For example, plasma volume increases dramatically within a week of hard training, while capillary growth occurs slowly after years of training. This knowledge will impact the relative amount of training we dedicate to achieving specific adaptations.

Thresholds and Diminishing Returns

If we put this Overload Principle into action, we are talking about regular exercise. When we train, we choose some specific **intensity and duration** of effort (or sometimes IT chooses us!). Then we repeat these efforts with some specific **frequency**. Add in the mode(s) of exercise and you have the 4 variables of a training program. Since even the most untrained body has a built-in reserve capacity to handle a substantial degree of stress, there is a minimum threshold for intensity and duration of stress that must be exceeded before additional adaptations are triggered. This is the **minimum training threshold**. For example, in untrained people starting an exercise program, we don't see significant improvements in exercise capacity unless the training intensity exceeds 50% of their maximal oxygen consumption, but this intensity

isn't too difficult to achieve. If you have been doing nothing, almost anything helps. However, the threshold level (in terms of the combination of intensity and duration of exercise) for further adaptation increases as we become more fit. In elite young and older athletes, the threshold for a **positive training response** may exceed 80% of VO2 max. So does this mean that every training session should be above this intensity? No, this is an important lesson to learn, usually discovered after repeated injuries, overtraining, and staleness. Exercise at below the higher training threshold can be important for **maintaining** existing adaptations while **allowing recovery processes to occur.** What we are faced with as we continue training is a **diminishing return** on our training investment. The better adapted we are to exercise, the more difficult it is to induce further positive changes. Emerging from this fact is the use of **periodization of training**, a common training term these days. At the elite level the diminishing returns on training investment are clearly evident as athletes train 3-4 hours per day in order to be 1% faster than if they trained 1.5 hours per day. And they gamble this 1% improvement against the greatly increased risk that they will become injured or sick due to the extra training load. So, we each have to decide how important that last 1% is to us.

2. The Principle of Specificity

I think it is safe to say that the media and shoe makers have combined to confuse many young and older athletes about the Principle of Specificity. Nike, and all the folks who sell exercise equipment would like you to believe that "Cross-training" is a key to peak performance. The concept sells more sports shoes and exercise machines, but is it true? Well, no. Any sport you pursue places highly specific demands on your body in at least two major ways. First, the exercise will have a **very specific pattern of joint and muscle coordination**. For a rower, there is absolutely no substitute for rowing. Ditto for swimming. Even when we try to duplicate the basic movement of a sports skill with strength training exercises, the transfer of increased strength to the actual sports movement is often small or absent. In the worst case this type of training can detract from performance of the real skill due to disruption of technique. Second, the **exercise will place high metabolic** demands on a very specific group of muscles. For example, running and cross-country skiing appear to involve many of the same muscles, used in a similar movement pattern. Yet, several research studies have demonstrated that there is NO relationship between VO2 max measured by treadmill running and VO2 max measured by cross-country skiing in a group of elite-trained skiers. In contrast, there is a strong relationship between on-snow skiing and performance on a skiing specific test such as the double poling test.

A high endurance capacity in a specific sport requires both 1) high oxygen delivery (cardiac output) and 2) high local blood flow and mitochondrial density in the precise muscles used. The only way to **optimally** develop the second component of endurance is to train those exact muscles by doing your sport!

Is there ever a place for cross training?

The answer to that question is definitely yes! BUT, we need to understand as athletes what the limited purpose and value of the alternate exercise modes are. For example, I work with some world class speedskaters from Holland on training issues and physiological testing. After a couple of years of observations, it is clear to me that they just cannot skate every day, at least not in a good competitive skating position. The stress on the legs is just too great. So, in order to achieve the training volumes that we think are necessary for success at that level, the skaters also do a lot of cycling, even during times in the year when ice is available. Our choice of cross-training is an effort to combine the needs for highly specific loading with the need of these elite level athletes to train at high volumes (20 hours per week or more during the preparation period). Take note though that during the racing season, essentially all hard training is performed on the ice, with low intensity and recovery workouts performed on the bicycle. So even when we use cross-training, we are keeping our eyes on the real goal. Or, take me, a 40 year old masters rower for example. During the Spring and Summer back in Austin, Texas, 90% of my endurance training was performed on the water, rowing. However, in the late fall and winter (non-competitve period), I rowed less on the water than I could have (no ice in Austin), probably half as much. Why? Mostly because I was mentally tired of rowing, but also because of weather and time constraints. Sometimes I would row on the indoor machine, by no means a perfect substitute for the technique of rowing, but a good simulation for developing basic rowing endurance. But to be honest, on most days, I hate being on that machine for more than about 45 minutes. Embracing the expression "the mind needs rest, but the body needs work", I would often mix in running or cycling on an ergometer with my rowing to increase my total aerobic exercise volume without growing mentally stale. A little bit of cross-training helped maintain my general aerobic base, while allowing me to mentally recharge my batteries in anticipation of another cycle of intense training on the water with my rowing partners.

Another reason to "cross-train" is to avoid injury and maintain muscular balance DURING a period of intense sport specific training. One of the keys to

success in sport is staying healthy over the long haul. Weight training by itself will almost certainly do nothing for a runner's 10k time, but if weight training maintains muscular balance in her abdominal wall and low back, preventing injury, then it is contributing to her becoming a faster runner. Why? Because it keeps her running! And, cycling isn't running. But if cycling takes the pressure off tired knees and hips on a recovery steady-state day, then it will probably make the next running workout better. Cross training should always be limited to those activities that allow us to do our event-specific training workouts with greater enthusiasm and intensity, or less risk of injury. It is a cautiously administered **supplement, not a substitute**!

3. The Reversibility Principle

If people were as economical as their bodies, we would not have problems with personal debt and excess world waste production. The human body is nothing if not thrifty! The iron and protein in those millions of blood cells that die each day is almost completely re-used to build new blood cells! The body does not **build** proteins it doesn't need (except maybe those that make up the appendix?), and it doesn't retain proteins that are no longer needed! For the athlete, the unfortunate consquence of this thriftiness is the rapid reversibility of training adaptations if training is stopped. In general, I think it is fair to say that those adaptations that occur fastest when we start training fade away fastest when we stop training. So, a week in bed with the flu will result in a substantial loss in blood plasma volume, but little change in mitochondrial enzyme concentration, and essentially no change in capillary density. Once over the virus, a couple of good traiing bouts will have blood volume back up to normal levels, and cardiac function back to normal as a result. However, take 3 months completely off from your training routine due to a big project at work and you will lose a lot of the adaptive foundation gained over the previous year of regular workouts. If you were highly fit before the break, it may take 6 months to come all the way back. What is clear is that training adapatations are always transient and dependent on chronic stress to the system. However, it does seem that people who have been really fit, and take a break, often seem to be able to return to high fitness levels FASTER than those who have not been highly trained before. Whether this is a function of good genetics for training responsiveness, a certain "muscle memory" in the brain or muscle cells of the detrained athlete, or just past knowledge of how to train is unclear, but it does seem to be real.

4. The Principle of Individual Differences

Last but not least on the list of Training Principles is the Principle of Individual Differences.

We All Start Somewhere . . . different

It is usually practical to describe physical characteristics based on some AVERAGE. On average, American men (no offense to my international readers) are currently 5' 9" (1.75 m) tall and about 180 pounds (82kg). But, walk down a busy street and you will see that there is considerable variability! It shouldn't be too surprising that there is also a lot of variability in our internal charactersitics. Heart size, muscle mass, bone diameter, fiber type composition, position of mucle attachments on bone, fat distribution pattern, joint flexibility, etc, all vary from individual to individual. Two examples: **On average**, a 25 year old untrained man will have a maximal oxygen consumption of 45 ml/min/kg. However, there are completely untrained people who have walked into a lab, got on a treadmill and had a VO2 max of 70 ml/min/kg. I tested a fellow exactly like this myself once. I was teaching a class and he "volunteered" to perform a cycling max test. I predicted his max for the class based on his exercise history (little if any). Imagine my surprise as he kept cycling and his VO2 kept climbing and climbing as I progressively increased the workload on the bike! He didn't bother to tell me until after the test that his sister had rowed in the Olympics! There are equally "healthy" untrained young men whose max is only 35 ml/min/kg. That's a 2X difference in aerobic capacity before they do their first workout! This is a physiological gap that will not be closed, no matter how hard the "less endowed" fellow trains. If the high VO2 guy trains very hard, he **might** reach 80 ml/kg/min, a 14% increase. The low VO2 guy can train equally hard and possibly reach 50 ml/kg/min, a larger 42% increase. The gap can narrow (to 60% here), but it will not go away. Genetics place limitations on our body.

Example number two: **On average**, the fiber type distribution in the thigh muscles of a male (or female) is roughly 50% slow and 50% fast fibers. However, in a study by Simoneau et al, 1989, muscle biopsies from the vastus lateralis (outside thigh) of 418 males and females revealed a range of from 15% slow fibers to 85% slow fibers in different people. Coefficients of variation approached 30%. Again we see that there is considerable genetic variation in a variable that has significant impact on performance. So, we each have to focus on approaching the outer boundaries of OUR OWN physical potential.

Different Strokes for Different Folks

At the Laval University in Canada, the University of Texas at Austin, and three other Universities in the United States, a major collaborative project was undertaken to determine the role of genetic variability associated with individual responses to an identical training program. Fittingly, this project was called the Heritage Study. Millions of dollars were spent to quantify and understand the genetic foundations of a phenomenom that athletes already know full well. **We all respond differently to a training program.** What this major study clearly demonstrated was that not only is our physiological "starting point" highly individual, but our **training response** is also highly variable. In this study, there were some subjects who essentially did not show ANY adaptation to a very well-controlled training program (measured for example as an increase in VO2 max), while others increased as much as 40% when doing the exact same training. Some athletes can do next to nothing 3 months then train like a madman, sweat, and spew chunks for three weeks and be in racing shape (ok, maybe too graphic). Others are "hard gainers" that seem to lose everything if they miss 2 weeks of training. Some people tolerate and even thrive on, a high volume of training to reach peak fitness. Others cannot tolerate the same workload, but reach similar performance levels if they intersperse more rest days. We each have a unique psychological makeup. We have different strengths and "weaknesses" within our physiological performance machine that should influence training plan design, and we have different hormonal and immune reactivity that will influence the level of stress we can tolerate and improve under. In the field of exercise physiology, we have learned a great deal about physiological adaptations and the general methods of training that conform to known physiology. This is very valuable information for the athlete to understand whether 24 or 64 (Of course I am biased on that score). But, remember, ANY exact training program that you copy from me or someone else is destined to be, at best an approximation of what will work best for you, and at worst, a total failure.

The Bottom Line

Ok, you love your sport and are motivated to improve, but with so many possible training methods and "experts", what can you do? Well, here is what I think.

First, understand what training does to your body. Learn the physiology of the sport (hopefully the MAPP will help). Know how your engine works. This

will help you critically evaluate the disparate training ideas that are thrown your way.

Next, examine and **learn the biomechanical principles that must be obeyed for performance success**. How do you maximize the efficiency of transfer of your engine power to performance velocity? There is no endurance sport that does not place a premium on good technique.

Finally, **keep a record of what you do!** Use a notebook and pencil, or a fancy computer program, but make yourself accountable to both the training you do in pursuit of your performance goals, and the results. If you do this, eventually you will have arrived at your own personal prescription for success, built from solid general principles, but fine tuned to your personal characteristics. "Success" will vary for each of you in absolute terms; completing a 10k, a new personal best, a city championship, or maybe a world veteran's record! But it all feels the same to the person who establishes the goal, develops a plan, and works diligently to achieve it! Then you can tell us about it on the MAPP!

6) THE TIME COURSE OF TRAINING ADAPTATIONS

So far, I have exposed you to some basic physiological variables that are known to 1) bear a strong relationship on endurance performance in every sport and 2) respond to training. By now, I hope you can recite with me the "Big Three" elements of endurance performance:

1. **Maximal Oxygen Consumption**
2. **Lactate Threshold (OBLA - Onset of Blood Lactate Accumulation)**
3. **Efficiency**

Number 1 is an oxygen delivery issue. A high maximal capacity for blood delivery means higher oxygen delivery and the potential for more muscle to be active simultaneously during exercise. VO2 is primarily limited by the maximum pumping capacity of the heart, and the specific arterial development to the active muscles.

Number 2 is an oxygen utilization issue. The greater the intensity of work we can achieve prior to the point when we begin to accumulate the inhibiting acidity of lactic acid, the faster sustained pace we can tolerate. The limiting adaptations are the capillary density, fatty acid breakdown, enzyme level and mitochondrial density in the specific skeletal muscles used in your sport. Combining elements 1 and 2 gives us the sustainable power output of your "performance engine".

Number 3 Efficiency, links sustainable power to performance velocity. The better the efficiency, the greater the achieved velocity at a given level of energy output. Since, ultimately, we have a limited "engine" size, improvements in efficiency are critical to additional improvements in performance time.

In this article I want to discuss the time-course of change in these variables. "How long does it take for my max VO2 to peak out?" "What about lactate threshold?" Understanding the answers to these questions will be important as we try to build appropriate training programs.

The First Wave of Change- Increased Maximal Oxygen Consumption *(see subcategory-(1.a.ii) above)*

In a **previously untrained person**, VO2 max is increased significantly after only one week of training! The reason for this early improvement appears to be

an increase in blood volume, which results in improved maximal stroke volume ***(see subcategory-(1.a.i) above)***. As training continues, VO2 max continues to increase, for several months, albeit at a slower rate of improvement. We have already discussed the fact that the heart appears to be remodeled by endurance training, developing a greater ventricular volume diameter, and other more subtle adaptations that make it a more effective pump. After about 3-4 months of regular exercise, the improvement in maximal oxygen consumption begins to level off dramatically. At this point, it is common to see about a 15-20% improvement in this variable. For example, a hypothetical male (who I will call Bjorn) with an initial VO2 max of 3.5 liters/min (at a bodyweight of 75kg, that's 47ml/min/kg) may increase to 4.0 liters/min, a 14% increase in **absolute** VO2. If in the process of training, Bjorn also loses 4kg (close to 10 pounds), then his **relative** VO2 max will have increased even more (from 3500/75 or 47, to 4000/71 or 56 ml/min/kg). This is a nearly 20% improvement. Unfortunately, after another 6 months of training, it will have increased little more, if any. If the level of training intensity remained the same after the first 4 months, then no further changes would be expected. If on the other hand, Bjorn continues to intensify his training over the next 6 months, a small additional increase might occur. This increase might be as much as 5 additional percent, bringing our example athlete up from an initial value of 3.5 liters/ min at 75 kg, to 4.2 liters/min at 70kg (he also lost another 1 kg of fat). That's 47 ml/min/kg up to 60 ml/min/kg due to a combination of both increased absolute VO2max (20%) and decreased bodyweight (6.7%), for a total improvement in relative maximal oxygen consumption of 27%. This is actually an unusually large improvement in this variable, but definitely plausible.

If our example subject started at a higher level of VO2 ***(see subcategory-(5) above)***, the relative improvement would almost certainly be less dramatic. The important point to recognize from this is that VO2 max increases fairly rapidly in response to chronic exercise, then plateaus. If our example athlete continues training another 5 years, his VO2 max won't improve any more. It might actually decrease slightly due to age related declines in maximal heart rate ***(see subcategory(1.c) above***. Depressed? Don't be. There is much more to endurance performance then the VO2 max.

The Second Wave of Change - The Lactate Threshold *(see subcategory-(3.a) above)*

At the same time Bjorn's VO2 max was increasing due to central and peripheral cardiovascular adaptations, changes were beginning to occur in his

skeletal muscles (let's assume Bjorn is a runner, so the adaptations of interest are happening in the legs).

Initially, an incremental exercise test on a treadmill revealed that Bjorn began to show a substantial increase in lactic acid concentration in his blood while running at 60% of his maximal oxygen consumption. Remember, his max was 3.5 liters/min. 60% of this is 2.1 l/min. So functionally speaking, **2.1 l/min** was his threshold workload for sustained exercise. If he runs at a speed that elicits a higher VO2 than 2.1, he fatigues quite quickly. However, over time, the overload of training induced quantitative changes to begin occurring in his leg muscles. Mitochondrial synthesis increased. More enzymes necessary for fatty acid metabolism within the muscle cell were produced. And, the number of capillaries surrounding his muscle fibers began to increase. Additional capillaries are being constructed. The functional consequence of these local muscular adaptations is a very positive one. Bjorn's running muscles use more fat and less glycogen at any given running pace. And, the glycogen metabolized to pyruvate is less likely to be converted to lactic acid and more likely to inter the mitochondria for complete oxidative metabolism. Consequently, Bjorn's lactate threshold begins to increase. After 6 months of training, in addition to a higher VO2max, his lactate threshold has increased from 60% to 70% of max, a 17% improvement in an absolute sense, but functionally much more. Why? Because the 70% is relative to an increased max! So, Bjorn has gone from an initial sustainable oxygen consumption of **2.1 liters/min** (60% of 3.5) to a new sustainable intensity of **2.8 liters/min**, a 33% improvement!

Now, the important thing to know is this. While VO2 max plateaus quite rapidly, lactate threshold does not. If Bjorn continues to train, and increase his intensity appropriately, his lactate threshold will continue to improve slowly for a longer period. Of course, improvements in lactate threshold also plateau, otherwise elite athletes that have been training for 15 years would have LT's of 100% of VO2 max! **But, the time course of adaptation is slower**, so the plateau occurs after a longer period of intense training, probably several years.

It is also important to remember that the lactate threshold is even more specific to the mode of exercise than the VO2 max. This was exemplified by a study performed by Coyle et al. and published in 1991. In this study, 14 competitive cyclists with nearly identical VO2 max values differed substantially in their lactate threshold determined during cycling (ranging between 61 and 86% of VO2 max). When the cyclists were divided into a "low" and "high" LT groups (66% vs 81% of maximal oxygen consumption), it was found that the two

groups differed considerably in the years of **cycling training** (2.7 compared to 5.1 years on average). However, they did not differ in years of **endurance training** (7-8 years of running, rowing etc.) When the low cycling LT and high cycling LT groups were asked to perform a lactate threshold test while running on a treadmill, the two groups were no longer different. Measured while running, the lactate threshold in both groups averaged over 80% of VO2 max. Similarly, if you are a runner and decide to add swimming and cycling to your training and compete in triathlons, you will immediately recognize that your running fitness does not immediately transfer to the bike, and of course not to the water!

The Third Wave of Change - Efficiency *(see subcategory-(3.b) above)*

The final element of our BIG THREE endurance adaptations is efficiency. I think we all know what it means to be an "efficient" person, or own a "fuel efficient car". But, what does the term mean when applied to endurance performance? It means the same thing, getting more done at lower "cost". Efficiency is defined as MECHANICAL WORK / METABOLIC WORK. For example, one (quite good) cyclist can sustain 300 watts power output for 1 hour on a cycling ergometer at a sustained VO2 of 4.3 liters/min. Another rider performing at the same oxygen consumption, squeezes out 315 watts, a difference in **efficiency** of 5%. Even though both riders have the same "metabolic engine" they have different power output capabilities. You don't do 40k time trials on a lab ergometer, though. So, thanks to my friend the cycling guru, Jim Martin, we can predict their actual performance time in a 40k time trial. If these two cyclists have identical aerodynamics and use aero bars, the times will be 56:10 vs. 55:15. This is only a one minute difference, but probably worth at least 2 or 3 places at the Masters Nationals Time Trial!

So efficiency makes a difference, often much bigger than the above example. And it also varies among different athletes. That's interesting, but not terribly useful for YOUR training. Your big question is probably "Can **My** Efficiency Improve With Training?". The answer is YES. In highly technical sports like swimming, efficiency differences between beginners and experienced swimmers can be absolutely tremendous! Swimmers already know this full well. In rowing, efficiency also improves dramatically at first, due to gross technical improvements. However, efficiency can also continue to improve after years of training. Dr. Fritz Hagerman followed one group of national class (U.S.) rowers for 8 years, measuring ergometer performance, VO2, lactate threshold, etc. Peak values for maximal oxygen consumption and lactate threshold stabilized after only 2 or 3 years in these hard training athletes.

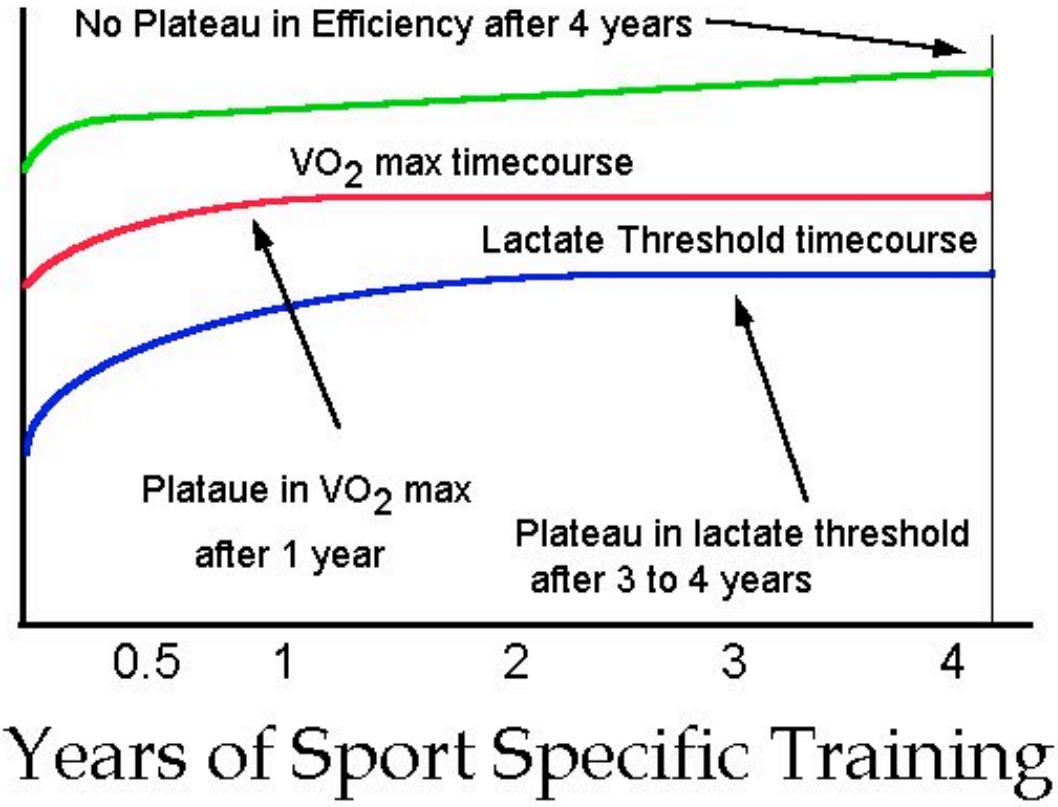
However, performance times on the water and on the rowing machine continued to improve over additional years of training. The reason? Slow improvements in rowing efficiency. One source that is independent of on-water technique may be optimization of workload distribution among the large muscle mass employed in rowing. Ultimately, the rowers who went on to become national team members and have success at the highest levels were more efficient than their peers.

What about the "less technical" sports like cycling and running?

For you cyclists, I call cycling less technical only in reference to the act of pushing the pedals, not all of the equipment and aerodynamics! Again there is evidence for significant improvements in efficiency even after years of training. In studies performed on "Good" vs. "Elite" cyclists carried out by Dr. Ed Coyle and colleagues at the University of Texas, it appears that elite riders sustain higher power outputs despite similar physiological values in part by learning to distribute the pedalling force over a larger muscle mass. In running, fomer U.S record holder in the mile, Steve Scott, was shown to have improved his running efficiency even "late" in his career.

The Bottom Line

Based on a tremendous amount of both laboratory and "field" data, I would propose to you that the order in which the BIG THREE endurance performance variables reach their peak is 1)VO2max, then 2) lactate threshold, then 3) performance efficiency. Putting it all together, and neglecting for now the negative impact of aging on maximal oxygen consumption ***(see subcategory-(1.c) above)***, we might get something like the figure below. The figure below is obviously very generalized. In reality, all three variables fluctuate during the year (off season vs competitiion period) as a function of training intensity and volume. Peak values after a given period of training will approximate this kind of pattern, though.



Obviously, if you are just beginning in an endurance sport, then all three elements will probably improve dramatically, almost no matter what you do! But, if you have been training in sport for a year or more, you must construct your training program with more and more care to continue making progress in those adaptations that have "room to improve" while maintaining the levels of those that have plateaued or are beginning to. Since for the masters athlete, the option of "just adding another workout" is usually not a viable one, this will often mean finding the right distribution of a limited amount of training time among a variety of workout types. In the next article on my agenda, "Understanding Intervals", I will start to explore some different training methods used by the endurance athlete, from a physiological standpoint. Stay tuned.

7) UNDERSTANDING INTERVALS
Matching training characteristics to physiological changes

Now we come to a fundamental training question that has interested me for years. Which is "better", Interval Training or Steady State Training? Obviously that is simplistically stated, but I will try to make things more clear as we go. Eight years ago, my master's thesis asked this exact question. One group of rats "volunteered" to run repeated 2 minute high intensity intervals every day (5 days/week), another group ran 60 minutes continuously each day, and a third group served as cage potato controls. We will come back to the results later. I learned from that study and have continued to learn since.

Interval Training Defined

In a dictionary, an *interval* is probably defined as a period of time or a specified distance. Well, that's true, but for us endurance athletes it means *repeated bouts of high intensity exercise with intermittent rest periods*. In endurance training circles, no matter what the sport, *Interval Training* has become a standard practice. Since the 1960's, interval training has come to be thought of as the key to endurance performance success. In some training programs, it accounts for 50-75% of the total training volume. One fellow I know who coaches runners has even written an article espousing "All Intervals, All the Time." After 10 years of research, reading, observing, and training, I think this line of thinking must be evaluated critically, so here goes. I will hit you with information from a lot of different angles, but hopefully what emerges will be a coherent conclusion.

The Physiology of Intermittent Exercise

In exercise physiology research, one of the true "Fathers" of the field is Per Astrand from Sweden. Today, Dr. Astrand is still showing up at International Sports Medicine research meetings at the age of 80 or so. His best known contribution, "Textbook of Work Physiology", co-written with Norwegian Kaare Rodahl and now in its 5th edition or so, remains a "must have" for the student of exercise physiology. In the 60s he performed some simple experiments that still have important training implications regarding the physiological impact of continuous versus intermittent exercise.

In a lab setting, a bicycle ergometer serves as a very useful tool for performing exercise studies. We can precisely control the workload that the cyclist must maintain, and since he/she is sitting still, it is easy to perform an extensive

array of physiological measurements, such as heart rate, VO2, blood lactate etc. (I prefer not to dwell on just how extensive!) The following experiments were performed using just such an exercise setup.

One subject was made to accomplish a certain amount of **work** (force x distance, a quantity that can be precisely measured on the ergometer) in 1 hour. This work could be accomplshed either by a **continuous** bout of exercise at a power output of 175 watts, or by **intermittent** exercise at a heavier load, separated by regularly spaced rest intervals. A double power output was chosen for this heavier load. Thus the desired work could be acomplished with 30 minutes of exercise at 350 watts within the span of one hour. At a work load of 175 watts, the subject could easily cycle for one hour continuously. Heart rate was only 134, VO2 was only about 55% of maximal, and blood lactate remained near resting levels. When the subject tried to exercise as long as possible at 350 watts, or double that workload, the exercise could only be maintained for 9 minutes. Heart rate was 190 bpm (maximal), VO2 was at max, and blood lactate had risen to 16.5 mM, an extremely high value indicative of extreme fatigue. If, instead, he exercised at the same 350 watts intermittently for a duration of between 30 seconds and 3 minutes, always with equal rest, he could perform the desired work within the hour. **However, the physiological responses differed tremendously depending on the interval duration.**

Exercise condition	VO2 (l/min)	Heart Rate	Blood lactic acid (mM)
		Continuous	
175 watts	2.44	134	1.3
350 watts **	4.6	190	16.5
		Intermittent #	
30 seconds*	2.90	150	2.2
1 minute*	2.93	167	5.0
2 minutes*	4.4	178	10.5
3 minutes*	4.6	188	13.2

** Could be sustained for only 9 minutes
# All physiological values represent peak values achieved during exercise
* Rest duration equaled work duration in each condition

INTERPRETATION

There are two major points I want to draw from Dr. Astrand's experiments:

1) Intermittent exercise allows a higher total volume of high intensity work. Performed continuously, the subject could only manage 9 minutes at 350 watts. Performed in 3 minute intervals, he could accumulate over 3 times as much total work (30 minutes, with great effort).

2) When the intervals were 3 minutes in length, the desired work could be accomplished within one hour, at great effort. However, when the work and rest periods were shortened, the physiological strain was dramatically reduced, even though total oxygen uptake during the hour was not markedly reduced. Specifically, if the intervals are less than 2 minutes in length, the physiological workload/stress is severely reduced despite the same total accumulated time (30 minutes here) and same interval intensity (350 watts workload here). If you compare the peak oxygen consumption, HR, and lactic acid concentration achieved during 1 minute intervals with 2 minute intervals in the table above, this difference is easily observed.

Why are the responses so different?

I think the best explanation for the difference was put forward by Astrand. He suggested that during very brief intervals, oxygen bound to myoglobin served as an effective buffer against the accumulation of an oxygen deficit (and lactic acid) during the exercise bout. Therefore after a 30 second bout, myoglobin oxygen stores were repleted during the rest period, and the peak demand on oxygen delivery was not severe. By analogy, the body manages to live expensively, and briefly deplete cash reserves, then always repay the small debt during a subsequent "debt recovery period". No long term debt accumulates. As the exercise bout lengthens, the capacity of the small buffering myoglobin oxygen store is outstripped, lactic acid production and accumulation becomes significant, and the burden of greater oxygen delivery during the work interval falls on the cardiovascular system.

Subsequent experiments by Astrand showed that if you shortened the work period and rest periods to smaller and smaller intervals, it was possible to perform at even higher power outputs without accumulating lactic acid or severely stressing the cardiovascular system.

Conclusions So Far

For a period of intermittent exercise that approximates a max VO2 workload to overload the cardiovascular system effectively, it needs to be of at least 2 minutes duration due to 1) lag time in the cardiovascular response and 2) the oxygen buffering effect of myoglobin.

Now, lets go back to my master's thesis. The question I set out to address with that study was this: "What is the impact of interval training and steady state training on the performance of the rat heart?" I wanted to evaluate the heart independent of the muscles, and make direct measurements, so I had to use rats. I bred a group of rats in the laboratory, meaning they were litter-mates and therefore, presumably quite similar genetically (they sure looked the same). After they reached adulthood, I divided them into three groups. One group sat around in cages, ate rat chow and got weighed periodically. A second group was gradually adapted over 4 weeks (several months in rat years) to running on a treadmill until they were running for 60 minutes, 5 days a week at an intensity that was equal to about 75% of VO2 max. The third group was adapted to an interval running program. These guys would run two minute bouts at a speed that required just over 100% of VO2 max, then slow down for two minutes, and so on for 10 bouts (after four weeks of buildup). The total running distance was the same for the two groups, only the intensity differed. To evaluate cardiac performance, I develped a surgical procedure under anesthesia (the rats, not me) that allowed me to directly and constantly measure cardiac output, intraventricular pressure development, heart rate etc, while subjecting the heart to a volume overload with saline infusion. What I found and reported at a meeting of the American College of Sports Medicine was this. In these previously untrained rats, 8 weeks of interval training was superior to 8 weeks of steady state training as an inducer of enhanced maximal cardiac performance. Interval trained hearts achieved higher peak stroke volumes during overload.

OK, So What?

Remember VO2 max? We have established that cardiac performance is a primary determinant of the VO2 max. The results of my thesis study suggested that interval training was the best way to enhance maximal cardiac performance, and therefore, presumably, VO2 max. In fact, in a study by a different laboratory, a significant increase in VO2 max occurred in previously untrained rats subjected to 5, very high intensity 1 minute bouts of exercise a day. That's 5 minutes of exercise. This improvement occurred without any

change in skeletal muscle oxidative capacity. There are other studies, on humans, that demonstrate the same finding.

Interval training allows us to accumulate a greater volume of stress on the blood pumping capacity of the heart. By using a large muscle mass, we promote maximal stroke volume responses. A high heart rate also is achieved as a function of the intensity. Finally, the periodic elevations and decreases in intensity may create special loading stresses on the heart that are adaptive. For example, during an interval, heart rate climbs high, then at the moment you stop the interval, heart rate immediately starts to drop, but venous return remains high. These exposures to additional ventricular stretch may help trigger ventricular remodeling (bigger ventricle volume). In addition, interval training may create a greater signal for changes in the compliance in the arterial system, but that is just more speculation.

OK then, ALL HARD INTERVALS ALL THE TIME, RIGHT?

NO, keep reading, the plot thickens. From what I have presented so far, and other research, I feel comfortable in saying that a program employing relatively low volume but high intensity endurance type exercise will be very effective in increasing the VO2 max of a previously untrained or (substantially detrained) person. For the untrained, interval training is a way of accumulating minutes of exercise at a higher intensity than our skeletal muscles are initially adapted to tolerate. In the untrained, the heart is better conditioned to endurance performance than the skeletal muscles. Therefore it needs a greater overload to adapt maximally. Intermittent high intensity training is also a powerful stimulus for increasing blood volume, which is a critical adaptation that contributes significantly to improved maximal cardiac output and VO2 max.

BUT, Here is the MAJOR PITFALL of THE ALL INTERVAL MENTALITY

The VO2 max is only the 1st wave of change ***(See subcategory-(6) above)*** for the endurance athlete. VO2 max plateaus quite early in the career of an adult runner or cyclist who trains hard and regularly (though its exact value will undulate several % from off-season to competitive season). SO, we have to ask "I am no beginner, is hard interval training also the best approach to improving the other components of my performance?"

Improving the endurance capacity of the skeletal muscles

Remember the 2nd Wave of Change? ***(See subcategory-(6) above)*** It is the improvement in lactate threshold, or the percentage of VO2 max that can be maintained without significant lactic acid accumulation. As, I have discussed previously, changes in the lactate threshold occur over a longer time-course than the improvement in maximal oxygen consumption, even if we train regularly. The site of adaptation moves from the cardiovascular sytem to the skeletal muscles. Additional mitochondria are synthesized, new capillaries are constructed, type II b fibers are converted to more fatigue resistant type II a. These adaptations occur progressively (again with seasonal undulation) over a period of several years. **The most powerful stimulus for change in skeletal muscle aerobic capacity is different from the most powerful stmulus for cardiac functional changes!** Hard but short interval training fails here. We MUST put in the hours of continuous constant intensity exercise to maximize these adaptations! This will range from steady state efforts at 65-75% of VO2max lasting 45 to 120 minutes to repeated "Anaerobic Threshold work" at 80-90% of VO2 max for 15 to 30 minutes.

Give Me More Evidence

Ok, you are one of these guys that likes to get in your boat or on your rowing machine every workout and hammer away for 500 to 2000 meters, then stop in the onrushing storm of lactic acid agony, only to repeat the process several more times after a few minutes rest. The workout leaves you exhausted, dry mouthed, and wobbly legged. Surely it will make you faster. You say, "Why bother training at less than race speed? If you want to race fast, you must train fast, always". Runners, Cyclists, swimmers, the same mentality can be found among you. Coming from a more speed and power oriented mentality, this was also my training inclination, for several years when I entered into endurance training. Heck, I was just making my interval trained rats do my workout! But, after reaching a plateau pretty quickly, I started looking, experimenting, and learning.

The German Rowers ease off the throttle

A few years ago, I came across extensive data collected on German national team rowers by their team of physiologists. They were regulary evaluated with blood tests during and after workouts. After accumulating a lot of measurements over a training year, some interesting results were reported. Eighty percent of the training volume among elite German rowers was

performed at a lactate concentration under 2.0 mM! (a value at or only slightly above resting levels) Only one or two percent of the training volume was at "RACE PACE". (Remember in competitive rowing, the events last 5.5 to 8 minutes, so race pace is above VO2 max.) From what I knew of their training back in the 50s and 60s, I had assumed that the Germans (and the Soviets) trained at brutal intensities, and those who didn't survive were replaced. Had the Germans become wimps? Well, actually at the exact time of these tests in the late 80s, they were the dominant world power in rowing with multiple world champions ranging across the boat class spectrum. So, whatever they were doing was working.

Why?

Why had German rowers, (and the Italians, another world power) adopted this training philosophy. Well, I dug up something interesting on that issue written way back in 1968 in a major rowing publication. Back in the early and mid 60s the German's training approach had indeed been much different, with a greater emphasis on high intensity intervals. What they found was that, to a great extent they did reach high performance levels with this training program. But, they were not seeing progressive improvement from year to year among their elite athletes. Every year they came up to the same level, fell back down in the off season, and repeated the process the next season. Then they changed the composition of the training to a higher volume, lower intensity (fewer killer intervals at max speed) and the long term progress began to occur. This makes sense when we consider the "Waves of Change".

Another reason relates to a different component of the physiological impact of high intensity intervals. Long term observation of elite German athletes has demonstrated (at least to them) that the body's tolerance for exposure to very high lactate concentrations and the extreme sympathetic stress associated with this type of training is quite limited. After a severe effort, immune function and other measures are disturbed for days. After a major international race, the disturbance can extend for weeks. Overtraining and tissue injury is much more likely during periods of extreme intensity. If training is interrupted due to injury or staleness, then no progress is made. We must be careful as coaches and athletes to apply the highest intensity work in careful, infrequent doses.

The Kenyans

There has been a tremendous amount of press in the running world about the recent dominance demonstrated by east Africans, particularly the Kenyans.

Not surprisingly, some have suggested that these runners are "genetically different" or train in "secret ways" that make them invincible in world distance running.

Well the Kenyans ARE setting world records right and left and kicking the rest of the running world's proverbial butt (Number 1, 2, 3, 4, 5 in 1996 Boston marathon). But do they have any physiological or training secrets? Well, to address that question, Dr. Bengt Saltin, formerly at the Karolinska Institute in Sweden, and now at the Copenhagen Muscle Research Center (Denmark), performed some interesting studies and published them in 1995. He measured maximal oxygen consumption, lactate threshold, economy, fiber type, etc in the top 12 Swedish distance runners. Then he went to Kenya and did the same thing among some of their best runners (with the considerable help of the famous runner of the 60s, Kipchoge Keino). On average, Kenyan and Swedish runners had high and similar VO2 max values (The very best Kenyans were somewhat higher than the very best Swedes, though). However, the really noticeable differences between the dominant Kenyans and the "also ran" Swedes was in their very low lactate and ammonia accumulation at high running speeds, and their excellent running economy. Remember the 2nd and 3rd Wave of Change?

Do the Kenyans do intervals?

Almost never. They do train intensely and often though. Here is what Saltin reported among a group of very successful younger runners. The runners train twice a day, morning and afternoon. 90% of the morning runs are through mountains and villages for 8-15 km (5-10 miles) at a moderate intensity (70 to 79% of VO2 max.) The other 10% are at a lower intensity. Then in the afternoon, they run again. This time the distance is only 3.5 to 5 miles, but 80% of these runs are at close to 90% of VO2 max. The other 20% are at very low intensity. If they feel tired they don't run as hard. This doesn't sound very complicated. Out of twelve workouts in a week, only one is an interval session, at 96% of VO2 max. A recent article about the great marathoner Cosmo Ndeti confirms this training strategy even among the older champion runners. The Kenyans definitely train hard, but they never do interval training at paces above VO2 max. As they improve, they run **longer** at the same relative intensity.

What about us Older Guys?

One of the goals of the Masters Aging and Rowing Study has been to evaluate how older elite athletes train. Fortunately I have received extensive training data from some excellent masters rowers, including national rowing champions and ergometer age group world record holders. The pattern I have observed is this: Most masters athletes don't train twice a day, not even quite every day. That's what having a full time job does for you, (and perhaps the wisdom that allows the mind to listen to the body when it says "I need a day off".) But, the best rowers still put in a lot of hours doing moderate to hard steady state rows on the water or on the erg. "Hours of Power" I call them. One ergometer rower who briefly held the world record in the men's 50+ age group reported 140 rows of one hour continuous on the ergometer in the year prior to his world record. Assuming race pace in his specialty is at 102 to 105% of VO2 max, the intensity of these rows was at about 73-75% of his 2500 meter race power, based on the meters rowed each session. This pace approximates 75-80% of VO2 max. Almost no training at higher intensities was performed until the weeks prior to a series of Spring races. Then he proceded to break 7:56 for 2500 meters 4 times in one month, culminating with a 7:52 world best! He built his performance engine with long steady state training, then he unleashed its impressive capacity in the brutal 8 minute races. It is also worth pointing out that his performances have steadily improved over about 5 years, despite being "middle aged". (He had discovered rowing in his 40s)

OK, No Intervals Any of the Time?

Not exactly. Here is what I think, based on the data and my own experience. **Interval training at above 100% VO2 max will not provide additional stimulus for improving maximal aerobic capacity, or lactate threshold, and may hurt.**

There is substantial research to indicate that there is little or no difference in the impact on maximal oxygen consumption among exercise intensities ranging from 80 to 100% of VO2 max. At intensities above 100%, the stimulus for improving maximal oxygen consumption is actually reduced, due to dramatically decreased training volume, and the inhibiting effect of lactic acidosis on cellular oxygen utilization.

During work within the 75-100% intensity range, improved exercise tolerance should be compensated for by increasing distance or time, not

intensity. This increased tolerance for exercise at a given submaximal intensity is indicative of skeletal muscle adaptations.

Aerobic interval training is definitely effective, but the emphasis should be on interval durations of from 10 to 20 minutes if your event duration is greater than 4 minutes. Intervals of 4 to 8 minute duration are going to be at intensities of 85 to 100% of VO2 max. This intensity and duration is optimal for maximizing/maintaining cardiovascular power. However, remember that this adaptation plateaus "early". Your incorporation of 4-8 minute intervals probably needn't ever exceed once every 8 days. Consequently, the majority of your training time should be aimed at optimizing the signal for other adaptations. Longer intervals of 10 to 20 minute duration will be at 75 to 85% depending on your abiity and training status, and are a very useful method for developing skeletal muscle endurance, "the Second Wave of Change". With both intervals the duration will generally force you into the right intensity range, if you are giving a good effort. The intermittant nature will help you to accumulate minutes of high quality work. I think 24 to 32 minutes of accumulated interval time is appropriate for most of us when performing the 4 to 8 minute intervals There will be little or no benefit from longer sessions. For the longer, lower intensity intervals I think 40 to 60 minutes is ideal. Twenty to 40 minutes will probably do most of the job. This means that on most of those busy days when you only have 30 minutes to train, a brief warm-up and one hard 20 minute steady state interval (temp run, AT effort, etc.) is more beneficial than resorting to a series of very short intervals. Rest intervals should not need to be very long, since lactate accumulation is not severe. For example, if you are attempting to do 5 times 5 minutes, a rest interval of at least 2 minutes but no more than 4 minutes should be used. For 20 minute intervals, rest will only be 5-6 minutes. If the intensity is appropriate, for a given interval session, you should be able to complete all the intervals at the same speed, albeit with increasing effort. Basically the long term goal is for the interval intensity to slowly become a steady state pace. In their excellent book "Training Distance Runners", David Martin (exercise physiologist) and Peter Coe (father and coach of world record holder Sebastion Coe) talk about interval training. They suggest that you should even be able to increase the speed of the last one or two intervals (with a lot of effort). This will indicate that you have not accumulated excessive lactic acid in the training session. If you are blowing out the first one and find yourself fading on subsequent intervals or cutting your workout short, then you are clearly not performing at the right intensity. More importantly you are NOT optimizing the training stimulus! You suffer, but the value of the session is compromised in two ways. First, the high lactate levels that develop due to excessive intensity actually inhibits oxygen utilization and

decrease fatty acid utilization by the mitochondria. Second, the prematurely abrupted workout leaves you exhausted, but your muscles under stimulated.

What is your favorite workout?

In the last two years, I found that a workout consisting of three 20 minute bouts at "friendly race pace" or just above my lactate threshold (my high lactate steady state), was my bread and butter workout in rowing. These sessions were performed once a week, always in the company of other single scullers. The duration keeps the intensity from getting too high. The competition with my partners kept it from getting too low! Other workouts were steady state rows at lower intensity for 60 to 90 minutes, with technical excellence (efficiency) being a primary objective along with aerobic conditioning. This was a significant change from a training program that revolved around 1, 2 and 4 minute interval sessions as the "Killer workout" each week. We did some of these short intervals also, but only as the race season approached and even then, less frequently than we had in previous years. All of my rowing partners found this training program to be excellent. We stayed healthy. We stayed motivated. And we got faster!

Is there ever a place for the really short, fast stuff ?

Yes, if your event duration is in the 3-4 minute range (1000 meters for rowers), then ANAEROBIC CAPACITY ***(see subcategory-(4) above)*** will play a contributing role. Even in these short events, aerobic endurance is still the foundation of success, but your ability to tolerate very high lactic acid levels and maintain good technique (rowers, swimmers) is critical over the concluding moments of the race. In this case we employ Anaerobic Intervals Here the method and goal is very different, and this must be recognized. In these sprint intervals, 30 seconds to 2 minutes in length, **1) a severe accumulation of lactic acid is expected.** Also, **forget your heart rate monitors.** What they read means nothing under these conditions. Rest intervals may be up to twice as long as work intervals. Intensity will be at 105 to 120% of VO2 max pace. **Lactic acid will not be completely eliminated during the rest, so each interval will start at a higher blood lactate.** The goal is to sustain speed as best as possible at higher levels of lactate. Here the total work time may be as little as 6 minutes to as much as 16 minutes. The value of these workouts diminishes once you can no longer maintain adaquate technique.

The **limited** potential for amplification of lactate accumulation capacity and tolerance comes at a high price within the overall endurance athlete's training

program (remember the German data). These adaptations are also achieved much faster than aerobic adaptations, so sprint training need not be implemented before 4-6 weeks out from a specific competition. It doesn't take too many brutal sprint sessions to have you or your athletes broken down at the side of the road/river/pool. For most endurance athletes, this mode of training should not account for more than 1 of every 10 workouts. More will put you at risk of becoming physically stale and psychologically brittle, and detract from the volume of training dedicated to aerobic adaptations.

Where does my heart rate monitor fit into all this?

I would recommend using the heart rate reserve formula. ***(see subcategory-(1.a.ii) above).*** This will allow you to come close to establishing a specific heart rate for the percentage VO2 values I am discussing here. Of course the heart rate tachometer only goes to 100%. That is max heart rate and will correspond to 100% of VO2 max. In general, heart rate does not reflect work intensity adequately for high intensity intervals (30 seconds to 3 minutes)

SUMMARY

In past articles in this section, I have discussed the major components of endurance performance. They are maximal oxygen consumption, lactate threshold, and efficiency. A friend of mine has added an article on short term muscular power (Anaerobic capacity). This last component plays a negligible role in long events but becomes increasingly important as the race distance decreases below 15 minutes. Anaerobic energy sources may account for 20% of the energy output in a 5-7 minute race. In a 2 minute race, about 40 % of total energy is being supplied anaerobically.

I have also discussed the timecourse of change of these components in the endurance athlete who continues training for several years. Now, in this article I hope I have added to this foundation by discussing the relationship between training characteristics and the impact on those components. Understanding the underlying physiology now helps us to put the training program together. Short, very high intensity interval training has only a small niche in the endurance athlete's training program. It feels like a race. It hurts like heck. But, it has limited value in building the performance engine. I am convinced that our focus must be on longer bouts of exercise as the foundation of our training program. This is easily understood by you marathoners, but may be surprising news to you 1000 meter rowers, middle distance runners and pursuit cyclists. The bigger take home message is this: Even for you "power-endurance

athletes", it is the consistency and volume of exercise at 70% to 90% of VO2 max in your training program that is going to have the single biggest long term impact on your progress, not the number of workouts in which you achieve complete rigor mortis and cotton mouth!

8) WHERE ARE THEY NOW?
Report on a 22 year Longitudinal Study of Elite Distance Runners

You Sure Can Get Old Studying Aging!

In research involving age related changes in physical function, there are two basic types of studies. The first and most common is the CROSS SECTIONAL study. In this type of work, an investigator recruits groups of people of different ages and/or activity levels and makes comparisons. In a sense, we are trying to take a snapshot of each group. For example, the MARS study that I have been working on is comparing the training habits of rowers in their 40s, 50s, 60s and 70s by decade. This is a reasonable approach (and the only one when the age difference is 40 years!) and we are learning a great deal of information. However, there is another way. This is the LONGITUDINAL STUDY. Here we examine a group of people at time A. Then we wait, and wait . . . and wait. Eventually time B comes along and we reevaluate the same people. To be honest this type of study isn't very good for a young scientist's career. They take too long! (unless you study aging in insects or rats since they have short life-spans.) But, they are an ideal method. Fortunately, we have a few "senior" exercise physiologists around the world who began studying elite athletes before you could even earn a Ph.D. in "exercise physiology". Dr. David Costill is one of those few.

My introduction to Dr. Costill first came through a book by the late Jim Fixx called "The Complete Book of Running", written in the late 70s. One of the last chapters was called "The Scientists of Sport", and profiled the developing work of Dr. Costill. During that time he tested over a hundred elite runners. He measured maximal oxygen consumption, economy, fiber type, etc., all variables that we now know contribute to endurance performance. Well, at that time I was a kid who went to 8th grade track practice in the morning, then worked in my own private self-built laboratory at night. After reading this chapter, my future became clear. I would be one of those new "sport scientists".

It has been over 15 years since then. I have earned a Ph.D. and am still in love with this exciting field of exercise physiology (but I do not miss 8th grade track practice!) I have now met Dr. Costill in person several times. And he is still doing research. Now, in 1996 he (and his colleagues) has published something else that really caught my eye. Over 50 of those elite runners of the late 60s and early 70s recently returned to his lab to undergo the same tests they

performed over 2 decades ago. This study provides us with one of the rare long term longitudinal studies of physical performance in well trained individuals. Since I kind of owe a debt to Dr. Costill from years ago, and feel this study speaks directly to many of the issues I talk about on the MAPP, I want to give it a pretty close look.

This is the reference for those who have access to the source:

Journal of Applied Physiology 80(1):285-290, 1996
Aging among Elite Distance Runners: A 22-yr Longitudinal Study
SW Trappe, DL Costill, MD Vukovich, J Jones, & T Melham.
Human Performance Laboratory, Ball State University, Muncie, Indiana
THE SUBJECTS- Description of the groups

Fifty three men were recruited from Dr. Costill's data base of over 100 runners tested in the late 1960s and early 1970s. The average age of the group when they were first tested was 29.4 years, but ranged from 18 to 55 years. From this group, four subgroups were selected based on their activity level over the intervening 22 yr. (average) period. The group selections were made based on interviews and questionnaires. The four groups were divided as follows: 1) those who continued to train intensely and compete in age group competition (10 men). We will use the shorthand he used and call this group **HT, (highly trained).** A second group was called **FT (Fitness trained)**. These 18 men continued to exercise over the 22 years but did so only for physical fitness. A third group (15 men) consisted of those who had not been regularly active for the last 5 or more years. This group was called **UT (untrained)**. Finally a fourth group was composed of older athletes who were in their mid 40s when first tested and had continued exercising for physical fitness They were called **FO (fit older).** These men were now all over 60 and more than 20 years older than the members of the other three groups. However, they had maintained a high level of fitness over the last 20-25 years.

TRAINING HISTORY

Us endurance guys like to keep training logs. These guys were no different. So this information was used to make comparisons of training volume, intensity and duration back in the 70s, in the intervening years, and currently. I have taken the liberty of reproducing that data (and a lot more to come) as reported in their research article in the Table below.

GROUP	Training Distance (km/week)	Training Pace km/hr	Training Frequency sessions/week
		Initial Period (1966-1976)	
HT	125.5	14.1	6.3
FT	98.7	13.7	6.4
UT	94.5	13.9	6.4
FO	62.7	13.6	6.1
		Intervening Period (1977-1987)	
HT	92.5	13.6	6.1
FT	66.8	12.5	5.5
UT	NT	NT	NT
FO	46.0	11.1	4.9
		Recent Period (1988-1993)	
HT	71.4	13.5	6.1
FT	43.7	12.2	5.5
UT	NT	NT	NT
FO	23.4	9.6	4.1

Back when these athletes were first tested, they were all training for competition at the local, collegiate, national, or international level. One thing is clear. All of them have decreased their training load. Even the HT group has decreased training volume by over 25%. However, the hard training group has maintained very similar intensity and frequency levels, compared to 20 years ago. It is also interesting to note that those guys who were putting in the most miles then are still putting in the most miles.

Training Status and Body Composition

The first "physiological" data we will look at are body fat and muscle mass changes among the groups as reported in the study. Sad to say, everyone gained some weight, but not everyone gained the same amount. Not surprisingly, the guys that stopped training altogether gained the most weight. And it wasn't muscle. The UT group went from 145 to 178 pounds (66 to 81 kg). Their bodyfat percentage rose from 9% to 22%. In contrast, the hard training (HT) group gained much less fat. Their bodyfat rose from 7.5% to 12.5%. The FT and FO groups were both in the middle. Their body fat rose almost identically from about 10 to about 17%, 22 years later. One other point; **The older guys also lost some muscle mass (about 7 pounds)**, while the

other three groups did not. **This supports the notion that running alone is not sufficient to prevent the significant muscle atrophy that is known to occur after age 50-60.**

VO2 max, Heart Rate max etc. Then and Now (22 years later)

	UT (n=15)		FT (n=18)		HT (n=10)		FO (n =10)	
Variable	Then	Now	Then	Now	Then	Now	Then	Now
VO_2 max L/min	4.61	3.87	4.51	3.76	4.57	4.17	3.98	2.74
Percent Decline	18%		17%		**9%**		31%	
VO_2 max ml/min/kg	70.7	46.7	64.1	48.9	69.8	59.2	60.3	40.7
Percent Decline	34%		24%		**14%**		33%	
Maximal Heart Rate	187	178	186	174	191	180	175	155
O_2 Pulse	24.6	21.3	24.3	21.7	24	23.2	22.9	17.8

Maximal Oxygen Consumption

The first thing that jumps out at you from the table above is the dramatic decline in maximal oxygen consumption in those former elite runners who have become sedentary. "Use it or Lose it" definitely applies here. Their absolute VO2 declined 18%. However, because of their weight gain over two decades, their VO2 per kg bodyweight declined 34%! This 17% decline per decade is actually greater than the 10%/decade typically observed in the adult population. This is due to the fact that they had attained a highly trained state before becoming sedentary. In contrast, the absolute decline in maximal oxygen consumption was half as much in the hard trainers (9%, or about 5%/decade). And because of their lower weight gain, the decline in V02 max factored for bodyweight was also much less pronounced. Within the HT group, there were two athletes whose results were almost identical (2% decline) to those from 20 years before. Not surprisingly, their training level had been the most consistent from past to present. The decline observed in the Fitness Trained (FT) group was also greater than HT. This is explained by the significant decrease in intensity and volume of training by this group. Finally, it is difficult to interpret the results from the Fitness trained old group (FO). Their VO2 max declined more dramatically during the 22 year period, but so did their training level.

Maximal Heart Rate and Stroke Volume

Maximal heart rate

Why does VO2 max decline? Remember the central role of heart function. Maximal cardiac output decreases with age, primarily due to a presumably linear decline in maximal heart rate. As discussed before, this is a physiological change that does not appear to be responsive to training. The HR max of the hard training group decreased just as much as that of the other same age groups (FT and UT). The decline in maximal heart rate was more dramatic in the older athletes over the same period (FO). Why this is true is unclear, but does help explain their more significant loss of aerobic capacity. Looking at the three "Young" groups only, it is also worth noting that the rate of decline in max heart rate was less (by almost 50%) than what would be predicted using the standard **220 - age formula**. The data above supports the use of a formula that predicts a lower heart rate decline between age 25 and 50, followed by a more dramatic decline after age 50. This has been previously recommended by some others. There are at least a half dozen formulas for predicting maximal heart rate across age. Clearly they are all estimates. Perhaps one day, I will gather all of these in one place. For now, just pick one you like.

Stroke Volume

The other component of cardiac output is stroke volume. This was not directly measured in this study. However, we can make some estimation of relative changes in stroke volume by looking at **oxygen pulse**, or oxygen consumption/ heart rate. In the data above we see that this index of stroke volume declined most dramatically in the groups that trained the least. The hard training group maintained oxygen pulse at near "young" levels. This makes sense physiologically. It is stroke volume that is increased most in the endurance athlete. Maintained high intensity training would be expected to help maintain a high stroke volume. It would have been interesting to compare resting heart rates then and 20 years later, but this data wasn't reported.

Muscle endurance capacity

Muscle biopsies were taken from the gastrocnemious (calf) muscle of the runners during the follow-up test only. As we would expect, the group with the highest training level had the highest activity of the mitochondrial enzymes citrate synthase and succinate dehydrogenase (two common markers for total

mitochondrial volume.) The untrained group was the lowest. This decline in muscle oxidative capacity would also be expected to contribute to the decline in VO2 max. However, it would contribute even more to a decline in lactate threshold. Unfortunately, this component of performance potential was not measured.

Running Economy

One other interesting component of this study was the measurement of physiological responses to a standard submaximal running intensity both in the 70s and 22 years later. All of the younger runners had performed several bouts of treadmill running at different speeds back when they were younger, to examine running economy. Two decades later the higher speeds could not be maintained by all the groups. However, many could still run at 12 km/hour without exceeding their VO2 max. So physiological responses at this running speed were compared then and now on 24 of the 53 subjects. All of the hard training group could maintain this speed. So what you will see below is the response to running at a standard speed of 12 km/hour (or about 8 min mile pace). From this data we will be able to discuss the issue of running economy, aging and training status.

	UT (n=6)		FT (n=8)		HT (n=10)	
Variable	THEN	NOW	THEN	NOW	THEN	NOW
VO_2, l/min	2.65	3.39	2.78	3.10	2.63	2.71
VO_2, ml/min/kg	40.3	41.8	39.5	40.1	39.4	38.5

Ok, here everyone was running at precisely the same speed and under the same conditions. However, the physiological cost of maintaining that speed was quite different among the groups. In the UT group, we see from the last two tables that becoming sedentary has not only decreased their VO2 max, it has also resulted in a loss of running economy. Running at the same fairly slow speed now requires 24% more oxygen! As a result, the sedentary guys were now using 78% of their maximal oxygen consumption to run 8 minute miles, compared to only 54% twenty years before! This is the difference between a leisurely jog and an above lactate threshold, exhausting effort. What is the cause for the increased energy cost of running? Primarily it is due to weight gain. When we factor out bodyweight as the authors did (VO2/ bodyweight), then running economy still decreased by 4%, a statistically

significant change in this study. I have gone one step further. When I scaled bodyweight allometrically to more accurately reflect the actual impact of body size on running cost, then the weight independent difference in economy is even greater, 9%. In contrast, the athletes who have continued training at high levels, experienced almost no change in running economy based on absolute changes in oxygen consumption. If anything, they have become slightly more economical compared to their elite days! So, thanks to both weight gain and some unspecified changes in biomechanics or muscle composition that have occurred over the years, the sedentary guys are now 20% less efficient compared to their hard training buddies! The fitness trained (FT) guys also have maintained better running economy than the untrained, but not as good as those who have maintained high levels of training. Once again, they are stuck in the middle of the pack.

Summary

This study substantially supports the findings of several other shorter term longitudinal studies of masters athletes as well as several good cross sectional studies. The main point is of course, **a continued high level of training can significantly reduce the magnitude of VO2 decline that inevitably occurs with aging.** Previously, a 9-10% decline in maximal oxygen consumption/decade has been suggested from studies of untrained healthy men. As suggested by other studies, the results above suggest that the rate of decline is halved (5% /decade and in some cases even less) in athletes who maintain a very high level of training volume and intensity. As we have discussed before. Continued training can maintain stroke volume at high levels, as well as skeletal muscle endurance capacity. Maximal heart rate decline with age, on the other hand, is not altered by activity level.

Lactate threshold intensity was not determined in this study. However, previous investigations have concluded that LT can be maintained if training intensity and volume are maintained.

The third component of the "BIG THREE" endurance performance components that I have discussed on the MAPP is performance efficiency. The results of Dr. Costill and colleagues' study suggest that running economy is maintained (at least at training speeds or below) if weight gain is avoided and training volume is maintained at high levels. In contrast, even when former elite distance runners stop running, they become less efficient runners. These data support the notion that some aspects of running economy are not in-born, but respond to training!

9) STRENGTH TRAINING AND ENDURANCE PERFORMANCE

In endurance performance, we are limited by weak links in the physiological system. Making an already strong link stronger doesn't keep a chain from breaking if you still have the same weak link. Throughout the MAPP, I have repeatedly discussed maximal oxygen consumption, lactate threshold and efficiency of movement as major components of any endurance performance. We have talked about the heart and how it responds to training. We have discussed the skeletal muscles and their primary adaptations. Now I want to think out-loud a little bit about how, or IF strength training fits into the endurance athlete's training program.

First let's define strength training. For the endurance athlete, I will call any exercise that is designed to increase the size and/or maximal strength of a muscle or group of muscles strength training. Many endurance athletes are lifting weights 2 to 3 times per week and swear by it. Others never lift a weight and excel.

Second, let's make sure we understand that strength training for health versus strength training for enhanced performance are two different beasts. I think there are excellent reasons to strength train for health. As we age (especially beyond 50 or so) our bodies tend to lose muscle mass. Retarding this change is definitely beneficial. Maintained or increased muscle mass helps to prevent body fat accumulation, maintain functional mobility, decrease risk of adult onset diabetes etc. From here out I am speaking only about PERFORMANCE!

Big Versus Little - Muscles in Isolation

Let's say that we remove a biceps muscles from two different endurance trained rowers. One muscle is 50% bigger than the other (cross-sectional area). We hook up these muscles to an artificial machine and perform a test (I know this is gross but it is just a hypothetical situation. We would never do this to well trained rowers!) Which muscle will be able to perform more work in a 6 minute all out test? Well, the bigger one will, of course. That is, assuming that both muscles are well adapted to repetitive work (lots of mitochondria) and both are receiving plenty of oxygen. So if all other things are equal, the big muscle outperforms the small muscle.

Big versus Little - Muscles as part of a Package

If we extend the above situation to say a big body builder and a skinny guy like me, does the bodybuilder win? Probably not (I hope not at least). Why? Because now the rules have changed, or I should say the performance limitations have changed. In the isolated muscle above I said that the muscles were 1) equally endurance trained and 2) supplied with unlimited oxygen. When we put the muscles back inside a real body, neither of these conditions are true.

Mitochondrial Dilution

When a bodybuilder trains, the goal is to make each muscle fiber as big as possible. Muscle fibers have contractile protein, mitochondrial protein, and other components. Increasing the relative proportion of one component (like more contractile protein) means that you have relatively less of everything else in the same fiber (like mitochondria). From an endurance standpoint this is not a good adaptation. We even give it a name in sports physiology circles, mitochondrial dilution. The bodybuilder's muscles may actually become more easily fatigued as they get bigger, because their mitochondrial density is not increasing at the same rate. The bodybuilder accepts that because the name of the game is size, not endurance.

It is possible for the endurance athlete to gain some muscle size and maintain mitochondrial density, but it requires that the volume of endurance training be maintained. If you are a runner and you decide to get stronger in the weight room by really doing a lot of strength training 3 days a week for an hour, you will probably drop some of your running volume to fit it in. After 6 months you have gained 5-10 pounds of muscle, you look really good, and you are running 2 minutes slower for 10k! Why? Well besides having to carry around 5-10 more pounds of muscle that you can't use when you are running, you have probably lost endurance capacity in those bigger stronger quads. So, you have a lower lactate threshold due to the detraining of your leg muscles, plus you are less efficient due to the increased bodyweight (and decreased training volume). Oh well, at least you LOOK Fast.

But I have read that strength training helps endurance performance !?

It can, definitely. But we have to look at the reasons more carefully. For example, perhaps you are a runner who has had a hip injury that lingered and lingered. Over time your running style accommodated and now you run with a "seated" style and do not employ your hip extensors effectively. A weight training program employing highly specific exercises designed to teach you to

activate your hip extensors, as well as strengthen them, can make you more efficient by improving work distribution in the leg muscles, and therefore faster runner. A lot of this change may be due to improved motor function as much as increased muscle mass. Specific strength training can help us to teach our brain to communicate with the right muscles. The same is true of the rower with the weak low back. Strengthening this area can correct the weak link and allow optimal connection between force generators and the oar. However, the concept that just making muscles bigger and stronger will automatically translate to faster endurance performance is Wrong!

Oh yea, What about Oxygen?

Remember VO2 max? What is the major limitation to VO2 max? Right, the maximum capacity of the heart to pump blood and deliver oxygen to the muscles. Anytime we are doing an activity that uses a lot of muscle (running, rowing, XC skiing, mountain biking), the challenge falls on the heart to match oxygen supply with the demand. Even in the world champion, the heart is incapable of pumping as much blood as the muscles could receive.

Consequently, adding muscle mass will not result in increased maximal oxygen consumption. The heart is already being asked to do all it can do. Your endurance machine is a set of highly integrated components. You have to look at how all the pieces fit together to produce the final product.

Learning from the Para-Olympians

Now having said all of that, here is a thought question for you. Watch an elite marathoner run across the finish line. He or she raises toothpick size arms into the air in victory and jogs off on those skinny but brilliant legs. No excess muscle there. Just the right amount to get the job done.

Now take a look at another marathon race, this time the wheelchair race for para-athletes. The winner rolls across the line with a final push of the arms, raises them in victory and you are caught staring at one impressively muscular set of pythons! Is this the marathon or the bench press? What gives? For the wheelchair endurance athlete, muscle mass is an important part of the package. The reason for this apparent contradiction in everything I have said goes back to the HEART.

The wheelchair racer is depending on a much smaller total volume of muscle to do the work of the marathon race. The total volume of muscle is small

enough that the heart is no longer the limiting factor! So, in this situation, gaining muscle mass in combination with endurance training results in a more powerful endurance engine. In fact, these unique conditions may result in a greater hypertrophic response to endurance training independent of supplemental strength training in a weight-room

Applying the Lesson

This example above came to me in a strange way. I got a message from an Australian who was familiar with the lifeguard boat races down under. He said these guys had bigger upper bodies than "regular" rowers and were very strong, but not quite as good on the ergometer. I had to think about that a bit to decide what it meant. Then I remembered "They don't have a sliding seat!" Which means of course that the legs are taken out of the game and rowing becomes an upper-body only sport. Hence the bigger upper body just like the wheel chair athletes. So, as I thought about that while driving up to Lillehammer for my first mountain bike race, the wheel chair scenario hit me, and I understood things better. I was pretty excited!

Now what can we learn from this? If you are in a sport like kayaking that is a small muscle mass endurance sport, then strength training plays an very important role because it helps to increase the size of your endurance machine. If you are a runner, then you will not benefit from the same volume of strength training and may actually lose speed. Running already employs a large mass of muscle that can work at a level that exceeds the oxygen delivery capacity of the heart.

Now, if you are a cross country skier, you have a unique situation. Your sport often requires that you use a lot of muscle simultaneously, making the heart the limiting factor and excess muscle mass wasteful. However, when you are double poling, the conditions change and the mass of endurance-trained upper body mass that you can engage becomes very important. Double poling is efficient and important in ski racing. So for the skier, strength training is far more important for the upper body than for the lower body. And for women, it is even more critical. The reason is that women start with a bigger gap between upper and lower body strength then men. Here in Norway, the elite junior women invest a lot of energy doing things like uphill double poling to strength train the upper-body in a highly specific way. They have much to gain by strength training.

If you are a rower, I am not sure what to tell you exactly. Increased upper body strength may allow better work distribution and therefore slightly improved rowing economy but I don't know that for sure. The act of rowing training already improves the rower's ability to generate force with both legs simultaneously compared to untrained people. Much of the rowers strength depends on coordination, not just muscle mass. Rowing has a mixed tradition when it comes to strength training. Some great programs do a lot, others do none. So the jury is still out. More on all this when I can be more definitive.

10) MUSCLE FIBER HYPERTROPHY VS HYPERPLASIA
Has the debate been settled? by Jose Antonio PhD

editors note: One of the fundamental questions in exercise physiology has been the mechanism of muscle adaptation to increased force demands (i.e. strength training). The simple and generally correct answer remains that muscles grow in size due to the growth of existing muscle fibers. However, under extreme conditions of muscle size and workload, there is substantial evidence that muscles can take advantage of a more spectacular mechanism; they can split to form additional new fibers, a mechanism termed hyperplasia. Dr. Antonio has been at the center of this controversial research and did his doctoral work in this area. I think this article is an excellent resource for beginning exercise physiology student and an interesting glimpse into the challenges of physiological research for all. His contribution adds significantly to the teaching value of this site.
-- Stephen Seiler

WHAT IS HYPERPLASIA?

Hypertrophy refers to an increase in the size of the cell while hyperplasia refers to an increase in the number of cells or fibers. A single muscle cell is usually called a fiber.

HOW DO MUSCLE FIBERS ADAPT TO DIFFERENT TYPES OF EXERCISE?

If you look at a good marathon runner's physique and compared him/her to a bodybuilder it becomes obvious that training specificity has a profound effect. We know that aerobic training results in an increase in mitochondrial volume/density, oxidative enzymes, and capillary density (27). Also, in some elite endurance athletes the trained muscle fibers may actually be smaller than those of a completely untrained person. Bodybuilders and other strength-power athletes, on the other hand, have much larger muscles (14,40). That's their primary adaptation, their muscles get bigger! All the cellular machinery related to aerobic metabolism (i.e. mitochondria, oxidative enzymes, etc) is not necessary for maximal gains in muscle force producing power, just more contractile protein. We know that this muscle mass increase is due primarily to fiber hypertrophy; that is the growth of individual fibers, but are there situations where muscles also respond by increasing fiber number?

EVIDENCE FOR HYPERPLASIA

Scientists have come up with all sorts of methods to study muscle growth in laboratory animals. You might wonder what relevance this has to humans. Keep in mind that some of the procedures which scientists perform on animals simply cannot be done on humans due to ethical and logistical reasons. So the more convincing data supporting hyperplasia emerges from animal studies. Some human studies have also suggested the occurence of muscle fiber hyperplasia. I'll address those studies later.

DOES STRETCH INDUCE FIBER HYPERPLASIA?

This animal model was first used by Sola et al. (38) in 1973. In essence, you put a weight on one wing of a bird (usually a chicken or quail) and leave the other wing alone. By putting a weight on one wing (usually equal to 10% of the bird's weight), a weight-induced stretch is imposed on the back muscles. The muscle which is usually examined is the anterior latissimus dorsi or ALD (unlike humans, birds have an anterior and posterior latissimus dorsi). Besides the expected observation that the individual fibers grew under this stress, Sola et al. found that this method of overload resulted in a 16% increase in ALD muscle fiber number. Since the work of Sola, numerous investigators have used this model (1,2,4-8,10,19,26,28,32,43,44). For example, Alway et al. (1) showed that 30 days of chronic stretch (i.e. 30 days with the weight on with NO REST) resulted in a 172% increase in ALD muscle mass and a 52-75% increase in muscle fiber number! Imagine if humans could grow that fast!

More recently, I performed a study using the same stretch model. In addition, I used a progressive overload scheme whereby the bird was initally loaded with a weight equal to 10% of the its weight followed by increments of 15%, 20%, 25%, and 35% of its weight (5). Each weight increment was interspersed with a 2 day rest. The total number of stretch days was 28. Using this approach produced the greatest gains in muscle mass EVER recorded in an animal or human model of tension-induced overload, up to a 334% increase in muscle mass with up to a 90% increase in fiber number (5,8)! That is pretty impressive training responsiveness for our feathered descendants of dinosaurs.

But you might ask yourself, what does hanging a weight on a bird have to do with humans who lift weights? So who cares if birds can increase muscle mass by over 300% and fiber number by 90%. Well, you've got a good point. Certainly, nobody out there (that I know of), hangs weights on their arms for 30 days straight or even 30 minutes for that matter. Maybe you should try it and

see what happens. This could be a different albeit painful way to "train." But actually the physiologically interesting point is that if presented with an appropriate stimulus, a muscle can produce more fibers! What is an appropriate stimulus? I think it is one that involves subjecting muscle fibers to high tension overload (enough to induce injury) followed by a regenerative period.

WHAT ABOUT EXERCISE?

The stretch induced method is a rather artificial stimulus compared to normal muscle activity. What about "normal" muscular exercise? Several scientists have used either rats or cats performing "strength training" to study the role of muscle fiber hyperplasia in muscular growth (9,13,17,18,20-22,25,33,34,39,41,42). Dr. William Gonyea of UT Southwestern Medical Center in Dallas was the first to demonstrate exercised-induced muscle fiber hyperplasia using weight-lifting cats as the model (20,21,22). Cats were trained to perform a wrist flexion exercise with one forelimb against resistance in order to receive a food reward. The non-trained forelimb thus served as a control for comparison. Resistance was increased as the training period progressed. He found that in addition to hypertrophy, the forearm muscle (flexor carpi radialis) of these cats increased fiber number from 9-20%. After examining the training variables that predicted muscle hypertrophy the best, scientists from Dr. Gonyea's laboratory found that lifting speed had the highest correlation to changes in muscle mass (i.e. cats which lifted the weight in a slow and deliberate manner made greater muscle mass gains than cats that lifted ballistically) (33).

Rats have also been used to study muscle growth (25,39,47). In a model developed by Japanese researchers (39), rats performed a squat exercise in response to an electrical stimulation. They found that fiber number in the plantaris muscle (a plantar flexor muscle on the posterior side of the leg) increased by 14%. Moreover, an interesting observation has been made in hypertrophied muscle which suggests the occurrence of muscle fiber hyperplasia (13, 17, 28, 47). Individual small fibers have been seen frequently in enlarged muscle. Initially, some researchers believed this to be a sign of muscle fiber atrophy. However, it doesn't make any sense for muscle fibers to atrophy while the muscle as a whole hypertrophies. Instead, it seems more sensible to attribute this phenomenon to de-novo formation of muscle fibers (i.e. these are newly made fibers). I believe this is another piece of evidence, albeit indirect, which supports the occurrence of muscle fiber hyperplasia.

EXERCISE-INDUCED GROWTH IN HUMANS

The main problem with human studies to determine if muscle fiber hyperplasia contributes to muscle hypertrophy is the inability to make direct counts of human muscle fibers. Just the mere chore of counting hundreds of thousands of muscle fibers is enough to make one forget hopes of graduating! For instance, one study determined that the tibialis anterior muscle (on the front of the leg) contains approximately 160,000 fibers! Imagine counting 160,000 fibers (37), for just one muscle! The biceps brachii muscle likely contains 3 or 4 times that number!

So how do human studies come up with evidence for hyperplasia? Well, it's arrived at in an indirect fashion. For instance, one study showed that elite bodybuilders and powerlifters had arm circumferences 27% greater than normal sedentary controls yet the size (i.e. cross-sectional area) of athlete's muscle fibers (in the triceps brachii muscle) were not different than the control group (47). Nygaard and Neilsen (35) did a cross-sectional study in which they found that swimmers had smaller Type I and IIa fibers in the deltoid muscle when compared to controls despite the fact that the overall size of the deltoid muscle was greater. Larsson and Tesch (29) found that bodybuilders possessed thigh circumference measurements 19% greater than controls yet the average size of their muscle fibers were not different from the controls. Furthermore, Alway et al. (3) compared the biceps brachii muscle in elite male and female bodybuilders. These investigators showed that the cross-sectional area of the biceps muscle was correlated to both fiber area and number. Other studies, on the other hand, have demonstrated that bodybuilders have larger fibers instead of a greater number of fibers when compared to a control population (23,30,36). Some scientists have suggested that the reason many bodybuilders or other athletes have muscle fibers which are the same size (or smaller) versus untrained controls is due to a greater genetic endowment of muscle fibers. That is, they were born with more fibers. If that was true, then the intense training over years and decades performed by elite bodybuilders has produced at best average size fibers. That means, some bodybuilders were born with a bunch of below average size fibers and training enlarged them to average size. I don't know about you, but I'd find that explanation rather tenuous. It would seem more plausible (and scientifically defensible) that the larger muscle mass seen in bodybuilders is due primarily to muscle fiber hypertrophy but also to fiber hyperplasia. So the question that needs to be asked is not whether muscle fiber hyperplasia occurs, but rather under what conditions does it occur. I believe that the scientific evidence shows clearly in animals, and indirectly in humans, that fiber number can increase. Does it

occur in every situation where a muscle is enlarging? No. But can it contribute to muscle mass increases? Yes.

HOW DOES MUCLE FIBER HYPERPLASIA OCCUR?

There are two primary mechanism in which new fibers can be formed. First, large fibers can split into two or more smaller fibers (i.e. fiber splitting) (6,25,39). Second satellite cells can be activated (11,16,17,43,44).

Satellite cells are myogenic stem cells which are involved in skeletal muscle regeneration. When you injure, stretch, or severely exercise a muscle fiber, satellite cells are activated (16,43,44). Satellite cells proliferate (i.e. undergo mitosis or cell division) and give rise to new myoblastic cells (i.e., immature muscle cells). These new myoblastic cells can either fuse with an existing muscle fiber causing that fiber to get bigger (i.e., hypertrophy) or these myoblastic cells can fuse with each other to form a new fiber (i.e. hyperplasia).

ROLE OF MUSCLE FIBER DAMAGE

There is now convincing evidence which has shown the importance of eccentric contractions in producing muscle hypertrophy (15,24,45,46). It is known that eccentric contractions produces greater injury than concentric or isometric contractions. We also know that if you can induce muscle fiber injury, satellite cells are activated. Both animal and human studies point to the superiority of eccentric contractions in increasing muscle mass (24,45,46). However, in the real world, we don't do pure eccentric, concentric, or isometric contractions. We do a combination of all three. So the main thing to keep in mind when performing an exercise is to allow a controlled descent of the weight being lifted. And on occasion, one could have his/her training partner load more weight than can be lifted concentrically and spot him/her while he/she performs a pure eccentric contraction. This will really put your muscle fibers under a great deal of tension causing microtears and severe delayed-onset muscle soreness. But you need that damage to induce growth. Thus, the repeated process of injuring your fibers (via weight training) followed by a recuperation or regeneration may result in an overcompensation of protein synthesis resulting in a net anabolic effect (12, 31).

HAS THE DEBATE BEEN SETTLED?

In my scientific opinion, this issue has already been settled. Muscle fiber hyperplasia can contribute to whole muscle hypertrophy. There is human as

well as rat, cat, and bird data which support this proposition (1-3, 5-8, 13, 17, 20-22, 25, 29, 35, 37, 47), a veritable wild kingdom of evidence. Does muscle fiber hyperplasia occur under all circumstances? No. There are several studies which show no change in fiber number despite significant increases in muscle mass (4,18,19,23,26,30,36,41). Is it possible that certain muscles can increase fiber number more so than others? Maybe. Can any Joe Schmoe off the street who lifts weights to get in better shape increase the number of fibers for instance in their biceps? Probably not. What about the elite bodybuilder who at 5'8" tall is ripped at a body weight of 250 lbs.? Are his large muscles purely the result of muscle fiber hypertrophy? I think it would be extremely naive to think that the massive size attained by elite bodybuilders is due solely to fiber hypertrophy! There is nothing mystical about forming new muscle fibers. Despite the contention that fiber number is constant once you're born (18, 19), we now have an abundance of evidence which shows that muscle fiber number can increase. Besides, there is nothing magical at birth which says that now that you're out of the womb, you can no longer make more muscle fibers! A mechanism exists for muscle fiber hyperplasia and there is plenty of reason to believe that it occurs. Of course, the issue is not whether fiber number increases after every training program, stress, or perturbation is imposed upon an animal (or human). The issue is again, under which circumstances is it most likely to occur. For humans, it is my speculation that the average person who lifts weights and increases their muscle mass moderately probably does not induce fiber hyperplasia in their exercised muscle(s). However, the elite bodybuilder who attains the massive muscular development now seen may be the more likely candidate for exercise-induce muscle fiber hyperplasia. If you are interested in a comprehensive scientific treatise on this subject, read a scientific review article that I wrote a few years ago (7).

KEY TERMS

• **anabolic** - in reference to muscle, a net increase in muscle protein

• **catabolic** - in reference to muscle, a net decrease in muscle protein

• **concentric** - shortening of a muscle during contraction

• **eccentric** - lengthening of a muscle during contraction

• **hyperplasia** - increase in cell number

• **hypertrophy** - increase in cell size

• **isometric** - no change in muscle length during a contraction

• **mitochondria** - is an organelle ("little organ") found within cells and is involved in generating ATP via aerobic processes

• **muscle fiber** - also known as a myofiber; is the multinucleated cell of skeletal muscle

• **myoblast** - an immature muscle cell containing a single nucleus

• **myogenesis** - the development of new muscle tissue, esp. its embryonic development

• **satellite cell** - are the cells responsible in part for the repair of injured fibers, the addition of myonuclei to growing fibers, and for the formation of new muscle fibers.

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11) VENTILATION AND ENDURANCE PERFORMANCE
Do our lungs limit how fast we can go?

So far, on the MAPP I have devoted a lot of attention to two major organ systems that are intimately linked in exercise performance, the skeletal muscles and the cardiovascular system. Now I want to factor a third, equally important system into the equation, the pulmonary system. I am going to discuss breathing and exercise.

The term ventilation is used in physiology circles exclusively in reference to gas exchange in the lungs. You will also see the word respiration, but exercise physiology types often like to reserve this word for use in a cellular metabolism context, so I will try to use ventilation when I mean breathing. Now, when you hear a non-smoker trapped in a smoke-filled room gasping something about "this room needs better ventilation," she means that the room does not have very rapid air-exchange with the outside, smoke-free air. Conversely, in a room that is well-ventilated, the air always seems fresh even when lots of people are crammed together in a small enclosed space, sucking in oxygen and blowing out lots of "waste-products." You non-physiologists prefer to call this situation "a party."

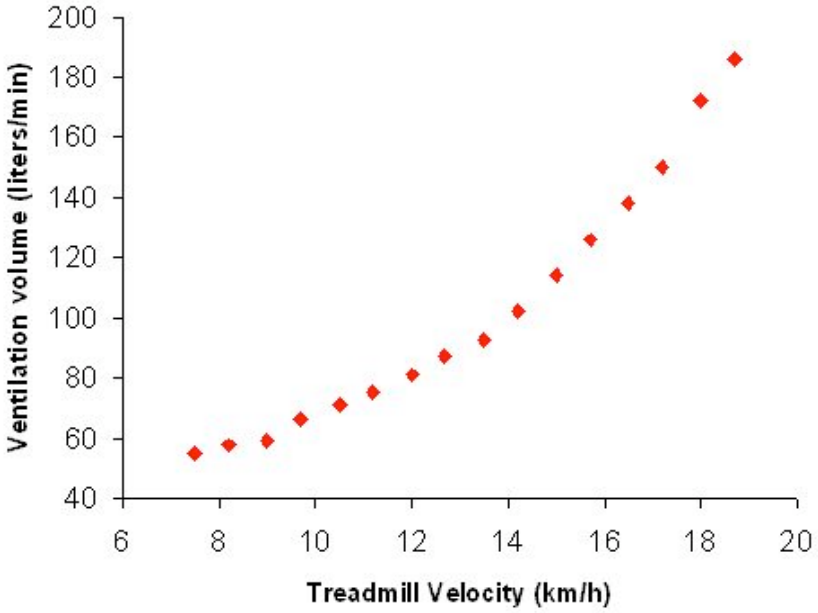
Rooms depend on air-conditioning systems, or big windows and a breeze for ventilation. Our body depends on the lungs, the diaphragm and several intercostal muscles, and a sensitive feedforward/feedback controlled regulatory system to control them. The purpose of the lungs is to ventilate the blood. Blood is the transport vehicle that carries oxygen to all of our cells, and carts off the constant production of CO2 that is produced as a by-product of both metabolism and pH buffering. The lungs are the site of pickup from and delivery to, the atmosphere. The greater the demand for oxygen delivery and CO2 removal, the greater the air volume that must circulate in and out of the lungs each minute. All animals of any size at all have had to come up with a ventilation system to get oxygen from the atmosphere down to the most hidden of the cells via the blood. Fish have gills. Insects have a system of air tubes called trachea. We mammals own beautiful pink lungs.

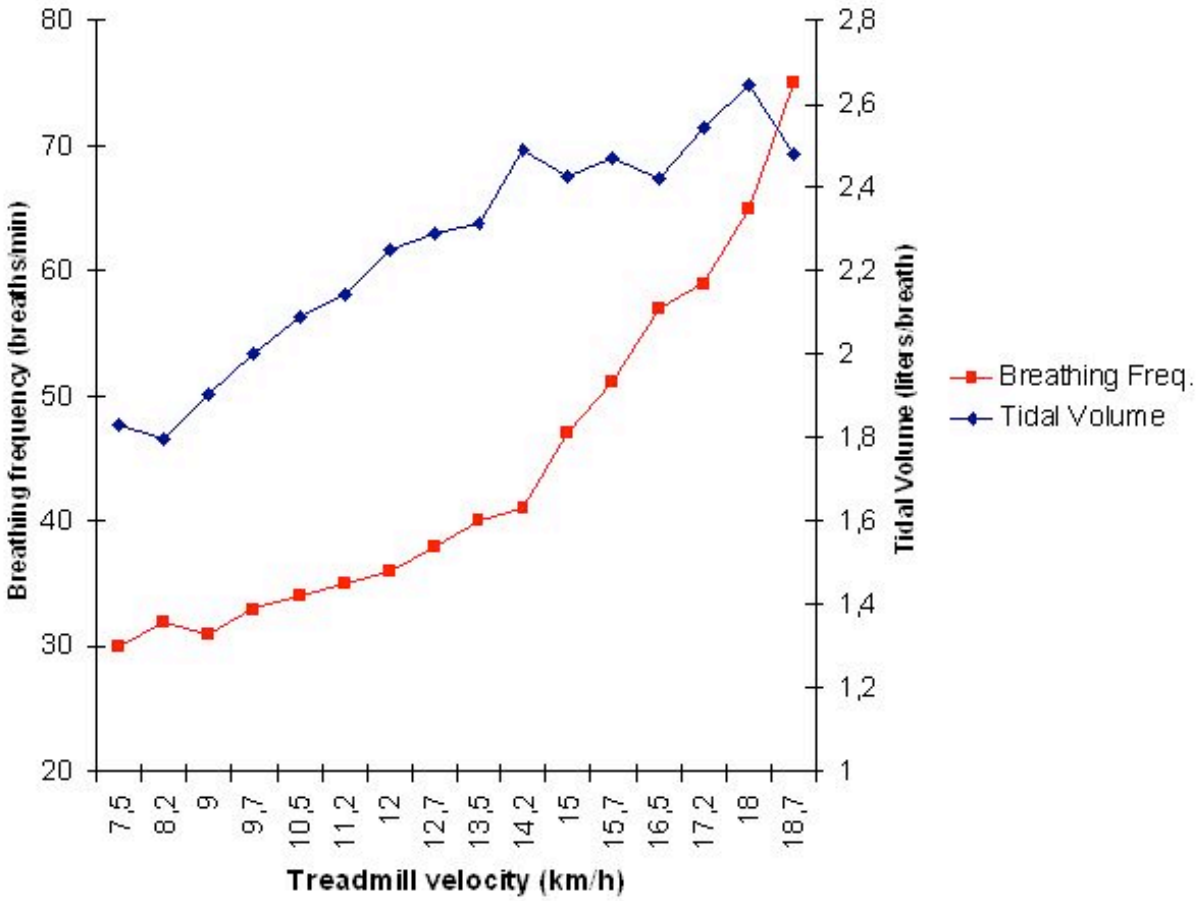
Three aspects of ventilatory function: air exchange, O2/CO2 exchange, and blood-gas carrying capacity

1. Air Exchange: Moving air in and out of the lungs

Sitting here in front of my computer, I am breathing about 12 times per minute (although if I try to count my own breathing rate, it will change because I am thinking about it). So my ventilatory frequency (Vf) is 12 breaths/min. Each breath has a volume of about 0.75 liter. This is my resting tidal volume, the volume of air flowing in and out of my lungs each breath. Multiplying ventilatory frequency (Vf) times tidal volume (TV) gives me ventilatory volume (VE), which is 9 liters/min in my example. This is the volume of air moving in and out of my lungs each minute. Somewhere in my files, I have the data from one of the VO2 max tests I did as a graduate student. During that test, my ventilatory volume (liters/min) peaked at 187 liters/min. That is over 20x greater than at rest. How do our lungs meet the demands of exercise?

Ventilation is regulated in much the same manner as cardiac output. The heart increases cardiac output by increasing both stroke volume and beating frequency. The respiratory nerves control ventilation similarly. At low exercise workloads, the dominant ventilatory adjustment is an increase in tidal volume, the volume of air being moved in and out of the lungs each breath. At high workloads, an increase in breathing frequency is the primary adjustment. The two figures below visualize this. The first figure shows the overall ventilatory response during a treadmill test with progressive increases in velocity every 60 seconds, and a constant incline of 5%. The second figure breaks the ventilatory response above into breathing frequency and tidal volume. This is real, unsmoothed data collected during a test of a well trained runner.





Unlike heart function, ventilation is under considerable voluntary control (with involuntary override mechanisms!). So, you might ask the question, "Can I control my breathing to make it more efficient?" For example, you could decrease the breathing frequency and take bigger deeper breaths to achieve the same total ventilation volume. Studies have indicated that normally the body spontaneously balances the depth of ventilation and the frequency of breathing so that ventilation is optimally efficient. You may also have noticed that at low exercise intensities, you can "play" more with your breathing, by varying its rate and depth. However, as the workload gets high, especially at workloads above the lactate (and ventilatory) threshold, the body assumes much tighter control on breathing and there is far less room for variation in breathing "strategies."

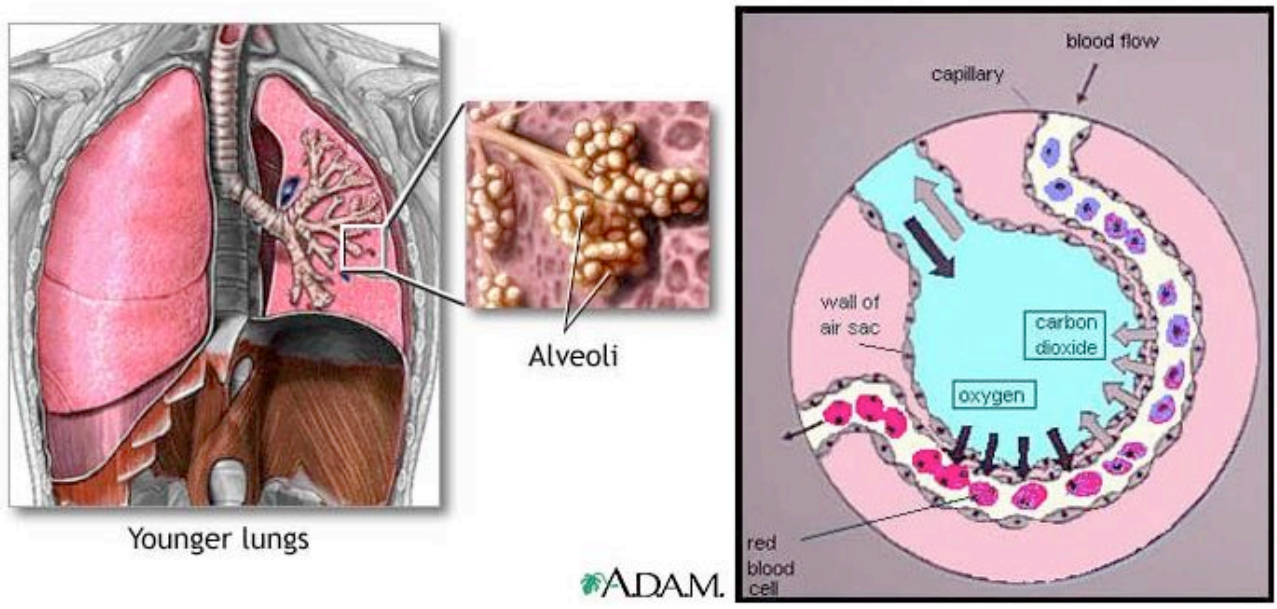
The actual act of moving air in an out of the lungs is accomplished in much the same way that a bathroom plunger works. Since the thorax is "sealed,"

expanding its volume causes air to rush into the lungs to fill the relative vacuum. When we inspire air, we do so by contracting the diaphragm, pulling it down (similar to pulling up on the plunger). If you are breathing "correctly" at rest, the main movement you will notice is your stomach bulging out a bit during inspiration. This is because the diaphragm is pressing down against the abdominal cavity. You are "belly breathing." At rest and low ventilation volumes, this is enough. However, when we need to move a lot of air in and out of the lungs, we not only increase the force of diaphragmatic contraction, we also contract muscles attached to the rib cage. This pulls the ribs up and out, further expanding the thorax and allowing more air to rush into to the lungs. All of this takes muscular work, so inspiration is an "active process." Expiration, or blowing the air back out, is basically a matter of relaxation when we are at rest. The elasticity of the muscles and tissues is sufficient to push the air out. The process becomes more demanding as we exercise harder. During heavy exercise we exhale more forcefully and deeply in order to ensure more rapid and complete exhalation of the old air. This process also takes energy in the form of muscular work. So, the bottom line is that breathing is not free to the body. And, it gets more energetically expensive at high workloads. I will come back to this point later.

2. Gas exchange: Moving oxygen and carbon dioxide on and off the red blood cells.

OK, now that we are moving the air in and out of our body, let's turn our attention to the lungs, and "get cellular" in our thinking. The lungs do some pretty cool stuff. The lung's challenge is to mix air and blood thoroughly and rapidly so that gas exchange can occur. Here comes another analogy. Let's say you have a gallon bucket (4 liters) of paint sitting with the lid open. How long will it take that bucket of paint to dry out? Days and days. But, if we spread the paint as a very thin layer over a very large wall, it will be dry in no time. By spreading the paint out we increase the total area of exposed surface between the paint and the air thousands of times and the water in the paint is quickly evaporated. At any given instant at rest, the lungs spread about 70 ml of blood (less than half of the volume of a coke can) in a "sheet" of capillaries with a total surface area of 70 square meters. That is like spreading a gallon of paint thin enough to paint a football field! The capillaries are so narrow that the red blood cells actually have to squeeze through. This also insures that the gas exchange across the red blood cell and capillary membranes is lightning fast. Simultaneously the lungs move the inspired air down a system of 23 branches of air passages terminating with about 300 million tiny spherical alveoli that form the terminal exchange tissue in the bronchial system. These two

exchange systems, the alveoli for air, and the capillaries for blood, are intertwined so microscopically close that oxygen and carbon dioxide molecules diffuse across the membranes and equilibrate almost instantaneously. Blood passes through the capillaries in about 0.8 seconds at rest and as little as 0.4 to 0.5 seconds during hard exercise. It is during this very brief exposure period that all the gas exchange between each red blood cell and the air in the lungs must take place before each return trip to the body!



Now perhaps one of you physiology student types is doing some analytical thinking that does like this: If resting cardiac output is 5 liters per minute, and maximal cardiac output were say, 25 liters/min, that would mean that 5 times as much blood passes through the lungs (and back to the heart and then to the body) and those capillaries per minute at max. Why doesn't the lung capillary transit time for each blood cell decrease from 0.8 secs at rest to 0.8/5 or a really fast 0.16 seconds during maximal exercise? Here is the answer. Normally, the lungs only use a fraction of the total capillary volume available. Small arterioles can regulate the entry of blood into portions of the lungs. During exercise this restriction is gradually removed and the capillary volume can increase by over 3 fold to about 250 ml. This helps to minimize the decrease in capillary transit time. It does not eliminate it though. How important is that? This is another point which we will hit on again.

3. Blood-gas transport: Delivering oxygen to the muscles

Now let's think about how the blood fits into all of this. Blood serves several important functions during exercise (heat removal, deacidification, glucose delivery, hormone communication to name a few), but the one I want to focus on is its role as a delivery truck for oxygen. About 40 to 50% of the total volume of blood is made up of red blood cells (RBCs). For example, if your "hematocrit" is 43, then 43% of your total blood volume is RBCs. More than anything else, RBCs are just tiny, flexible sacks of hemoglobin. Each RBC contains hundreds of hemoglobin molecules, and each hemoglobin molecule has room to carry exactly 4 oxygen molecules. When I say "carry" I mean "bind" in chemistry lingo. There is some very fancy chemistry going on here that we would be in deep trouble without, but I will not try to explain it other than to say that hemoglobin molecules are engineered to hold on to oxygen tightly enough to carry it out of the lungs, but loosely enough to release it in the capillaries feeding the skeletal muscles (and other organs of course). This whole process is designed to function best when the atmospheric pressure is near sea level. If we get 1500 meters or more higher than that, the system begins to break down, and hemoglobin leaves the lungs without a full load of oxygen. This is why it is more difficult to breathe and exercise at altitude.

The capacity for blood to deliver oxygen can be summed up using an equation:

Hemoglobin concentration X oxygen binding capacity of hemoglobin (ml O2/g hb) X percent saturation of hemoglobin = oxygen carried in a given volume of blood.

Hemoglobin concentration is expressed in grams of hemoglobin per deciliter of blood (g/dl). Typical values range between 12-14 for women and 14-16 for men. The binding capacity of hemoglobin for oxygen is a constant and equals 1.34 ml O2/g hemoglobin. Finally, the percent oxygen saturation of hemoglobin when it leaves the lungs is normally about 96% (it is not 100% largely because the lung tissue has its own blood supply and this small volume of deoxygenated blood mixes in with the fresh stuff).

So, for an average person with a hemoglobin of 15, the oxygen volume contained in each liter of delivered blood will be:

15g/dl x 1.34 ml O2/g hgb x 0.96 saturation x (10dl/l) = 193 ml O2/ liter blood.

If we substitute in 12 for the hemoglobin concentration (someone with anaemia) and 18 (a very high value occasionally seen in trained athletes at high altitude), we see that for the same cardiac output, the volume of oxygen carried by the blood would vary between 154 and 232 ml per liter, depending on the hemoglobin value. It is not hard to see how the blood oxygen carrying capacity affects the VO2 max. Remember, the muscles can only use what the heart can deliver.

If hemoglobin concentration is higher, the blood can carry more oxygen. This is an important point with relevance to altitude training, illegal EPO use, gender differences in VO2 max, anaemia etc. *(I should note here that there is a downside to increasing haemoglobin concentration in the blood and that is increased blood viscosity. The body normally maintains an appropriate balance. If the blood becomes too thick, flow resistance increases and the risk of blood embolism increases, hence the dangers of EPO use.)* Second, when the blood leaves the lungs it is normally fully saturated with oxygen. This means that the lungs are very effective at ventilating the blood, even in untrained folks. This is one of the reasons why in the big scheme of things we basically disregard lung function as an area for improvement in the athlete's endurance machine. But, this issue is worth taking a closer look at.

Ventilation Issues in Endurance Performance

Is ventilation volume a limiting factor to maximal endurance?

Sometimes you hear people say "I ran out of wind." Is that really possible? Can we reach a point in exercise when ventilation just can't keep up with demand? The answer is no, assuming you don't have acute asthma or some other severe pulmonary dysfunction. We can measure a person's maximal voluntary ventilation (MVV), the maximal volume of air they can breath in and out while at rest, and compare it with their maximal ventilation during exercise. What we see is that untrained people only use about 60 to 85% of their maximum ventilatory capacity even at maximal exercise. For example the MVV for an average male might be nearly 200 l/min. However, during a treadmill VO2 max test, they reach a peak ventilation of only 140 l/min. Highly trained athletes use more of their capacity, perhaps over 90%, but ventilation capacity is still not a limitation on performance. Unlike the story with cardiac output, even during maximal exercise, the ventilatory capacity is not maxed out.

By the way, the highest ventilation volume I have read about in a human was 263 l/ min recorded on a really big male rower with a chest about the size of a

beer keg. Ventilation volumes in excess of 200 l/min have also been recorded in elite female oarswomen. Understandably, rowers get most of the big ventilation prizes because they are really big for endurance athletes, at least among humans. The highest ventilation rates I have heard about in any athlete, irrespective of species, was in a racehorse. They have ventilation volumes of about 1500 liters/min!

Does ventilation performance decline as we get older?

Data from Åstrand presented in his Textbook of Work Physiology addresses this question. He compared ventilatory parameters in a group of male and female physical education students when they were in their 20s, again when they had reached their 40s, and a third time when they were in their 50s. The longitudinal study covered 33 years in all. While other aspects of the students' capacity declined, basic lung function was very stable. Total Lung Capacity was unchanged. Maximal Tidal Volume was unchanged and Maximal Ventilation during exercise was only decreased a few percent in 33 years. One change that does seem to occur consistently due to aging is an increase in the "residual volume" as a percentage of total lung capacity. This means that less of the actual lung volume is dynamically used during ventilation. This age-related change can be accounted for by loss of lung elasticity with age. Overall though declining lung capacity does not seem to be a big factor in the performance limitations of the aging athlete. Lung function is not the weak link in endurance performance, assuming you stay away from the cigarettes.

Uncomfortable Positions: Can body position influence ventilation capacity?

Now, one issue that needs to be considered when we discuss ventilation is body position. In most sports situations the chest is very free to expand. Running and cycling do not impose any mechanical limitations on ventilation. Even down in the aerodynamic position, maximal ventilation in cyclists does not appear to be compromised. However, swimming adds resistance to breathing because we have to move both the ribs and the water surrounding the body when expanding the chest. Coupling this with the problem of coordinating breathing with the brief periods of time the mouth is out of the water probably results in a relative under-ventilation in swimming.

Ventilation during rowing: Special Problems?

Another sport that has gotten some special attention from the ventilation folks is rowing. This I know more about, so I will elaborate a bit. During rowing, the body is squeezed up with the chest against the knees over 30 times a minute, limiting diaphragmatic excursion. That might create some breathing problems, but it is not the biggest issue. The real issue is the fact that rowers also use the same abdominal and intercostal muscles used for breathing to support the back during the powerful extension employed each stroke. Rowers isometrically contract all of these muscles to apply a high interthoracic pressure at the moment of the catch, when the oars take the water, to reinforce the connection between oar, back and legs. It is impossible to breathe and constrict all the abdominal and thoracic muscles at the same time.

The consequences of this competition are debated. The results of several, but not all studies suggest that elite rowers are not able to achieve the same ventilation volume at max during rowing as they achieve during cycling. The differences are not huge, but they may be significant. For example in one recent study elite rowers achieved a peak ventilation of 198 l/min during a VO2 max test cycling, but only 171 liters/min during rowing. The VO2 max values for the rowers were not different between rowing and cycling (5.03 l/min vs 5.09 l/min). This suggests that the small degree of under-ventilation at max experienced in rowing does not limit maximal oxygen consumption. However, some physiologists interpret these results differently. It is generally accepted that elite endurance athletes achieve their highest values of VO2 max when they are performing the sport that they train for. In other words, elite runners excel most during treadmill tests. Elite cyclists max out slightly higher on cycling tests etc. In several studies national class rowers have demonstrated the same VO2 max while rowing as they did while running, or even cycling. This has not been a unanimous finding, but it appears that VO2 max for highly trained oarsman during rowing is lower than it "should be", when consideration is given to their training specificity and the very large muscle mass employed in rowing. A mechanism for this problem may be a slight ventilatory limitation imposed by the unique demands of rowing. Personally, I am inclined to believe that VO2 max is limited in rowing for a different reason. The muscle contraction frequency is two slow and at too high intensity to allow optimal blood flow to the working muscles, and muscle pump action by the working muscles. If this is true, then higher stroke rates might produce increased aerobic power. This is consistent with the trend in elite rowing to move toward higher stroke rates, but it is not proven. But, now that I have mentioned stroke rates, that brings up another interesting ventilation issue.

Breathing to the beat: Entrainment of ventilation rate to movement rhythm

If my wife joins me at the rowing club for a workout on the rowing machines, an interesting phenomenon occurs. Hilde is not a rower, so she always seems to adjust her rowing cadence so that it matches mine. I don't think she does it on purpose, but her rowing rhythm naturally entrains onto mine. This is problematic when I am doing intervals and she is rowing steady state! Our ventilatory system does the same thing. Ventilation tends to match with running, cycling or rowing cadence in a consistent pattern. For example, in cycling, we sometimes see athletes exhale in unison with the downward kick of the same leg, every 2nd or third stroke. This entrainment process does not seem to be a bad thing. In fact, since it is more prevalent in experienced athletes, it is probably an adaptation that promotes efficiency by minimizing the mechanical constraints to breathing created by limb movements.

Breathing pattern seems to be an especially important issue in rowing. Steinacker et al (1992) investigated ventilatory responses during incremental rowing exercise and observed two distinct breathing patterns. Type 1 was one complete breathing cycle per stroke cycle, with expiration occurring during the drive and inspiration during the recovery phase. Type 2 was two complete breaths per stroke, one during the drive and one during the recovery. When the intensity reached a certain point, the rowers automatically switched from type 1 to type 2. All these elite male rowers entrained their breathing to the stroke rate. Another study found the same patterns in elite female rowers. Untrained subjects tested during rowing only rarely exhibited this pattern. To make things more interesting, it appears that the breath during the stroke is "smaller" than the breath during the recovery phase.

Now if we extrapolate those findings to a racing situation at a high 40 strokes per minute, what we would expect is a ventilation rate of 80 breaths per minute. This is very high! In running or cycling max tests we usually see maximum ventilation rates of about 50 to 60. In contrast, ventilation rates as high as 88 breaths per minute have been observed during competitive rowing, accompanied by a relatively low tidal volume. All of this data suggests that the mechanics of rowing place unique demands on ventilation. Well trained rowers adapt to these demands by developing very strong ventilatory muscles and adapting a unique breathing rhythm which makes the most of the brief periods of relaxation during the stroke.

The Great Arterial Desaturation Debate

New topic. Earlier in this breath-taking novel of mine, I discussed the issue of hemoglobin saturation with oxygen. I said that the lungs were so good at oxygenation that the blood always leaves the lungs saturated with oxygen, even during hard exercise when cardiac output is high. This means every RBC picks up a full load of oxygen before leaving the lungs. Now I am going to contradict myself a bit.

Here is the central issue. The blood returning from the periphery must eliminate its carbon dioxide load and fully re-saturate with oxygen during the brief time it passes through the lung capillary network on the way back through the heart and out to the body again. Normally this is not a problem. It only takes about 0.45 seconds for the hemoglobin to become fully saturated during its passage through the twisting capillaries. It takes even less time to unload the CO2. Since the transit time is 0.8 seconds, there is time to spare. Even during exercise there is enough time, unless.....you are a really fit athlete with a very high cardiac output and VO2 max.

Recent studies with highly trained endurance athletes (VO2 max over 70 ml/kg/min) have shown a significant degree of arterial desaturation. This means that for the guys with the really big cardiac outputs, the blood is rushing through the lungs so fast that hemoglobin hasn't taken on a full load of oxygen before leaving for the muscles. The result is that instead of being 96 or 97% saturated with oxygen, the blood leaving the lungs may only be 89 or 90% saturated in the athletes with very high cardiac outputs and VO2 max. Clifford et al. (1990) reported a drop in arterial saturation from 105 mm Hg at rest to 88 mmHg during the last minute of a maximal rowing test in elite rowers. This means that the arterial blood was becoming slightly hypoxic when they reached very high workloads. All other things being equal, VO2 max might be up to 5 % higher or so in the elite types if the lungs could fully saturate the blood at maximal cardiac output. Support for this assumption comes from the fact that when well trained athletes breathe a higher concentration of oxygen while performing in a lab, they reach a slightly higher VO2 max.

What does this information mean in regards to how we train? Nothing. There is nothing we can do to prevent this desaturation in folks with the really high cardiac outputs, short of having them wear an oxygen tank while performing. Unless you have a VO2 max of about 5 liters/ min or higher, it is not really an issue anyway. Consider arterial desaturation a small physiological tax on the "cardiovascularly endowed."

The Rising Cost of Breathing

The ventilatory muscles, like all muscles need oxygen to support continuous exercise. It turns out that the diaphragm is one of the body's best endurance muscles, perhaps even in second place behind the heart. It has a high percentage of type I fibers, a high capillary density, and high concentration of oxidative enzymes, compared to skeletal muscles. Animal studies have demonstrated that the diaphragm improves its endurance capacity (mitochondrial enzyme concentration) with training, but not more than about 20-30%, because it is already pretty well equipped for chronic work. However, with high intensity training, other muscles involved in breathing like the internal and external intercostals and the abdominal muscles become more active and also improve their endurance capacity. Since these muscles are less trained to begin with, they respond more to endurance training. These accessory breathing muscles are not trained at low exercise intensities but become active when we really start moving a lot of air.

From the above, plus your own experience, you can figure out that breathing becomes more demanding when ventilation rates get very high. There are studies which have actually measured the oxygen cost of breathing at different intensities. You might think of this as the tax on oxygen delivery. The body has to deliver blood to the ventilatory muscles so that they can help the lungs supply oxygenated blood to the rest of the body. To make things worse, this "tax rate" increases when you are working at very high intensities. The oxygen cost per liter of ventilation (VE) doubles from low to very high exercise intensities.

The bottom line is that while the oxygen cost of breathing is only perhaps 3-6% of total VO2 at low intensities, it can be as high as 10 to even 15% of total VO2 in young adults with greater than average VO2 max. In the fit older athlete, a high oxygen cost of breathing may occur at lower ventilation rates due to the increased stiffness of the chest wall.

Because physical training results in a reduced ventilatory response (at any given level of CO2 production) during intense exercise, in the highly trained, the high cost of breathing is somewhat reduced. One interesting physiological question is whether or not the respiratory muscles "steal" blood flow from the skeletal muscles at intensities near VO2 max. The scenario goes like this. Cardiac output has already maxed out, but hyperventilation is still climbing, so the respiratory muscles need more oxygen. Physiologists have predicted that

the respiratory muscles would get their share of blood flow at the expense of skeletal muscle blood flow, but direct experiments are lacking. Does this mean VO2 max would decrease? No, it would be the same. However, the peak work rate achieved at VO2 max would be reduced, because more of the consumed oxygen is going to supply “supporting organs” instead of the skeletal muscles.

When you add this to the problem of desaturation, we see that there are some additional limitations on maximal performance that enter into the picture when we start dealing with extremely well trained endurance athletes. These guys have such high cardiac outputs and work capacities that the ventilation machinery starts to demand a lot of the total available oxygen in order to keep the machine running.

Do the Breathing Muscles Fatigue?

The final issue I want to discuss brings us back to a common theme in endurance performance, muscular fatigue. We know the skeletal muscles fatigue (lose force generating potential) during endurance exercise. Do the breathing muscles get tired?

To make things short and sweet it appears that they do fatigue. Tests of maximal ventilatory function after a hard endurance session show a temporary drop in, for example peak expiratory force. This is an indirect way of measuring how much force the diaphragm can generate.

But, does fatigue of the ventilatory muscles limit performance?

To date the best technique for answering this question is to unload the ventilatory muscles and observe whether performance improves. These techniques have included using helium oxygen mixtures and breathing assist devices. Unfortunately, the methodology is not perfect. For example, using lighter than air helium may make the hoses and mouthpiece easier to hold in the mouth and actually decrease the cost of rowing. The results are unclear. At workloads below 85% of VO2 max it appears the respiratory fatigue has no influence on performance. However, the results of some studies suggest that at intensities approaching VO2 max respiratory fatigue may contribute to performance limitations. The scientific jury has not reached a verdict on this question.

Can I Apply Any of This to My Training?

So, if you made it to the end of this novel, you may be disappointed to learn that there are no secret breathing tricks that will push you over the top. In general the lungs are wonderfully equipped for doing their job. Training does improve the ventilatory system in some ways, but it is not the weak link in healthy athletes. In recent years, there have been a handful of studies published where the impact of inspiratory muscle training on various aspects of pulmonary and endurance performance have been investigated. This involves essentially weight training for the breathing muscles, where resistance is generated by using some kind of device that reduces airflow during inspiration and forces the inspiratory muscles to work harder against greater resistance. Neither peak pulmonary function nor maximal oxygen consumption have been shown to change with this form of training. However, a couple of studies have shown modest increases in either time to exhaustion or time trial performance during cycling, using placebo controlled designs. How does this work? Perhaps stronger inspiratory muscles allow high ventilation to be achieved at lower breathing frequencies. This would decrease the oxygen cost of breathing and free up some blood flow for the working muscles. Perhaps.

If there is another area where we can benefit from attention to breathing, it would be the issue of entrainment. Good athletes develop breathing “rhythms” that tune in to the rhythms of their movements. This probably promotes efficiency. When you feel yourself performing at your physiological redline, your breathing may be a place to turn your attention. If you are a runner or cyclist, focus on the diaphragm and the abdominal muscles for moving the air in and out, instead of the intercostals attached to the chest. Heaving the chest more than necessary costs extra energy. “Belly breathing” makes sense. If you are a rower “belly breathing” doesn’t work too well. We just have to learn how to breathe between the strokes.

12. AERODYNAMICS AND CYCLING by Jim Martin

This article comes from Jim Martin Ph.D, an engineer, associate professor in exercise science at the University of Utah, and a friend of mine from my doctoral student days. Jim has been a Masters Champion on the track, and has coached the EDS cycling team. He also has spent about as much time as anyone doing wind tunnel testing for cycling, both at the GM and Texas A&M wind tunnels. This article was written for triathletes, but the information still applies to the straight cyclist. It is very popular to use the term 'aero' to describe bicycles, wheels, helmets, and handlebars. However, do we really know exactly what 'aero' means, and what the consequences of aerodynamics are to you? We measured drag in the wind tunnel of seven riders, then had them ride at three steady state velocities while we measured power with an SRM crank and wind conditions with an anomometer. The results indicate that our predicted power matched our measured power with a standard error of 5 watts, and demonstrate that this is a valid model for power during real world cycling.

Knowing the power required for a given riding velocity may be meaningless if you don't know how much power you can produce. If you, as a triathlete or duathlete, are equally well trained at cycling and running, and have average running economy (1.6 kcal/kg/mile) and average cycling efficiency (19% gross cycling efficiency) your sustainable power output can be estimated from this simple equation: Power (watts) = 60 x Body weight (lb) / 10k run time (minutes). Based on this equation, Table 1 presents the estimated power output for 4 categories of triathletes / duathletes. Keep in mind that if you are estimating your power in a multi-sport event, you should use your 'multi-sport run' time, whereas if you are estimating your cycling time trial performance, use your 'run only' time. These estimated power outputs will be used to illustrate the effects of aerodynamics on a variety of riders.

TABLE 1. *Estimated cycling power output for a 70 kg person based on 10k multi-sport running time*

	Elite	**Well Trained**	**Trained**	**Recreational**
10k Time	35 min	40 min	48 min	60 min
POWER	264 watts	231 watts	192 watts	154 watts

Although much attention is focused on the aerodynamics of equipment, the most important aerodynamic consideration for a bike and rider combination is the rider. A typical 70 kg rider on a regular bike with standard wheels will have a drag of about 8 lb, a better position will reduce drag to about

7 lb, and an excellent position will yield a drag of 6 lb. Based on these drag numbers, and the power outputs estimated above, equation 1 can be used to predict the effects of these positions on cycling performance on a flat course with no wind shown in Tables 2 and 2a. The differences in performance with no change in power are remarkable, ranging to about 6 minutes when changing from a typical to an excellent position.

TABLE 2: *Predicted 40k time, flat course, calm conditions, 3 body positions, standard wheels.*

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	8.0	62:49	65:51	70:16	76:01
Good	7.0	60:14	63:07	67:22	72:57
Excellent	6.0	57:23	60:10	64:07	69:47

TABLE 2a: *Predicted time savings for a 40k based on 2 body positions compared with a typical position, flat course, calm conditions, standard wheels.*

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	8.0	62:49	65:51	70:16	76:01
Good	7.0	2:35	2:44	2:54	3:04
Excellent	6.0	5:26	5:41	6:09	6:14

The key elements of a good aero position are:

1. Horizontal torso. Defined by having your chest, or better yet, your back parallel to the ground, this is absolutely the most important element, as it can result in **large magnitude changes in aerodynamic drag**. Unfortunately, it may be the most difficult to achieve, because as you approach this position, your thighs start to hit your torso. This interference imposes limits on your body's aerodynamic position, but is due to traditional bike geometry (i.e. seat tube angles of 73 to 75 degrees). The way to overcome this limitation is to go to a more forward position, which will allow you to roll your whole body forward. Note of caution: a forward position seat post and long steeply-dropped stem may allow you to assume a good aero position, but will result in a bike that is not well balanced, and may be dangerous to ride. A much better approach is to buy a frame that is designed to be ridden in a forward position. These positions are uncomfortable in two ways. First and foremost, by rotating your hips

forward to get your torso horizontal, you are rotating your weight right on to your soft and tender parts. Specifically, riding in this position may exacerbate the condition of prostatitis that is common among cyclists. Extra seat padding helps but does not eliminate the problem. A truly anatomical saddle that distributes your body weight over the whole seat might really help. Some riders try to alleviate this problem by tilting the nose of the saddle down, but this only results in a tendency to slide off the saddle and to strain your shoulder and arm muscles. Secondly, and to a much lesser degree, you tend to get a sore neck the first few times you ride. The discomfort lessens with time and can be minimized with stretching and massage. These drawbacks are minimal because you don't have to ride the forward position daily to go fast on it. My experience with Team EDS, as well as my own bike is that you only need to ride it once a week (maybe less) to stay adapted to the position.

2. ***Narrowly spaced elbow pads.*** Narrow elbows are an essential detail of an aero position. However, the magnitude of improvement is much less than what is achieved by adopting a horizontal torso position. Research conducted by Boone Lennon has shown that subtle changes in elbow width and aero bar angle may have significant effects on drag. This research was performed on traditional geometry bikes, with the torso adopting the characteristic cupped shape, and probably illustrates the need to block air flow out of the torso area. More recent data on riders in a horizontal torso position shows much less effect from these variables. I do not believe these two findings are contradictory, rather, they indicate that once the torso is horizontal there is little you can do to improve or impair aerodynamic drag.

3. ***Knee Width*** can change aerodynamic drag by up to half a pound. Pedaling with your knees close to the top tube is an essential part of good aerodynamics. Is there a trade-off between position and power output? If done badly, maybe, but if done well, no. Recently, Heil et al., (MSSE, May 1995) have investigated this question, and the results tend to show that your cardiovascular stress for a given power is increased by decreasing the trunk to femur angle. Therefore, if you lower your elbow position, you may need to move the saddle forward to maintain your trunk to femur angle while getting a lower, more nearly horizontal torso position.

4. ***The effects of aerodynamic wheels*** can be substantial. They can lower the aerodynamic drag by about 0.4 lb. compared with standard wheels with

round-wire spokes and require about half the power to rotate. For the following examples, I will use a Specialized 3 spoke front and a lenticular rear disc.

Tables 3 and 3a show the predicted effects these wheel will have on 40k time trial performance.

TABLE 3: *Predicted times in a 40k by using aero wheels compared to standard wheels, flat course, calm conditions, 3 body positions*.

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	7.6	61:40	64:38	68:54	74:39
Good	6.6	58:58	61:47	65:55	71:23
Excellent	5.6	55:57	58:39	62:35	67:47

TABLE 3a: *Predicted time saved in a 40k by using aero wheels compared to standard wheels, flat course, calm conditions, 3 body positions*.

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	7.6	1:09	1:13	1:22	1:22
Good	6.6	1:16	1:20	1:27	1:34
Excellent	5.6	1:26	1:31	1:32	2:00

The difference made by aero wheels is about a one to two minutes over 40k. When I was preparing this talk and I got to this part, I didn't believe the model's prediction. So I recruited a friend and went out to a fairly flat loop and rode at constant power with regular and aero wheels. The results were almost exactly what the model predicts. This study needs to be repeated with better control such as wind and road grade measurement, but it provides anecdotal evidence that the predicted effects of wheels are realistic.

Similarly, the effects of aerodynamic frames can be substantial. The best frames can reduce drag an additional 0.3 lb. compared with round frame tubes. The critical areas of a frame seem to be the leading edge (fork, head tube, handlebars) and the area between the rider's legs. The frames that perform the best tend to have air-foil shaped leading edges and seat tubes (or no seat tubes). The effects of an aero frame are estimated in Table 3.

TABLE 4: *Predicted 40k time, flat course, calm conditions, 3 body positions, aero wheels, aero frame.*

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	7.3	60:53	63:47	68:04	73:40
Good	6:3	58:05	60:51	64:55	70:21
Excellent	5.3	54:59	57:39	61:30	66:38

TABLE 4a: *Predicted time saved in a 40k by using an aero frame compared to a standard frame, flat course, calm conditions, 3 body positions.*

Position	Drag @30mph	Elite	Well Trained	Trained	Recreational
Typical	7.3	0:47	0:51	0:50	0:59
Good	6:3	0:53	0:56	1:00	1:02
Excellent	5.3	0:58	1:00	1:05	1:09

As you can see above, the effects of an aero frame result in saving about an additional minute.

The effects of light weight components seem to be a topic of interest for many triathletes/duathletes, however the effects of weight on cycling performance may not be as significant as one expects. To illustrate the effects of weight I have modeled a very tough out and back 40k with a constant grade of 3% which results in 600m or about 1970 feet of climbing/descending with aerodynamic bikes that weigh 22 lb. and 17 lb., and a slightly less aero bike/position that weighs 17 lb. The results are shown in tables 5 and 5a.

TABLE 5: *Predicted 40k time, 3% grade out and back course, calm conditions, 2 body positions, aero wheels, 3 bikes.*

Bike Wt	Drag @30mph	Elite	Well Trained	Trained	Recreational
22 lb	6.3	65:04	69:38	76:55	87:24
17 lb	6:3	64:37	69:05	76:12	86:27
17 lb	6.8	65:52	79:22	77:31	87:47

TABLE 5a: *Predicted changes in 40k time due to weight and aerodynamics, 3% grade out and back course (600m or ~1970ft of climbing/descending), calm conditions, 2 body positions, aero wheels, 3 bikes.*

Technical Factors	Elite	Well Trained	Trained	Recreational
• Time saved with 5 lb lighter bike	-0:27	-0:33	-0:43	-0:57
• Time lost with 0.5 lb more drag, 17 lb bike	+0:48	+0:44	+0:36	+0:23
• 0.5 lb More drag 23 lb bike	+1:15	+1:17	+1:19	+1:20

An extremely light bike on a very tough climbing course will only save you about 30 seconds to 1:00, but if this lighter bike compromises your aerodynamics even a little bit, you will be SLOWER by 23 to 48 seconds. Interestingly, lighter weight is more of a help to slower riders. Increasing drag by 0.5 lb. slows you down by about 1:15 on the same weight bike.

Till now, I've modeled everything in calm conditions, however, I personally have rarely ridden in calm conditions. Wind effects can be remarkable, largely because you spend a longer time in the head wind than you do in the tailwind, and consequently, the slower head wind portion has a greater effect on average velocity. Table 6 demonstrates the effects of 5 and 10 mph winds on an out and back course, direct head wind one way, tail wind the other.

TABLE 6: *Predicted 40k time, flat out and back course, windy conditions, good body position, aero wheels, aero frame.*

Wind	Drag @30mph	Elite	Well Trained	Trained	Recreational
Calm	6.3	58:05	60:51	64:55	70:21
5 mph	6:3	58:45	61:39	65:52	71:31
10 mph	6.3	60:48	63:58	68:40	75:02

13) Lactate Threshold

For over a year I held out that lactate threshold testing would eventually provide a test that would settle many disputes regarding exercise. Most of this hope was based on Brian F. Sharkey's writings. I now understand this belief was in vain. Since then, other publications by George A. Brooks, PhD (SPORTS, Volume 8; Number 1, January 1988 and Sports Science Exchange, Volume 1, Number 2, April 1988), (furnished courtesy of Ted Lambrinedes, PhD) cast weighty doubt that lactate threshold is reliable.

Generally described, lactate threshold is that level of sustained work whereby the blood lactate does not diminish as more of it is produced by the working muscles. Apparently, this threshold is increased with training effect.

Lactate testing is defined in a number of ways; thus, the first problem with it as a standard is definitional. In most disciplines this alone would be adequate grounds to discard it.

Lactate testing is riddled with inconsistencies that are impossible to control in the lab as well as in the field. The results of this test vary significantly dependent upon the time of day, the food ingested and proximity to the test, the site of blood withdrawal, the specific muscles worked and the time lag since the end of the exercise bout. These and other problems make lactate testing hopeless for meaningful standardization. Of course, this realization does not stop the exercise physiologists from using blood lactate testing to gain publicity with its sensationalism. Witness the June 1996 issue of Scientific American.

Hi, again. This time I would like to continue on with my discussion of training for endurance athletes. Last time, I talked a little about VO2 max and mentioned that it is frequently considered to be the prime determinant in endurance performance. Well, this time I would like to discuss another factor which may even be more important than VO2 max in determining performance: the concept of the lactate threshold (LT).

There are a couple of concepts similar to LT which, although not entirely identical, I will consider to be synonymous to LT. These are the anaerobic threshold (AT), ventilatory threshold (VT), and onset of blood lactate accumulation (OBLA). These are not entirely the same concept but, for the sake of this discussion, I will use them interchangeably. Essentially, what these terms refer to is the exercise intensity (or threshold) that you can maintain for a

long period of time and are usually expressed as a percentage of VO2 max. Put another way, above LT, you can only continue for a limited time due to the extreme accumulation of lactic acid in the blood which causes pain along with shutting down the energy producing capacity of the muscle.

Let me try to explain the LT with an example. Again, from last time, you may remember that I said VO2 max was frequently considered as the prime determinant for performance. Well, that would be great if you were physically able to exercise at 100% of your VO2 max, which you can't. So, let's take two athletes and do a little comparison. (I will be using extreme values to help illustrate the point).

Let's say athlete 1 has a VO2 max of 70 ml/kg/min which is very good. Athlete 2 has a mere VO2 max of 50 ml/kg/min which is about average. But, for some reason, athlete 2 always seems to win in races against athlete 1. Well, let's say we test the LT (I'll talk about the methods a little later) and find that athlete 1 has a LT at 50% of their VO2 max while athlete 2 has a LT of 90% of their VO2 max (again, LT is the percentage of VO2 max that you can sustain for a long period of time).

So, for these athletes, we find their effective VO2 by multiplying by LT. So, athlete 1 is able to perform at 70 ml/kg/min x 0.5 = 35 ml/kg/min. Athlete 2 can perform at 50 ml/kg/min x 0.9 = 45 ml/kg/min.

So, in the long run, athlete 2 is able to maintain a higher VO2 than athlete 1 which helps to explain why athlete 2 always wins in races. Admittedly, the difference in LT would not generally be this large but I picked these values simply to make a point. Most healthy people have the capacity to utilize about 70-80% of their VO2 for extended periods of time but this percentage can be raised with training.

So, what exactly does the LT (or OBLA, VT, or AT) exactly mean physiologically. Well, as you may know, during high intensity exercise, lactic acid is produced due to the lack of oxygen and the conversion of glycogen to lactate. Well, lactate causes the pH of the muscle to drop (it's an acid) which causes pain and also makes the chemical machinery of the muscle function less than optimally. Even at rest, you are producing some lactate. However, the rate of clearance (Rc) from the tissue is the same as the rate of appearance (Ra) so that there is no net increase in concentration. Well, during low-intensity steady-state exercise, although lactate levels increase, the body is still able to clear it from the tissue quickly enough to avoid buildup (Rc=Ra).

Well, at some critical exercise intensity (i.e. threshhold), the body becomes incapable of clearing the lactate from the tissue quickly enough (Ra>Rc). The end result, lactate accumulation and pain. And, depending on your capacity for pain, exercise generally stops quickly thereafter.

So, LT and OBLA are really referring to that point when lactate begins to accumulate faster than it can be cleared. VT refers to a point during exercise when ventilation (number of breaths/minute) begins to increase from a normal linear progression. This occurs due to a large increase in carbon dioxide levels in the blood. The body uses the base part of bicarbonate to buffer lactic acid and the excess CO2 generated must be blown off with extra breathing. AT is a term that sort of refers to the exercise intensity at which anaerobic (without oxygen) processes take over during exercise as this is generally associated with high lactate levels. AT is somewhat of a misnomer as all exercise use some portion of aerobic and anaerobic processes but it has gained popular acceptance. While LT and OBLA are pretty much identical, the VT generally occurs after the LT has been passed as a certain amount of lactate must build up to cause the overcompensatory effect of breathing to clear the excess CO2.

I'll wrap up the first part right here with a final comment. Some of you may be thinking "Well, that's great, but what do I do to raise my LT?" Well, I'll leave you with one word: intervals.

Hi again. Welcome to the second part in a three part (maybe?) series about the lactate threshhold (LT) and intervals for endurance performance. Before I get into a discussion of intervals and how to develop an interval program, I would like to talk a little bit about how LT is measured.

The most accurate way to measure LT is in the laboratory. But, even there, there are a few different ways to have it measured. The first and probably easiest is by collecting breath data. By performing an incremental exercise test, where the intensity is gradually raised, it is possible to measure a variety of data with a computer including oxygen uptake as well as number of breaths. Well, by graphing the data obtained, you can see a point where breathing rate begins to increase exponentially while exercise intensity is still increasing linearly. This is generally considered to be the LT, or more accurately the Ventilatory threshhold (VT) which as we said before occurs a little after the LT is passed.

A second way is by taking blood at various intervals and measuring lactate levels during the afformentioned exercise test. By looking at when lactate

levels begin increasing constantly, the LT (or OBLA) can be determined. The biggest drawback to this is the need to take blood samples during exercise and the difficulties involved with it (i.e. safety, needles, etc).

Well, as with direct VO2 max testing, both methods require significant cost and equipment as well as trained personnel. But, for the majority of people, direct testing may be overkill and unnecessary as it is possible to adequately determine your LT with a minimum of equipment and a couple of helpers.

The easiest way to use LT is just to assume that it falls within 70 - 90% or so of your heart rate reserve. Calculate this by taking 220 - age and subtracting your resting heart rate. Then multiply this value by 0.7 or 0.9 and add your resting heart rate back in. By playing around with where in this range you do your interval training, you can sort of hone in on where you LT is.

A second way to determine LT (which should be used with the above method) is monitoring your breathing frequency during exercise. After warming up, you merely begin to increase the speed/intensity of exercise until you feel your breathing becoming rapid and uncontrollable. This will more or less signify the onset of blood lactate which necessitates extra breathing to dispose of the carbon dioxide generated. If you have access to a pulse monitor (or can accurately take your pulse during exercise), you can determine at what point you are reaching LT. Recall, though, that you are really measuring the ventilatory threshhold (VT) with this type of assessment and that the LT generally occurs before the VT. So, you should back off the intensity a bit to arrive at your LT. Again, assuming you are training correctly, the heart rate at which your breathing becomes uncontrollable should increase indicating that you have raised the percentage of your VO2 max that you can perform at.

A third, and probably more quantitative method is the test Conconi developed by Francisco Conconi and Michele Ferrari for cyclists. Although it is probably easiest for cyclists to perform, other athletes can use it as well. Basically, you will be plotting heart rate versus speed. At some point, the normal linear relationship of heart rate will deviate from speed. The point of deviation is more or less the LT. Ideally, you need a pulse rate monitor and some friends to record pulse and speed. Also, if possible do the test on a track or treadmill or indoors on a wind trainer for cyclists to make speed measurement more accurate. Also, if on a track runners should run behind a pace bicycle to help with speed measurement. For runners, set a 50m section of track and mark the endpoints. You should be timed for this 50m in order to get accurate speed information.

Basically, after a sufficient warmup, you should begin exercise at a moderate pace. After every lap (or 60 or so seconds on the bike), record your pulse and speed and increase speed by 0.5 km/hr for runners or 1 mi/hr for cyclists. Continue doing this until you drop (err,terminate the test). Then graph the data and determine at which point heart rate deviates from speed (there should be a straight line for the majority of the test). Again, the point of deviation is pretty much the LT. The test can be repeated every month or so and, with correct training, the point at which heart rate deviates should go up.

That's it for now. I will conclude next time by discussing intervals: what they do, and how to do them.

Well, I had originally intended to talk about interval training at this point, but (as is usually the case), I will digress a little bit so that the discussion of intervals will (hopefully) make a little more sense. What I'd like to talk about this time is the different energy systems in the body, what they do, what they are good for and how they are trained.

There are three energy systems in the body and each has a different capacity to generate energy during daily activities. However, all three share the same ultimate goal: ***the generation of adenosine triphosphate (ATP).*** ATP is the only form of energy which can be directly utilized in the body and, as such, is ultimately the rate limiting substance in exercise. You can have all of the glycogen or fat in the world inside your body, but if you can't turn it into ATP, it won't do you any good.

Before I get to the descriptions, let me briefly (very briefly) outline what generally happens during exercise. The ATP molecule has a lot of stored potential energy in the form of the chemical bond on the inorganic phosphate. The general structure of ATP is Adenosine-Pi-Pi-Pi where Pi is inorganic phosphate. The bond between the second and third phosphate can be cleaved generating Adenosine diphosphate (ADP), Pi, and energy. The energy is subsequently utilized by the body and the ADP is eventually regenerated back to ATP by the breakdown of another substance which contributes a Pi.

Creatine phosphate (CP) system: The CP system is used only for very fast, intense bouts of activity. The body only has the capacity to use creatine as a substrate for about 5-10 seconds. Basically the CP molecule (a creatine + an

inorganic phosphate) is broken down and the phosphate is used to regenerate ADP to ATP which can be used quickly for energy. Then a rest is needed to allow the creatine to reform as CP. which can take upwards of several minutes. This system does not require oxygen to operate (anaerobic) and is very rapid. Thus, this system is only really utilized by strict power athletes (throwers, olympic lifters and very short sprints). It probably has the potential to be improved with training, but doesn't really have a lot of applicability to most athletes (i.e. endurance).

Aerobic glycolysis: This is probably the system that most endurance athletes utilize for the majority of training and racing. It has the greatest potential to generate energy but it requires oxygen to work and is rather slow in working. Thus, it is well suited to sub-maximal, steady state exercise and can generate almost an unlimited amount of ATP from the oxidation of fats and glycogen. However, it is unsuited to high intensity exercise as it is unable to function without adequate oxygen and cannot generate energy quickly enough to meet the body's energy requirements. During aerobic glycolysis, glycogen is broken down primarily to pyruvate as an end product. This pyruvate can be utilized as another source of fuel and recycled into the system. This system is improved by continuous training which increases mitochodrial density and number (which allows the muscle to oxidize fats and sugars more easily). Other adaptations such as increased capillarization (which will increase blood flow and hence oxygen availability) also occur with training.

Anaerobic glycolysis: This is the system that is called into play during high intensity bouts of exercise. When adequate oxygen is not available to the cells to fuel aerobic glycolysis, this system takes over. It can only use glycogen and glucose for fuels (it cannot use fats) but it operates more quickly than aerobic glycolysis although its ability to generate ATP is limited. As opposed to aerobic glycolysis where pyruvate is generated as the end product, anaerobic glycolysis tends to generate our friend lactate during exercise. As previously stated, high lactate levels cause lots of pain and lower the pH of the muscle which shuts down the energy. This system is improved by high intensity training (i.e. intervals). One of the adaptations to interval training is that the Type IIb fibers (which are recruited most during high intensity exercise) actually become somewhat more aerobic. Thus, the point at which lactate begins to heavily accumulate in the blood (lactate threshhold) is higher. Another adaptation is the Type IIb fibers become more able to use lactate as a fuel and recycle it. Basically, the body learns to clear lactate from the blood more quickly which helps offset the increase normally seen with high intensity exercise.

So, why did I bother to go into this discussion? Well, first off, I thought it might be generally helpful to understand exactly what is meant by aerobic and anaerobic glycolysis. Also, when I talk about intervals next time (promise), I will try to tie in how different lengths of intervals may stress the different energy systems differently which will have a bearing on how the interval program is developed.

Ok, well, finally, I would like to talk about interval training. No more digressions this time, just on to the meat of the matter.

Although the difference is really semantical, I would like to differentiate between different types of intervals. After each type of interval's name will be the approximate amount of time for the type of workout. As with weight training, duration of interval and intensity of interval are inversely related so that the harder you go in a given interval the shorter it must be.

1. **Threshhold training:**
   - o **Tempo:** continuous training right at the lactate threshold (20-40 min)
   - o **Cruise intervals:** 2 shorter tempo runs (10-20 minutes each) separated by a period of easy recovery. Threshhold training will help to improve the LT significantly.

2. **Intervals:** intermittent training above the LT and near 100% VO2 max
   Each interval should be between 2 and 10 minutes with a total amount of on-time of 15-25 minutes. Any more time spent on intervals yields little improvement but greatly increases the chance for injury and overtraining. Long intervals will help train the Type II fibers to clear lactate better due to the high levels of lactate generated.

3. **Repetitions:** these are very high intensity intervals at greater than VO2 max. Due to the high intensity, time is limited to 30-90 seconds max. These are useful for improving form and recruitment patterns given sport. Total time per workout is 5-10 minutes. Shorter intervals (sometimes referred to as alactic acid intervals) in the 10-20 second range are limited more by CP stores than by lactic acid buildup and are useful for improving the CP energy system (see last post).

4. **Fartlek:** Scandanavian for speedplay. This refers to unstructured

interval workouts and are very free form. They are a good way to introduce the body to intervals early in the season. They might include easy distance broken up by the occasional hard surge up a hill or sprint.

Depending on the requirements of a particular sport, different lengths of intervals should be performed. This has to do with specificity of adaptation. For example, 100 meter sprinters should emphasize running 100 meter repetitions (or slightly longer) at top speed. Also, practice starting would be a good idea as frequently races can be won or lost in the start. By contrast, tempo intervals would not be as useful for sprinters as it would not be sports specific since no sprinter will ever run more than a few minutes at most continuously.

For cyclists and other endurance athletes, probably some combination of the above workouts would be necessary. During longer races (i.e. a marathon) most of the race will be spent at about the lactate threshhold. However, there will be times when either short surges are needed (10-30 seconds) to catch a competitor or longer surges (several minutes) will be necessary to crank out a really fast mile to work on a lead. Also, having the ability to sprint at the end of a long race might also make the difference between winning and losing. To train for a final sprint, you might do a threshold workout and try to end with some short intervals. The possibilities are endless.

But who else can interval (and other high-intensity training benefit). For the recreational exerciser, interval training is probably not all that necessary unless you want to reach new levels of cardiovascular fitness. However, some recent evidence (which I posted a few months ago) seems to indicate that high intensity training (not necessarily intervals) may actually be as much or more effective than long slow exercise for fat loss due to the sheer number of calories burned. Yes, the percentage of fat burned won't be as high during high intensity exercise but, since the absolute number of calories burned will be higher, the total number of calories from fat will be the same or more. And, high intensity work allows you to get done more quickly. If you're interested, I'll be happy to re-post my article about the "Fat Burning Myth" which gives a bit more detailed info about this.

But, who else?? What about the recreational competitor? Well, again, it's sort of debatable. If you want to really reach new heights with your competing, some sort of interval training will be necessary to improve your speed in your chosen sport. Also, there's one other type of athlete that I think can benefit from intervals: the intermittent sport athlete. By that, I mean, persons who are

involved in sports which are stop and go rather than continuous. Some examples would be tennis, basketball, racketball and similar activities. Although many people would consider these to be aerobic, when you think about it, they are really just a series of short, high-intensity bouts of exercise. For example, in racketball, you might play for 10-30 seconds for a given point at a very high intensity. Then you rest a few seconds and repeat. Yes, you do need a good deal of aerobic endurance to last the length of the match, but the individual points are really nothing more than intervals.

Well, I will wrap up this segment here and continue into another part. Next time, I plan to talk about a very important part of interval training which is the rest time between intervals. Finally, I will talk about putting together an interval package within the context of a full training program.

Well, as is usual with my posts, this series just refuses to end. But, hopefully, I can bring it to a close in this post. Last time, I talked about the different types of intervals, what purpose they serve, and briefly how they can be applied to different sports. This time, I would like to talk about the rest interval which is another important aspect of interval training.

Rest interval refers to the time spent between intervals recovering from the pain. There are two different apporaches to rest intervals: active and passive rest. Again the utilization of one over the other will depend on the specific nature of the sport.

Active rest: this has the benefit of keeping blood moving through the legs which will help to clear out the lactic acid and aid in recovery. It is quite simply a brief period of low intensity work to allow the body to recover but movement is never completely stopped.

Passive rest: this really has no particular benefits but it's inclusion may be more sports specific. Basically, after the interval, you get to sit still and hurt (err, recover).

For example, rarely in a cycling or running race do you come to a complete stop. There will be times when you will need to speed up or slow down or whatever during the race though. Thus, doing intervals with passive rest probably wouldn't be that applicable. However, with regards to intermittent sport athletes like football , racketball, and tennis players, etc., after the high

intensity bout, you tend to sit still briefly (i.e. huddle, recovering the ball) before going again. Thus, passive rest may be more specific.

Another factor to consider in deciding on rest intervals is on the length of interval. Very generally speaking, the rest interval should be long enough to allow the heart rate to recover to about 120-130 or so. This indicates that the body is ready for another interval. Sometimes, though, you will see the rest period described relative to length of the interval (as in 1:2, 1:3 or whatever). A rest interval of 1:2 would mean that you rest twice as long as the length of the interval before doing the next one (i.e. a 2:00 rest for a 1:00 interval).

However, there are times when you might wish to do intervals with a shorter rest interval than is necessary for total recovery. For example, in cycling races, frequently, a hard surge of several minutes might be followed by a brief respite then another several minute surge. If this type of activity has not been trained, you may not be able to keep up during this period. The drawback to very short rest periods is the huge amount of lactate generated which is very painful. However, this type of training will generate significant adaptations.

Well, as always, this is getting too long, so I'll finish in part 6 with how to use intervals in your workouts.

Ok, so how do you go about integrating intervals into your program. Well,first and foremost, you shouldn't even consider including intervals in your workouts until you've built a sufficient base of six months or more. After this point, you might consider using intervals as a part of your workout. Also, intervals should not be performed year round and should be severely limited (if not omitted) during the off-season. Generally, interval training should begin at the end of the off season, switch into high gear during the months just before competition, and then be curtailed and maintained during racing season when the emphasis should be on racing. I'll talk more about this when I discuss periodization for endurance athletes.

At this point, let me state that there is no set formula for interval workouts. More so than really any other aspect of endurance training, interval workouts are an art rather than a science. So, I can't tell you exactly what will work for you as everyone is different and has different needs and will respond differently. Instead, I'm going to try to give you some suggestions as a starting point and then you can experiment from there.

Personally, I've found that including some very short speed bursts (about 20 seconds) into off-season distance work is one good way to begin acclimating the body to the intensity of intervals. Every fifteen minutes or so during these long workouts, throw in a 20 second burst of speed. The intensity should still remain fairly low but just enough to get your legs moving faster.

Another way to begin intervals is with Fartlek training. As I mentioned before, fartlek refers to the practice of throwing in the occasional speed burst during a workout. For example, during a run in the woods, you might surge up a hill and then run easily for a while and then throw in another little sprint. This type of workout should not be terribly structured (i.e. I'm going to do 4X440 meter repeats with a 1:00 rest interval) and should be fun. They are useful for breaking the body in to interval work.

A third way to introduce the body to intervals is with an interval pyramid. A teacher of mine liked to begin his interval phase by doing 1:00 with a 1:00 active rest, 2:00 with a 2:00 active rest, up to five minutes and then back down to 1:00.

Personally, I've had good success by starting out with relatively short intervals of 1:00 followed by a 1:00 rest doing about 10-20 repetitions. Then as the season progresses, I'll raise the time of the intervals by about 30 seconds per week and bring the number of repetitons down to keep the total on-time between 15 and 25 minutes. I'll eventually get up to 10:00 intervals which are really more of a threshhold interval. Rest time varies as the times increase. When I'm doing short intervals (1-2:00), I generally stick with 1:1 for my rest interval. But, as the intervals increase in length, I have a tendency to keep the total length of each rep (interval + rest time) at 5:00 until I get to 5:00 minute intervals and then 10:00 when the interval is longer than 5:00. Chalk it up to my being anal. However, this doesn't preclude including short intervals though and then I will occasionally throw in some short repetitions on my interval day.

Also, realize that due to the high intensity nature of intervals, you shouldn't do more than two interval workouts (and I prefer one) per week, especially when you begin doing them. Otherwise, you risk overtraining and injury. In animal studies, there was no increase in adaptations between intervals two days per week and six days per week, so it's probably better to go with less rather than more.

Oh, yeah, during an interval workout, you should always begin with a low-

intensity workout of 10-15 minutes, perhaps followed by some stretching. Then you can do your intervals. Be sure to include a cool-down after the intervals to help clear some of the lactic acid away. The cool down should also be 10-15 minutes long. Ideally the cool down and warm up should be the same exercise as the intervals (i.e. if you're running intervals, don't warm up on a bike, warm up running).

Whew. Well, at long last, I've managed to finish out this three part series (yeah, right) on the lactate threshhold and interval training. If you have any sports specific questions feel free to send them my way and I'll do my best to help you. Next time, I will talk about developing year long training programs for endurance athletes and discuss periodization as it pertains to them.

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Lactate Threshold - Implications for Training and Performance

Adapted from information by the Physical Conditioning Department of the USOTC Sports Science and Technology Division

Lactic Acid: When energy is required for muscular work, fat and gylcogen (the storage form of glucose) are broken down to produce molecules of ATP which, in turn are used for muscular contraction. Fat and glycogen can also be broken down to produce energy in the absence of oxygen (anaerobically) while fat cannot. The by-product of anaerobic bearkdown of glycogen is lactic acid.

Lactate Threshold (LT): Resting levels of lactate in the muscle and blood are relatively low - approximately 1 mmol/L. As exercise intensity increases, lactate levels remain fairly constant until a point known as lactate threshold (LT) and it occurs when the breakdown of gylcogen becomes predominantly anaerobic and/or the rate of lactic production exceeds the rate of removal. The LT is also indicative of the rate of glycogen usage. The rate of glycogen usage at intensities above the LT is approximately 18 times faster than for the same energy yield then intensities are below LT. For endurance events where glycogen availability may be a limiting factor in performance, the ability to sustain a high workload before LT is reached is advantageous.

Lactate Threshold and Performance: There is a high correlation between distance running performance and the velocity at which LT is reaches. The treadmill velocity corresponding to LT has been found to be one of the best predictors of marathon and 10K running performance as compared to other commonly used measures (such as VO2 max). The LT, when expressed as a percentage of VO2 max, is relatively high in the endurance-trainined athlete. In sedentary individuals, for example, the LT may occur at 50-60% of VO2 max, whereas in endurance-trained athletes it typically occurs at around 70-80% of VO2 max. This is advantageous for the endurance athlete as they can work at a higher pecentage of VO2 max and therefore faster pace, without large increases in lactate levels.

Lactate Threshold and Training: The LT is very responsive to training. While VO2 max will increase minimally in an endurance-trained athlete, LT will show significant improvements with proper training. Measuring your LT several times throughout the year is important for monitoring your progress as well as allowing you to adapt your training as improvements are made.

Generally speaking, the threshold/breakpoint on your graph will occur at a slightly lower lactate level than what you can maintain during a race, thus your LT intensity may represent a minimum intensity for LT or tempo training. Training beyond the LT is also important for performance in certain events as it enables the athlete to sustain intense hours of exercise and to tolerate the high lactate levels which accompany such efforts. However it is important to be aware that very high lactate values resulting from high-intensity workouts may have deleterious effects. The acidosis associated with high lactate levels can cause damage to the muscle cell wall which may take from 24 to 96 hours for recovery. Thus it is important to allow sufficient rest following a high intensity workout.

The best way to train using your LT is to train at heart rates and/or paces which correspond to various lactate levels. For examples of the kind of training you can do using your LT, read the next section Levels of Training.

The Lactate Threshold

In exercise physiology, there have been few topics more frequently investigated, or more vigorously debated than the lactate threshold. It is the details, not the basics that create the big research problems. However, it is the basics that have great application to training and performance. So, we'll stick to those.

What is Lactic Acid and Where Does it Come From?

When you consume carbohydrate, it consists of several different sugar molecules; sucrose, fructose, glucose to name a few. However, by the time the liver does its job, all of this sugar is converted to glucose which can be taken up by all cells. Muscle fibers take up glucose and either use it immediately, or store it in the form of long glucose chains called glycogen. During exercise, glycogen is broken down to glucose which then goes through a sequence of enzymatic reactions that do not require oxygen to proceed. All of these reactions occur out in the cell fluid, or cytosol. They can occur very rapidly and yield some ATP in the process. This pathway is called the anaerobic (no oxygen) glycolysis (glucose breakdown) pathway. Every single glucose molecule must go through this sequence of reactions for useful
energy to be withdrawn and converted to ATP, the energy molecule, that fuels muscle contraction, and all other cellular energy dependant functions.

The Metabolic Fork in the Road

There is a critical metabolic fork in the road at the end of this chemical pathway. At this fork, glucose has been converted from one 6 carbon molecule to two, 3 carbon molecules called pyruvic acid, or pyruvate. This pyruvate can either be shuttled into the mitochondria via the enzyme pyruvate
dehydrogenase, or be converted to lactic acid via the enzyme lactate dehydrogenase. Entry into the mitochondria exposes the pyruvate to further enzymatic breakdown, oxidation, and a high ATP yield per glucose. Conversion to lactate means a temporary dead end in the energy yielding process, and the potential for contractile fatigue due to decreasing cellular pH

if lactic acid accumulation proceeds unchecked. Like a leaf floating in a river, the pyruvate molecule has no "say" in which metabolic direction is taken.

Which Way will MY pyruvate go during exercise?

I am sure you have surmised that that is a critical question with big implications for performance. I will try to answer the question at three levels: a single muscle fiber, an entire muscle that is active during exercise, and the entire exercising body.

The Muscle Cell at Work

In a single contracting muscle fiber. The frequency and duration of contractions will determine ATP demand. ATP demand will be met by metabolizing a combination of two energy sources: fatty acids and glucose molecules (ignoring the small contribution of protein for now). As ATP demand increases, the rate of glucose flux through glycolytic pathway increases. Therefore at high workloads within the single fiber, the rate of pyruvic acid production will be very high. If the muscle fiber has a lot of mitochondria (and therefore more Pyruvate Dehydrogenase), pyruvate will tend to be converted to Acetyl CoA and move into the mitochondria, with relatively little lactate production. Additionally, fatty acid metabolism will account for a higher percentage of the ATP need. Fat metabolism does not produce lactate, ever! If lactate is produced from glucose breakdown, it will tend to diffuse from the area of high concentration inside the muscle cell to lower concentration out of the muscle fiber and into extracellular fluid, then into the capillaries.

The Whole Muscle at Work

Now let's look at an entire muscle, say the vastus lateralis of the quadriceps group during cycling. At a low workload, glycolytic flux is low and the pyruvate produced is primarily shuttled into the mitochondria for oxidative breakdown. Since the workload is low, primarily slow twitch fibers are active. These fibers have high mitochondrial volume. As workload increases, more fibers are recruited and recruited fibers have higher duty cycles. Now ATP demand has increased in the previously active fibers, resulting in higher rates of pyruvic acid production. A greater proportion of this now is converted to lactic acid rather than entering the mitochondria, due to competition between LDH and PDH. Meanwhile, some Fast twitch motor units are starting to be recruited. This will add to the lactate efflux from the muscle due to the lower mitochondrial volume of these fibers. The rate of lactate appearance in the

blood stream increases.

The Body at Work

The vastus is just one of several muscles that are very active in cycling. With increasing intensity, increased muscle mass is called on to meet the force production requirements. All of these muscles are contributing more or less lactic acid to the extracellular space and blood volume, depending on their fiber type composition, training status and activity level. However, the body is not just producing lactate, but also consuming it. The heart, the liver, the kidneys and inactive muscles are all locations where lactic acid can be taken up from the blood and either converted back to pyruvic acid and metabolized in the mitochondria or used as a building block to resynthesize glucose (the liver). These sites have low intracellular lactate concentration, so lactic acid diffuses INTO these cells from the circulatory system. If the rate of uptake or dissappearance of lactate equals the rate of production or appearance in the blood, then blood lactate concentration stays constant (or nearly so). When the rate of lactate production exceeds the rate of disappearance, lactic acid accumulates in the blood volume, then we see the ONSET of BLOOD LACTATE ACCUMULATION (OBLA). This is the "Lactate Threshold" (LT).

Performance Implications

Lactic Acid production is not all bad. If we could not produce lactate, our ability to perform brief high intensity exercise would be almost eliminated. However, As I am sure you are aware, lactic acid is the demon of the endurance athlete. Cellular accumulation of the protons (increased acidity) that dissociate from lactate results in inhibition of muscle contraction. Blame those heavy legs on the protons! The bottom line is that exercise intensities above the OBLA point can only be sustained for a few minutes to perhaps one hour depending on how high the workload is above the intensity at OBLA. Exercise at or below this intensity may be sustainable for hours. The causes of fatigue at these sub-LT intensities include carbohydrate depletion and dehydration.

Factors that Influence the Rate of Lactate Accumulation in the body

• **Absolute Exercise Intensity** - for reasons mentioned above.

• **Training Status of Active Muscles** - Higher mitochondrial volume improves capacity for oxidative metabolism at high glycolytic flux rates. Additionally, improved fatty acid oxidation capacity results in decreased glucose utilization

at submaximal exercise intensities. Fat metabolism proceeds via a different pathway than glucose, and lactic acid is not produced. High capillary density improves both oxygen delivery to the mitochondria and washout of waste products from the active muscles.

• **Fiber Type Composition** - Slow twitch fibers produce less lactate at a given workload than fast twitch fibers, independent of training status.

• **Distribution of Workload** - A large muscle mass working at a moderate intensity will develop less lactate than a small muscle mass working at a high intensity. For example, the rower must learn to effectively distribute force development among the muscles of the legs back and arms, rather than focusing all of the load on the legs, or the upper body.

• **Rate of Blood Lactate Clearance** - With training, blood flow to organs such as the liver and kidneys decreases less at any given sympathetic stimulation. This results in increaed lactate removal from the circulatory sytem by these organs.

Measuring the Lactate Threshold

We have previously discussed the value of a high maximal oxygen consumption for the endurance athlete. A big VO2 max sets the ceiling for our sustainable work rate. It is a measure of the size of our performance engine. However, the Lactate Threshold greatly influences the actual percentage of that engine power that can be used continuously.

Most of you will never have this measured in a laboratory, but a brief description of a lactate threshold test is still useful, because it will lead us into some specific applications for your racing and training. The test consists of sucessive stages of exercise on a treadmill, bicycle ergometer, swimming flume, rowing machine etc. Initially the exercise intensity is about 50- 60% of the VO2 max. Each stage generally lasts about 5 minutes. Near the end of each stage, heart rate is recorded, oxygen consumption is measured, and a sample of blood is withdrawn, using a needle prick of the finger or earlobe. Using special instrumentation, blood lactate concentration can be determined during the test. After these measurements, the workload is increased and the steps repeated. Through a 6 stage test, we would expect to achieve a distribution of intensities that are below, at, and above the intensity of OBLA or the lactate threshold. The data from a test would generally look simililar to the example below.

Interpreting the Data

For purposes of interpretation, let's say that the athlete above had a maximal heart rate of 182, and a VO2 max of 61 ml/min/kg. These were also determined using a bicycle test. So they are good values for comparison. Looking at the green dots, we see that blood lactate concentration does not begins to increase until during the 4th workload, from a concentration of about 1 mM to 2.5 mM. This is the break point. The subjects VO2 was 45 ml/min/kg at this point. So we determine that his LT occurs at 45/61 or about 74% of VO2 max. If we look at the heart rate at this point, it is 158. Now we have a heart rate at lactate threshold. 158 = about 85% of his max heart rate. This is useful for the athlete. When he is cycling, he can judge his training intensities based on this important value. If he is a time trialist, this would approximate his racing heart rate for the hour long event. It is important to understand that the % of VO2 max and the % of max heart rate at the LT are not necessarily the same %age, as demonstrated in this example.

So, Do I race at My LT Intensity?

This depends on your race duration. If your are rowing 2000 meters, running a 5k race etc, your exercise intensity will be well above the AT. Consequently, the blood lactate measured after these events is extremely high in elite athletes, on the order of 15mM (resting levels are below 1 mM). In races lasting from 30 minutes to 1 hour, well trained athletes also perform at an intensity above LT, but by a smaller margin. It appears that in these events, top performers achieve what might be termed a "maximal lactate steady state". Blood lactate may increase to 8 to 10 mM within minutes, and then stabilize for the race duration. A high but stable lactate concentration may seem to contradict the idea of the LT. But, remember that blood lactate concentration is the consequence of both production and clearance. It seems likely that at these higher lactate concentrations, uptake by non-working muscles is optimized. At any rate, measurements in cyclists, runners and skiers demonstrate the fact that elite performers can sustain work levels substantially above the LT for up to one hour.

Specificity of the Lactate Threshold

It is important to know that the lactate threshold is highly specific to the exercise task. So if this cyclist tries to get on his brand new, previously unused, rowing machine and row at a heart rate of 158, he will quickly become

fatigued. Rowing employs different muscles and neuromuscular patterns. Since these muscles are less trained, the cyclist's rowing LT will be considerably lower. This specificity is an important concept to understand when using heart rate as a guide in "cross training activities", as well as for the multi-event athlete.

Effect of Training

For reasons mentioned above, training results in a decrease in lactate production at any given exercise intensity. Untrained individuals usually reach the LT at about 60% of VO2 max. With training, LT can increase from 60% to above 70% or even higher. Elite endurance athletes and top masters athletes typically have LTs at or above 80% of VO2 max. Values approaching 90% have been reported. The lactate threshold is both responsive to training and influenced by genetics.

Your Lactate Threshold (LT) is the point at which your muscles start producing more lactic acid than they can eliminate. We can get a good idea of when this occurs by relating it to an athlete's maximum heart rate. **Most athletes reach their LT when they achieve around 90% of their max HR.** Training at or near your threshold (zone 4) produces a couple of desirable training effects. First, it gets your muscles used to working with the presence of lactic acid. This is called t**olerance**. Secondly, with proper training in this few beats per minute, allowing you to ride harder (faster) before the accumulation of lactic acid begins. This training is vital for all disciplines of bicycle racing. This is where you live during a long time trial, a solo or small group break in a road race, or for the entirety of a cyclocross or mountain bike race.

We start LTs after at least one full 5-week mesocycle of base training. We then enter a 5-week LT mesocycle where we concentrate on this type of workout up to 3 times per week. LTs are continued throughout the season with the frequency depending on the athlete and the discipline. So we begin. Your first LT workout will probably be 2x10 min. intervals in zone 4 with 10 min recovery between work phases. The following workouts will increase the work phase gradually with specific amounts of recovery prescribed. During the recovery phase your HR should drop to zone 2, but not below. If you let your HR slip down into zone 1 you will find it more difficult to get back into the work phase. Also, during recovery, keep your cadence high around 100 RPM. The work phase begins as soon as you start the effort not when your HR gets to zone 4. Don't sprint up to zone 4; gradually push yourself there over 45 seconds or 1 minute. These workouts can be done inside or out, but make sure you warm

up for at least 30 minutes prior to the first work phase and cool down for at least 30 minutes after the last. Outside try to ride a course that has no or few stops for each work phase (possibly a 3.5 mile loop or an out and back). Don't worry if you do have to stop, but keep in mind non-stop is optimal. Here are a couple of ideas to spice up the workouts: Try doing the work phase on a long gradual hill (avoid any steep downhills where your HR can drop too much). Try "criss-crossing" in your zone. Start the work phase, get to zone 4, and continue accelerating gradually over a couple of minutes until you reach the top of the zone. Once you reach the top, ease off slightly and let your HR fall to the bottom of the zone. Repeat this several times until you have reached the end of the work phase.

These workouts should be hard, but they should not destroy you. If you have trouble reaching your zone 4 after about 5 minutes of trying, you are probably too tired, sick, or not prepared for the workout. Stop, ride easy or go home and rest. Let me know as soon as you can if this happens, so we can make adjustments to your schedule.

Lactate Threshold

LT is also known as anaerobic threshold. The LT is the level of effort (usually expressed as a percentage of VO2 max.) at which the body begins to produce more lactate than can be removed. Above this point there is a rapid increase in blood lactate levels. Some physiologists also call it the pulse rate deflection point (Conconi Test). It is also the maximum effort you can maintain for a long periods. Obviously the more you exceed your LT, the more quickly lactic acid will accumulate and impair your performance.

As most cyclists don’t have access to lab facilities, you can estimate your LT with a 30 minute (about 10 mile) time trial. The average heart rate you can maintain is a good approximation of your LT.

The LT will improve with training, and cyclists with a higher LT can work at a higher level of energy expenditure for longer periods, defeating opponents of equal (or even greater) physical strength but with lower LTs. Anaerobic intervals will improve the LT and are designed to be done at heart rates above your personal LT. They can be sustained for only 15 sec to 2 minutes, but will improve your LT fitness.

Recent work has focused on the blood lactate threshold (LT) as a reflection of

an individual's level of training. The lactate threshold is that % of VO2 max. at which the cardiovascular system can no longer provide adequate oxygen for all the exercising muscle cells and lactic acid starts to accumulate in those muscle cells (and subsequently in the blood as well). At high levels of activity (below 100% VO2max), it is likely that there are always a few muscle cells (not muscles, but a small number of cells within those muscles) that are relatively deficient in oxygen and thus producing lactic acid. But this lactic acid is quickly metabolized by other cells that are still operating on an aerobic level. At some point, however, the balance between production of lactic acid and its removal by body systems shifts towards accumulation. This point is the LT and it is usually slightly below 100% VO2 max, and it will improve with training (move closer to 100% VO2max). As those with an increased LT not only experience less physical deterioration in muscle cell performance but also use less glycogen for ATP production at any level of performance, an improvement in LT allows the individual to perform at maximal levels for a longer period of time before muscle performance deteriorates because of a lack of adequate energy (glycogen) stores.

14) Training with Periodization by Chad Butts

Part 1: Introduction

This is the first article in a series written for www.spokepost.com on the periodization of weight training for cyclists looking to improve there strength and power during the long winter months of upstate New York.

Cyclists looking for peak performance FROM a strength-training program must realize that the days of going to the gym all winter and always lifting till you could barely walk out of the gym are gone. To get the most FROM resistance training, the program needs to be periodized, just like training on the bike. There will be easy days and hard days, easy weeks and very hard weeks. For example, take a yearly training plan for bike volume. At the start of the season your volume slowly progresses to a maximum point before decreasing to accommodate increases in training intensity. The same should be true with weight training. Periodization will allow you to monitor and change the level of intensity throughout the phases of your program to ensure the proper stimulus and provide enough recovery. Training on the bike is also devoted to more than one technique or system. There are intervals, endurance rides, threshold intervals, sprints, etc., each of these developing a different system. Likewise, a periodized strength program works different systems at different times to maximize the development of strength and power.

There are basic principles to any strength-training program as well. One of the most important is using proper form during exercises. Never increase the weight if you cannot perform the exercise properly in the same manner and range of motion. Tendons and ligaments take longer to adapt to a strength program than muscle. It is very important to give them time to adapt to the program or a whole winters worth of weight training may be ruined because your knees cannot take it when the LOAD increases. A proper warm-up is also a very good idea. The more intense the session, as later in the program during the maximum strength phase, the longer the warm-up should be. Look for doing 5-10 minutes on the bike or stepper followed by 2 sets of 15 squats with just the bar. After that some light stretching to get the muscle good and loose. Developing abdominal and back strength and endurance is very important for the weight-training cyclist. Your legs constantly push and pull on the pelvis each pedal stroke and it is the abs and back that stabilize the pelvis, which the legs work from. The less stabilization you provide, the less power you can produce.

Because cyclists are training for strength and power on the bike your exercises

need to simulate, as close as possible, your position on the bike. Whenever you are performing an exercise you need to ask yourself if you are as close to your riding position as possible. Is your stance the same distance apart when clipped INTO your pedals? Are you training the same range of motion as your pedal stroke? What is your foot angle? Are you pushing with the balls of your feet and not your heels? Every nuance you can think of to make your training specific to cycling will enhance the specificity of your exercises and enhance the amount of strength/power you can transfer to the bike.

Other considerations when starting a strength program is the amount of time you have to devote to the program, how many days per week, what exercises and what ORDER to perform them, number of repetitions, training method, training load, rest interval between sets, number of sets, etc. Each one of these will vary depending on the phase you are in.

Of course there are many other components to a properly periodized resistance program and they change based on the goals of the athlete. For cyclists, a resistance program needs to be based around muscular endurance, the primary factor determining endurance success. Power endurance must also be addressed in a cyclists program because power is no good unless you can sustain it.

Periodized weight training involves a long-term plan broken up INTO specific phases, each training a specific component (i.e. energy systems, hypertrophy, power, etc.). In ORDER to get the most out of your program it must be organized ahead of time. Creating the program, and doing it correctly, is the first step. A template to train by provides more objectivity for your workouts and keeps you ahead of your workouts so all of your focus can go INTO that session rather than planning ahead of time. Just do what your program tells you. At best your program will meet all of your expectations and produce all of the results you expect. At worst, your program will fail to produce the results you expected but at least you have a program of what was done which, with the help of a coach or trainer, can be changed. Next year you will have a program that is more suited to you.

The annual training plan should be broken up INTO 5-6 DISTINCT phases, depending on your goals and weaknesses. Since cyclists typically do not weight train the whole year, a typical program may run FROM Oct./Nov. to March/April. Each of the specific phases within the plan contains a certain number of microcycles or weeks. It is important that before each phase you test your 1-repetition maximum (RM) for each exercise. This is how you will determine the

LOAD for each exercise. It is much safer and more practical to do a 3-5 RM test for each exercise and then estimating your 1RM using a maximum weight chart.

A good weight program for cyclists should include an anatomical adaptation phase (AA), a maximal strength phase (MxS), a power phase (P), endurance phase (End), and maintenance phase (M). Depending on the amount of time and previous training history of the athlete, a hypertrophy phase (H) can also be added.

Each one of these phases has specific requirements for the load, # of reps, # of sets, rest interval, speed of execution, etc. For this first installment we will look at each of these factors and how they can affect the workout and in the articles to come we will look in depth, at each phase of a well-periodized strength program.

Training volume is the quantity of work performed. In its truest sense, volume is the total amount of weight lifted and can be calculated for a single session, microcycle, or macrocycle. This number is the amount of weight lifted per rep., multiplied by the reps/set, multiplied by the sets per exercise. Adding each of the exercise totals will give you that session's total volume. Performance gains occur when this volume is progressively increased with adequate recovery over a period of time. That is what a periodized program is. Novice strength trainers must start slower and accumulate less yearly volume than a veteran weight trainer to avoid detrimental overuse injuries. Muscular endurance and maximum strength training requires a large volume of training due to the higher loads and increased number of repetitions performed.

Intensity for a strength-training program should be prescribed using percentage of 1RM (maximum amount of weight that can only be lifted once). As stated earlier, 1RM can be calculated safely by warming up properly and performing each exercise at a weight that you can lift approximately 3-5 times. Once you have found a weight that you can lift in this range look up the weight and number of repetitions on a maximum weight chart to see what your estimated 1RM would be. It is much safer and just as accurate this way than to try and test an actual 1RM! The minimum LOAD required to see strength gains is 60% of 1RM. The only time you should be below a 60% LOAD is during the adaptation phase (first one) and during recovery weeks. The LOAD is determined by the phase and what adaptations you are trying to develop.

The number of sessions per week is a big issue with a lot of people because of time constraints. The amount of time you need to spend in the gym depends on experience and how well you respond or recover to each training phase. For the

most part, an experienced weight lifter and can tolerate 3x/wk the benefits will be greater than a 2x/wk schedule. However, there is always the law of diminishing returns, which requires consideration. Also, a novice may be able to handle 3x/wk during the adaptation phase but may have to switch to 2x/wk during the strength phase because he/she cannot adapt quickly enough. It is most important to get 3 sessions during the initial phases of the program, when LOAD is low and most of the benefit is coordination and tendon/ligament strength. During the later phases when LOAD begins to increase it is less of a benefit to have 3 sessions/wk because you will need to recover, as fast as possible and for some people, novices and very heavy training, 3 days is too much to allow full recovery.

The number of exercises should depend on the length of time spent in the gym. For the most part, especially during the high intensity cycles, you should not spend much more than an hour in the gym at a time. After an hour, hormones circulating in the blood take a nose-dive, and further work can be detrimental to the building and recovery process.

The number of sets, repetitions/set, lifting method, rest interval, and selection of exercises all depend on the phase you are in and the adaptations you are looking for. Each one of these we will go over in more detail in the coming articles.

Part 2: Adaptation Phase

Muscle is a highly trainable and adaptable tissue. However, the tendons and ligaments surrounding the joints are not as adaptable as muscle tissue and require more time to adjust to the stress of strength training. Therefore, it is necessary to start a strength-training program with a period devoted to progressively adapt the muscles and support structures (ligaments, joints, tendons, etc) for the intense training of later phases. Muscles will always adapt faster than tendons and ligaments. The main goal during this adaptation phase is overall body conditioning not just of the leg muscles, but the abdominal and back musculature as well. Upper body exercises could also be done during this phase. It is important that intensity start at a low level, approximately 40-50% 1RM and increases be made slowly after complete recovery from the previous sessions. If done properly, this phase will build joint integrity, which can prevent overuse injuries later on in the season and earlier injuries resulting from intense strength training. There is plenty of time during later phases for cycling specific exercises, so for now focus on a balanced program working both sides of the joint, flexors

and extensors, agonists and antagonists.

The amount of time to schedule for the adaptation phase largely depends upon strength training experience. The more experienced, the less time required for adaptation. If you have strength trained for the past 3-4 winters but have not in the past 6-months then you should still schedule 6-weeks of adaptation. Those just starting a strength-training program should schedule at least 8-10 weeks of adaptation. Many athletes fail to start strength training early enough to allow 8-weeks of adaptation and end up with an injury. Adaptation is a very important phase because it primes the muscles and support system for the rest of the program. Insufficient adaptation here will lead to higher injury rates and lackluster improvements later on.

The number of exercises should fall within the 10-12 range. Any more than that makes training volume too stressful and impractical and any less makes a well-balanced whole-body workout difficult. The lower work intensities of this phase make it possible to perform more exercises in a session, training the muscles and joints in a multitude of ways. The best method for a large number of exercises at relatively low intensities is circuit training. Circuit training is accomplished by alternating muscle groups or exercises one after another until all of the exercises are gone through once. Then after a brief rest period they are run through again. This continues until all sets are completed. After each set you quickly move to the next exercise giving little chance for heart rate to recovery. This method also trains the cardiovascular system for this reason. Because you are training opposing muscle groups you can quickly move from one exercise to the other while also getting the proper recovery before working the same muscle again. Recovery between sets or exercises should be 20-50 seconds and 2-3 minutes between circuits.

Before beginning the adaptation phase it is important to test for 1RM. The best way to do it is to test for 3-5 RM (weight you can accomplish in good form for 3-5 repetitions) and then using a weight chart to estimate your 1RM.

The types of exercises in this phase should include:

1. Abdominal - Pick 3 - 4 exercises and rotate through them, two one day and another two the next session. Try to get at least these exercises: obliques, crunches on a stability ball, and reverse curls.

2. Back - Depending on how your back responds will determine how many exercises you can do in one session. The low back is used constantly and takes

longer to recover to strength training and is more susceptible to injuries so be careful and pay attention to proper form when lifting. To start pick one exercise, such as the dead lift or back extension and stick with it for 2-3 weeks. After 2-3 weeks try and add the other and begin to do both during a session.

3. Multi-Joint Exercises - squats, lunges, step-ups - These are the best exercises for building base strength and support. These exercises are also good for developing general strength and stability.

4. Single-joint Exercises - Knee extensions, hamstring curls - These are great ways to target a selected muscle group. Make sure to work both sides of the joint equally or run the risk of injury due to poorly balanced joints.

5. Leg Press machines - These are good exercises for targeting the prime movers of the leg without stressing the abdominal and back muscles.

6. Upper Body Exercises - Some upper body exercises can help negate the initial soreness in the shoulders and arms felt during those first few weeks of long distance rides. But upper body strength is debatable for cycling unless you are looking at improving your sprint. However, it can be beneficial to have a little upper body strength to help prevent serious injuries during a crash. If you are one of those people who gains mass easily then one day a week of upper body weights is good enough to get benefits without adding a lot of weight.

The order of exercises is important and can have an impact the quality of your training sessions. Try not to group exercises that stress the same area of the body close to one another. For example, don't try free squats after doing back extensions or dead lifts. The back exercise may limit or affect free squat performance and chances are you will not be working the legs enough and stressing the back too much. It is less important at the beginning of the phase but becomes more important later when loads increase.

After you have tested your 1RM it is necessary to determine the loads for each exercise. Certain abdominal and other exercises do not require large weights or 1RM testing but instead are trained by performing the exercise to failure a number of times. For all other exercises start at 40-50% 1RM. If you are just starting weight training and have not had any previous sport training then start at 40% 1RM. As you progress through the phase the load will increase and repetitions will decrease. This will continue until a peak of 70% 1RM for this phase. Once you are able to handle 70% 1RM comfortably it is time to move to the next phase. An example of a 10-week loading program follows:

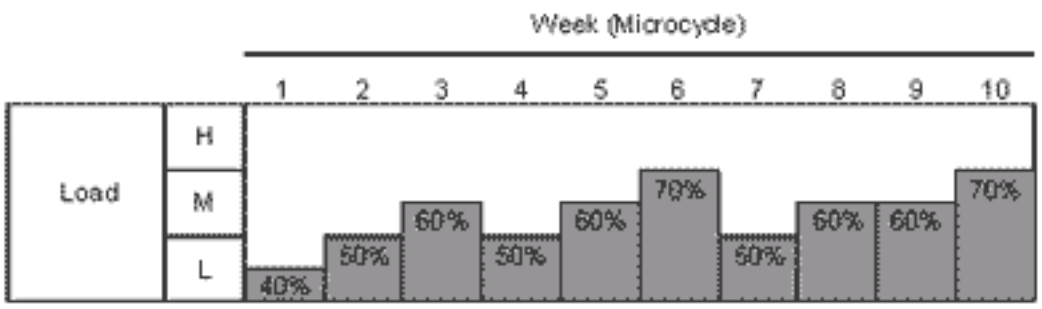

Figure 1. Load (%1RM) progression for a 10-week adaptation phase

The other part of training volume is repetitions and sets. The number of repetitions should be reduced over the weeks as load increases starting with 15-20 for the large multi-joint exercises and 12-15 for single joint exercises. As the weeks continue and the load jumps to the 60-70% 1RM range you should drop the repetitions to 12-15 for the multi-joint exercises and 10-12 for the single joint exercises. Begin with two sets for the first two weeks of the program and then to three on week three. Start the next recovery (Week 4) with two sets and then jump back up to three for weeks 5 and 6. Jump back down to 2 sets for the 7th week and back up to 3 for the remaining 3-weeks. Remember that during weeks 1,2,4, and 7 you are back to 15-20 repetitions.

The main goal of this phase is to progressively increase the training volume. Remember that volume is LOAD X REPS X SETS. So lets take get some data for the aforementioned example. Say for a given exercise your 1RM is 200 lbs. So we begin week 1 exercising at 80lbs (40% 1RM) performing 15 repetitions for 2 sets. The volume for this one exercise once per week is 2400 lbs. If we were to figure out the weekly volumes for the rest of this phase for this one exercise it would look something like this:

	Week									
	1	2	3	4	5	6	7	8	9	10
Volume (lbs)	2400	3000	4320	3000	4320	5040	3000	4320	4320	5040

Figure 2. Weekly training volume for one exercise per week.

Keep in mind that this is only one exercise and these volumes are only one session a week but the principle remains the same and when additional

exercises are added and the frequency increases to 3 times per week only absolute volumes will change while the relative load increases and decreases per week would remain the same. You can see the progression in volume over the course of the 10-weeks followed by recovery weeks to provide the proper amount of rest. A program like this will prepare and strengthen the muscles and support system causing a smooth transition to the next phase when load increases even more.

Try to get three days per week during this phase. It is important that while the load is low, the muscles and support structures need as much stimulus as possible.

The next article will discuss hypertrophy training, the next phase, and why it may or may not be good for cyclists. Thanks for reading.

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Part 3: Hypertrophy Phase

Should cyclists and other endurance athletes focus on building muscle mass during their strength-training program? That is a question that arises among many cyclists and coaches, "I want to get stronger, but I don't want to gain a lot of mass and weight." First we must realize that bigger does not mean stronger. Bigger individuals do not necessarily have a lot of lean muscle mass, which is what makes you strong. The larger the muscle, the more force it can produce, plain and simple. So should cyclists and other endurance athletes include a hypertrophy, muscle building, phase into their strength-training program?

It is no big surprise that the highest rated cyclists within the USCF (United States Cycling Federation) also have the highest anaerobic power outputs. This higher anaerobic power gives these cyclists the ability to attack, respond, climb short steep hills, and sprint faster than those with less anaerobic power, which enhances their chance of success in competition.

Studies researching the effects of weight-training and cycling performance show improved short-term anaerobic power performance as well as long-term cycling performance signified by increased time to exhaustion at a given sub-maximal

workload (Hickson et al.1988, Marcinik et al. 1991). The increase in cycling performance of the aforementioned studies correlate well with the increase in leg strength of the subjects involved. However, the muscular adaptations responsible for the increases in anaerobic power and work capacity are still unknown. These gains in performance may be the result of increased muscle fiber size and the changes in contractile properties induced by strength training. The increased myofiber size following weight training may improve slow twitch muscle fiber velocity (VMax) and reverse the decline in VMax of fast twitch muscle fibers and peak tension development in all fibers (Fitts and Widrick 1996). Since larger, stronger, faster muscle fibers generate more force, cyclists who strength train, and include a specific hypertrophy phase to increase muscle fiber size, may be able to exercise or perform longer at a given sub-maximal workload due to stimulation of less muscle mass and reducing the force contribution from each active muscle fiber.

The basic premise behind this hypertrophy training is that a bigger muscle is more forceful. A muscles cross-sectional area is directly related to its strength. By adding a muscle building (hypertrophy) phase in our strength-training program, we can maximize the size of the muscle early on, when endurance training volume is low, and maintain these bigger muscles throughout the rest of the program. Many do not realize that endurance training actually reduces the size and power of the muscles over the course of the training year. By starting the season with larger muscles, you will end the year with more muscle mass than if you had not weight-trained at all.

To get the best results of hypertrophy training the best results come from the method of body builders. It is important to realize that this phase does not cause nervous system adaptation and mostly results in increased muscular size. Training the nervous system will be the focus of later phases. This method of training will be focused on the prime movers of cycling, the glute, hamstring, and quadricep muscles. The primary objective of this phase is to cause large chemical changes in the muscle, which affect the muscle fiber components. This method uses moderate loads, 70-80% 1RM, while performing as many repetitions as possible, usually between 8-12. It is very important to execute the maximal number of repetitions possible, to the point of muscle failure, and in some cases continuing to hold the contraction as long as possible. The goal is to maximally fatigue the muscle. Obviously there are certain exercises where this method will be contraindicated, i.e. free squats, and advised only with a spotter.

To reassess your strength after the adaptation phase and update your training loads you should re-test for 1RM. Begin the hypertrophy phase lifting 70-80%

1RM, one that allows only 10-12 repetitions. If you can perform 15 repetitions it is time to increase the weight until you are back within the 10-12 range.

The number of exercises will decrease form the previous phase to 5-8. All of these exercises should be the prime movers of cycling while continuing abdominal and back exercises. The speed of execution should be moderate to fast during the concentric (lifting) phase of the exercise and slower for the eccentric (lowering) phase to simulate the speed of contraction used during cycling.

Perform between 4-6 sets of each exercise and allow 2-3 minutes of rest between each set and 3-5 minutes between each exercise. Remember, the goal is to exhaust the muscle, so start with an exercise and complete all sets before moving to the next one.

Be sure to stretch the muscles following the workout. A stretched muscle will recover much faster by aiding blood flow to the area and maintain muscle fiber length. It is also important to replenish energy stores following such exhausting workouts. Proper nutrition will aid in training recovery and allow you to train as hard as possible.

There are many other methods of hypertrophy training, each with their own masochistic twist but the difference between them is most likely marginal as long as the basic concept of muscular failure is followed.

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Part 4: Strength Phase

Following the hypertrophy phase comes the strength phase. During this phase loads increase to their maximum level and focus is shifted to many brief all-out sets designed to maximally stress the nervous system and large muscle fibers. It is during this phase that we take advantage of the muscle mass gained from the last phase and start training the nervous system to recruit as much muscle as possible.

The ability of the muscle to produce maximal strength is largely, but not solely, determined by it's cross sectional area (CSA). This is why the strength phase is often preceded by a hypertrophy phase. However, strength is also determined by the ability to recruit a large number of muscle fibers and training the muscle to recruit them in proper synchronization. Since we have done our best to increase muscular size, this phase will focus on maximal stimulation of the muscle fibers and training proper synchronization of the nervous system to these muscles.

A Lifting weight is composed of several phases, the concentric (lifting phase), isometric (static) phase, and the eccentric (lowering) phase. Cycling is somewhat unique in that it only has concentric contractions. The pedal stroke is a powerful concentric contraction followed by a recovery phase and repeats over and over. Since the method of lifting should be specific to the sport of cycling, most of the emphasis should be placed on the concentric phase of the lift, contracting in an explosive burst of power. However, eccentric contractions (lowering) should not be ignored and is actually the portion of the lift that produces the most tension within the muscle. So do not start a concentric-only lifting routine simply for specificity sake, but really concentrate on the lifting phase, more on this later.

In contrast, the abdominal and back musculature is in a constant state of isometric contraction to stabilize the pelvis while cycling. These muscles are never shortening (concentric) or lengthening (eccentric) to a large degree but are still producing a lot of force. It is the similar to pushing against a brick wall. You are not moving the wall so your muscles are not shortening. The wall is not moving you so your muscles are not getting longer (eccentrically), yet your muscles are still producing force. This is what the "core" musculature is doing the entire time you are cycling. They contract enough to hold the pelvis stationary providing a solid base of support from which the legs can produce power. When these core muscles fatigue at the end of long rides or from long periods of high power output, as in a time trial, they cannot stabilize the pelvis as efficiently resulting in back pain and a loss of power. So specific training of the abdominal and back muscles for the sport of cycling requires isometric and endurance

exercise. These exercises should be added to your routine during the strength phase and rotated with other “core” exercises (crunches, back extensions, etc.).

During the strength phase the goal is to create maximal tension within the muscle, activating as many fast twitch (FT) muscle fibers as possible. This requires lifting very heavy loads that result in a high recruitment of FT fibers from the first repetition rather than eventually stimulating them as others fatigue, like hypertrophy training.

Exercises in this phase should not be performed under a state of fatigue. Training for strength is different than hypertrophy training where the goal is to push to the point of total exhaustion and burning fatigue. Strength training requires lifting heavier loads (85-100% 1RM), which can only be performed for 3-6 repetitions. Strength training also requires longer rest periods of at least 3-minutes. You should get to the point of fatigue but it should not be burning fatigue but the muscles just cannot produce the force required to lift the load. There is a distinction between these two methods and sometimes it is blurred, but at the end of each set your legs should not be burning as much as hypertrophy training with 80% 1RM for 8-12 repetitions.

The strength phase is also the beginning of very specific exercises that mimic the motions of cycling. The previous phases were preparing the muscles (adaptation) and making them as big as possible (hypertrophy) which does not necessarily require many cycling specific exercises. The goal was to get the muscles ready. Now the goal is to teach the nervous system and muscle to contract specific to the sport so that the strength gained can be carried over to the bike. Remember, strength is the result of big muscles and training the nervous system to recruit these muscles in the proper sequence and order specific to your sport. So in order to accomplish this all exercises during this phase should be specific to the sport of cycling. No more free squats, two-legged leg press, etc. All exercises should be performed one leg at a time, as in cycling, and with the body position (hip angle, feet width, etc.) as close to your bike position as possible. Examples of these exercises are lunges, step-ups, single-leg press, single leg extension (watch those knees), single leg curl, etc. A spin bike can also be used as a great strength exercise by increasing the resistance and turning as many repetitions as possible for 20-30 seconds. This is about as specific to cycling as you can get. You will have to again test for 1RM at the beginning of this phase but don’t forget that you must test for 1RM with the exercises you plan to do and if you are doing all single leg exercises these 1RM tests must also be performed with each leg (test for 4-6RM and extrapolate 1RM from the maximum weight chart). Be very careful not to injure yourself and do these only after you have completed the

other phases to make sure the muscles and support structures can handle it.

Since this method will only permit approximately 5-6 exercises within an hour only prime movers and multi-joint exercises should be trained during this phase. Remember to keep the exercises as well as your position as specific to cycling as possible.

Training loads for this phase must be within 85-100% 1RM. The number of sets will increase to 6-9 since you will only be able to perform 2-6 repetitions per set. Remember, this should be a periodized program, don't start off doing 95% 1RM for 8 sets. Start off at the lower load range for 6 sets and then gradually increasing either weight or number of sets over the next 6-8 weeks, increasing volume each week. Do not forget to schedule a recovery week of easy lifting every 3-weeks. All lifts should be performed in an explosive powerful manor in order to maximally stimulate the large fast twitch muscle fibers and train the nervous system for maximum power!

The maximal lifts during this phase overload the central nervous system (CNS) stimulating it to fire as many muscle fibers as possible and in the right combination or synchronization. This makes intent and effort of the lift very important. Even though the load will be heavy and the speed of the lift will be slow every lift should be done with the intent to move the weight as fast and as explosive as possible. Complete concentration and all out effort is required for each and every lift to get the maximum benefits.

Part 5: Power Phase

Following the strength phase it is time to focus on training maximal power, the speed at which your muscles can produce force. It is important that this phase be preceded by a strength phase. The athlete must be able to move the load explosively. No visible increments of power are possible without clear gains in strength. The greater the strength, the easier it becomes to overcome resistance or inertia and the more explosive a movement will be.

The first two weeks of the power phase should be devoted to easing into a power-training schedule. The strength gained in the previous phase is of no use to a cyclist who requires acceleration and power for launching and covering attacks and sprints. The bigger, stronger fast-twitch muscle fibers now need to be further modified through power training to contract faster, increasing the rate of

force production.

Proper intent and focus during every repetition will ensure you are maximally stimulating each muscle fiber. Physiologically, this type of training is stimulating primarily FT muscle fibers. They are trained to contract quickly, decreasing time to peak force and increasing the coordination and recruitment capabilities of the nervous system (NS). The NS also decreases its stimulation of opposing muscle groups that may otherwise counteract and slow down the movement. Adaptation results in better synchronization of muscle stimulation and increased recruitment of the muscle mass. Motor units and their firing patterns become larger activating a greater number of muscle fibers in a shorter period of time. Although cycling is predominately an endurance sport, winning and losing is determined by a rider's power!

The goal of this phase is to perform each repetition with as much force and speed as possible. Again, it is important to start every exercise, set, and repetition with the intent to lift the load as explosively and as powerful as possible. The exercises in this phase will remain as specific as possible and should remain unchanged from the previous phase.

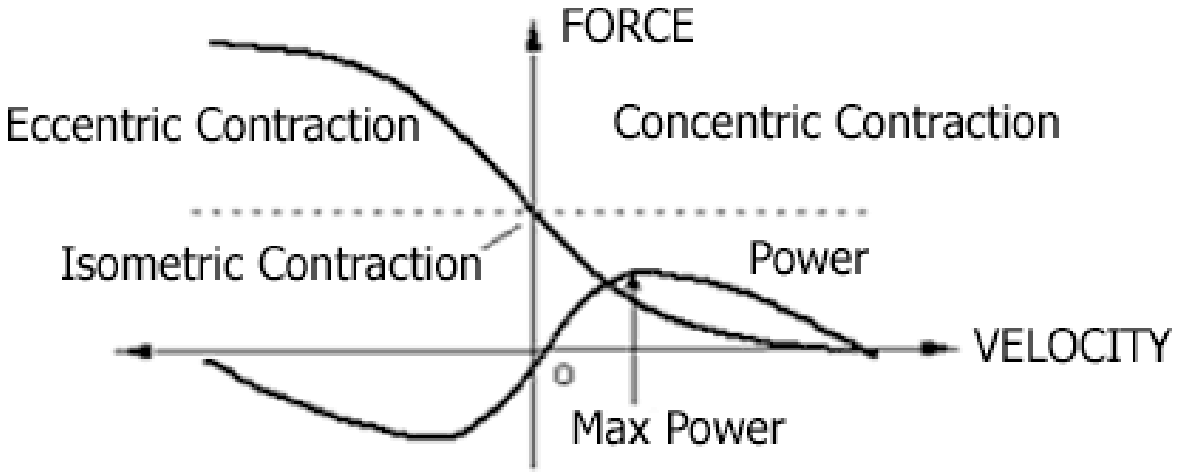
During the first few weeks of the power phase cyclists need to be very diligent, not wasting any energy on unnecessary exercises. Only the most specific exercises should be performed with the highest intensity possible (when I say intensity I mean concentration on each lift, being explosive and powerful, as if you were trying to jump through the roof with the weight on your back)! Following the initial two-weeks of the power phase some additional exercises may be performed but it is best to stay as specific as possible. Continue endurance and isometric work for the abdominal and back muscles.

The most specific method of power training for cyclists is training specific movements mimicking the pedal stroke as close as possible. This means single leg exercises through an identical range of motion at the knee and hip joint as when mounted on your bike. Also keeping foot stance equal to axle width, and foot angle (heel pointed in or out) the same as when clipped in. Don't be afraid to get specific and actually measure these things with tape and a goniometer for the joint angles. Only once you do this and then practice these ranges of motion and stances while lifting weights will it be as specific as possible. Soon you will become familiar with these specifics and doing the exercises outside of these angles will seem foreign. When possible focus on pushing through the ball of the foot except when heavy loads place too much pressure and soreness develops. Placing a 45-pound plate on the floor and just using the front of the foot to do

your lunges etc. is also a good way to get specific. Again, be careful when if you are attempting this because it places a lot of pressure on the meta-tarsal and seasmoid bones in the foot and has the potential for causing stress fractures. So only do this with exercises with a low relative load.

The load during weight lifting for power should be between 30-50%. Figure one shows the relationship between force and velocity. The graph depicts concentric and eccentric muscle actions. Muscular power is determined by the product of these changes (P = FV) and reaches a maximum at approximately one-third of the maximal velocity and one-half of the maximal force (Zatsiorsky, 1995). In other words, maximal power is exhibited when the external resistance requires 50% of the maximal force or 1RM. This is the load you want to work at because it is what you are trying to maximize. It may seem like the load is very light but remember that the goal during this phase is to produce as much power and explosion in the lift as possible to enhance nervous system coordination and firing. If you were training at a higher weight the explosion would not be as fast and the NS would not get the maximal benefit leading to sub-maximal power development. Keeping the loads light, at 30-50% 1RM, permits a lifting speed and velocity that maximizes the power produced giving the muscles and nervous system the best possible stimulus.

Figure 1. Depiction of the Force-Velocity curve.



Since the key to power training is the speed and power of the movement a low number of repetitions is suggested as long as there is proper intent and focus. All repetitions should be performed dynamically, non-stop, and at the highest rate possible. Be as smooth as possible and never snap the joints as the limb extends!

Cyclists must train to use their strength at a very high rate of contraction, and not just at the top of the pedal stroke. Those cyclists with the greatest power not only apply a greater amount of force at the top of the pedal stroke but also continue to produce force and acceleration around the entire pedal stroke. They can do this because they have trained to be powerful allowing them to not only match the speed of the crank, but to accelerate it throughout the pedal stroke.

The whole point of power training is you guessed it, to increase power. But the reason increasing power is beneficial to cyclists is that the nervous system increases stimulation and becomes more efficient leading to better coordination and recruitment of muscle mass. Soon, less force is necessary to maintain a given movement or workload. The smaller the force required means less muscle has to be activated to execute a given task and more muscle fibers are spared, leaving more unfatigued fibers to be activated if needed or saved until the final sprint. It's all about efficiency. Simplified (there are other factors), the fewer the number of muscle fibers required for a given quantity of work, the greater the efficiency and endurance. Strength is nothing to cyclists unless trained to be fast and powerful.

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Part 6: Muscular Endurance

The last phase of a periodized strength program for cyclists, with the exception of a maintenance phase, is the muscular endurance phase. Throughout this training program the muscles have enlarged in the hypertrophy phase, hopefully, got stronger during the strength phase, increased power in the power phase, and now we are trying to maximize muscular endurance. A powerful set of legs does not do a cyclist much good if they can only last for one hard attack. We need to train the muscles to retain most of the power gained previously, and be able to produce this power again and again.

The main goal of endurance training is to enhance the muscles ability to deal with, or delay the onset of fatigue. This type of training can also improve anaerobic endurance as well with the use of relatively large loads for a high

number of repetitions, usually between 50-100. The ultimate benefit to endurance weight training is an increase in physiological efficiency of the muscles. Endurance training after a periodized program for strength allows the muscles to be able to sustain a greater force or power output for a longer period of time than if they were solely trained for endurance. Also, as a result of strength training and increases in muscle size, a smaller number of motor units are required to perform a given task. So not only do the muscles become more efficient, but they can also do more work.

Endurance weight training should not increase the size of the muscle diameter and may even lead to a slight decrease in fiber size as the muscle adapts to become more metabolically efficient, just as it does with long hours on the bike. But this type of training is critical for increasing the muscles ability to complete work effectively since fewer muscle fibers are needed to perform a given task. In order to fully adapt to the endurance phase, 8-10 weeks of this phase is required.

There can be two types of endurance training methods. One is for training shorter 5-10 minute anaerobic efforts and another for long, aerobic activities. The first is ideal for more intense efforts lasting 2-10 minutes such as attacks and long sprint finishes. The second method is used to train for longer efforts greater than 10 minutes where most of the energy is coming from aerobic metabolism. Both methods are beneficial to cyclists because both efforts occur during a race. The two methods have similar physiological effects except short endurance training will require a larger load and shorter duration.

Short endurance training is best performed using an interval type method of lifting in which the rest interval between each set is inadequate for full recovery from the previous set. This constant exposure to such high levels of fatigue trains the muscle to cope with the pain and exhaustion of such efforts. The load for short endurance training should be 40-50% 1RM. The load, number of exercises, rest interval, and speed of execution should remain the same while only increasing the number of repetitions, typically every other week.

The number of exercises should be 4-8 with 2-4 sets per session. Since the sessions are not as structurally demanding on the muscles, exercises sessions can be stretched to 1-1.5 hours. The rest interval should be no more than 1.5-2 minutes and the frequency should be 2-3 times per week. The number of repetitions is the only factor that should change during this type of endurance training and usually is increased every other week. Start with sets of 50-60 reps at a time (first 3-4 weeks) and progress to grouping exercises such as the lunges and leg press, performing 50-60 reps on each with little to no rest in between.

Then progress in the next couple of weeks to three exercises in a row and then to four in a row the last couple of weeks. Keep the rest interval between these sets low, 2-3 minutes. These are demanding workouts so be sure to come into them recovered and with and energy drink!

Since cycling is an aerobic sport, which requires the application of force against a given resistance for long periods of time, the training must imitate this by performing very a high number of repetitions and doing them nonstop. Only very short rest periods are afforded since dealing with severe fatigue is the goal. Instead of repetitions, sets are determined by minutes. Sounds like fun huh…

Since the duration is longer, the load is lowered to 30-40% 1RM and the number of exercises remains the same. Sets remain 2-4 and the rest interval should never surpass 30 seconds between sets. An example would be starting with 4 minutes of nonstop work on the leg press at 30% 1RM and then moving on to the hamstring curl for 4 minutes, and then lunges for 4 minutes, etc, etc, nonstop and then taking a short rest period of 2-3 minutes. Over the next couple of weeks increase the duration of the sets to 7, 10, and 15 minutes respectively while keeping the number of exercises the same. That's right, this is about 45 minutes of nonstop lifting by the end of the phase!

Cycling involves both types of endurance so I would suggest long endurance workouts twice a week and a short endurance workout once during the week. If you are only doing 2 sessions a week, just alternate between the two.

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Part 7: Maintenance

The conclusion of a 6 month periodized weight program is a blessing for most endurance athletes but before you say goodbye to your fellow muscle heads at the gym you may want to seriously think about a maintenance phase and continuing it, at least once a week, throughout the season. The strength gained from training remains as long as the neuromuscular system maintains those cellular adaptations. Once strength training ceases, these adaptations are lost. So during the racing season a cycling specific strength plan may avoid the drastic reduction in strength seen from complete cessation of strength workouts.

Cycling is an endurance sport and through the training year putting in lots of

miles will actually cause muscles to decrease in size. This is a natural adaptation to becoming more aerobically fit. Many cyclists who strength train have a very strong early season when strength benefits are at there peak, but as the season continues lack of strength training leads to decreased muscle strength and power. To maintain good strength and power performance, cyclists need to incorporate a maintenance phase during the competitive season either on the bike or in the gym, or both.

For cycling, endurance is the dominant component of training and should constitute some portion of the maintenance phase. For this reason many cyclists do not lift heavy weights during the season. This does not mean occasional strength training does not play a role. Cycling is also a power sport, and maintaining maximal strength is important because if strength declines, power and endurance decline with it regardless of how well trained in those areas.

On the bike strength workouts combined with gym training offer the best solution to maintaining the strength acquired from the gym, and endurance got from the bike. Power should take care of itself with other bike workouts typically performed throughout the year.

However, strength training should take a back seat to bike training during this phase. The volume of strength training will be very low, 1-2 times a week, so that the most effort can be placed for technical and bike training.

Alternating gym and bike workouts every other week can be an effective way of breaking up the stimulus. If there is no racing during the week two sessions may be useful. Strength training on the bike involves a high gear and low cadence while climbing a steady incline of 4-6 minutes. Cadence should be in the 50-60 range.

When working it the gym, limit your session to 2-3 exercises of the prime movers. So basically the same exercises as the strength and power phase. Exercises such as leg press, lunges, step-ups, etc. are good choices. Make the session efficient, spending no more than 30-45 minutes in the gym. Remember, this is a maintenance phase and the goal is to maintain our fitness not improve it, so doing as little as possible to maintain strength will afford more energy for adaptations to bike training.

Keep the sets low, 1-4 depending on what you are training. More sets for strength and less for endurance. Resting intervals can be longer than usual and you should recover completely during the break. For strength maintenance the

load should be higher than that seen in competition but no so high that you run the risk of over-training and injury. The load should be 5-10% lower than that during the normal strength phase, so about 75-85% 1RM. Decreasing the load prior to major competitions and emphasizing power will enhance recovery and explosiveness.

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Part 8: Strength & Cycling

Strength training and cycling gets a lot of press this time of year, most notably from on-line cycling chat rooms discussing the best things to do during the off-season to prepare for the coming year. Usually, some dogmatic insists that strength training has nothing to offer an aerobic sport such as cycling leading to a barrage of replies insisting strength training has beneficial and performance enhancing capability for cyclists. I too believe that strength training is beneficial in the development of a stronger cyclist, one that should receive more attention. There is no doubt that cycling is an aerobic sport and strength training does little to improve a cyclist's aerobic system and endurance, unless untrained to begin with. However, due to the continual and constant interplay of aerobic and anaerobic efforts that occur during a race, improving cycling performance is a different matter.

Anaerobic power is a crucial part of racing success and is often the determinate in winning and losing. You can have the strongest aerobic system in the race but if you don't have a strong anaerobic system to get you in the winning break and then attack it, you will be very unsuccessful. The higher rated cyclists within the USCF undoubtedly have significantly higher anaerobic power outputs than lower rated cyclists (Tanaka, 1993).

First of all, most of this discussion is a matter of definition. Is training for strength the same thing as performing 12-15 repetitions at 80% of your 1 repetition maximum (1RM)? Is an endurance weight training protocol considered strength training? Even though the loads lifted are typically lower than with strength training they are usually heavier than the forces applied through normal cycling? So when someone says that strength training does not benefit cyclists, what kind of strength or weight training protocol are they talking about? A well-periodized weight-training program, one that switches focus from adaptation, hypertrophy, strength, power and endurance will undoubtedly enhance cycling ability and

performance when performed at the proper time and combined with appropriate on-the-bike training. This increase would be greater than if strength-training solely on the bike.

Strength training will not improve aerobic performance when defined as an increase in VO2max . Although, athletes concurrently training for strength and endurance show similar increases in VO2max as athletes training just for endurance (Kraemer et al. 1995). Even though strength training does not improve endurance it probably does not hurt it either given the proper training. It has been proposed that the body cannot adapt maximally to both training stimuli (aerobic and strength training) if they are initiated simultaneously (Hunter et al.). Starting weight training early in the cycling season, soon after the end of the previous season, may allow athletes to handle the added stress of training for endurance later on without much trade-off in either adaptation. However, research conducted by Hunter et al. has shown that well-trained endurance athletes may not experience the attenuated gains by adding strength training to an advanced endurance-training regimen (Hunter et al.). Starting a strength program early is important because it ensures maximal adaptation and strength gains before the addition of endurance training, which may limit or slow further increases in strength.

Strength training promotes an increase in the size or cross sectional area of all fiber types, including the aerobic type I muscle fibers (Fitts and Widrick 1996). Researchers have discovered that type IIa fiber percentage increases and type IIb fiber percentage decreases following strength and endurance training (Fitts and Widrick 1996). This suggests that even strength training causes fiber type transformations from type IIb to IIa. Strength training may also further improve the contractile properties of the type I fiber, most notably the myosin light chains, a part of the myofiber that deals with the velocity of shortening (Vmax). Strength and power training may alter the properties of the muscle fibers mainly through the expression of faster myosin light chain isoforms. This adaptation also occurs with endurance exercise but may occur to a greater degree with the increased loads and forces demanded of the muscle during strength and power workouts in the gym. These changes increase power and peak tension development of the fibers trained (Fitts and Widrick 1996). Endurance exercise causes type II fibers to express a slower isoform of the myosin light chains causing reduced energy expenditure and increased efficiency, a beneficial adaptation for endurance. Strength training concurrently with endurance training may reduce the expression of this slower isoform and result in a more powerful aerobic fiber.

These adaptations from strength training cause each muscle fiber to produce

more force, reducing the relative force requirements from each fiber when presented with a sub-maximal load. This would also result in less fibers being activated to accomplish a given sub-max workload and delay the recruitment of less efficient type II muscle fibers (Hickson et al. 1988).

I am not saying that these are not aerobic events, but neglecting to strength train may increase the loss of power seen with endurance adaptations, decreasing aerobic and anaerobic power outputs. Although cycling is mostly aerobic and average race power outputs average a surprisingly low 150-200 watts, a level easily maintained by most cyclists, it is the brief attacks and sustained time trailing that usually determines the winners and matching these high power outputs and sustaining them long enough will give you a much better chance at success!

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15) RACING TECHNIQUES
So, You Want to be a Cat 3? by Bill Laudien

I've been riding the B group this season and it's enlightened me to the fact that many riders lack an understanding of some of the basics. This isn't a criticism of them as much as it's a comment on the effect of not having a club system that teaches new riders how to ride their bikes. There are countless books and articles on how to train, but all those intervals are pretty useless if you're riding the entire race on the wrong side of the field and attacking on the down hills.

I'll also point out that there are a fair number of upper category riders who, despite their incredible strength and abilities, could still use a primer on some of this stuff. We've all seen our fair share of cat 2's pull off to the wrong side in a paceline. Anyway, I offer up this information as things that I've learned over the years. I'm not a particularly accomplished rider, but I've found that this information has been helpful in getting me through races against better riders and has added to my enjoyment of racing. I hope it does the same for you.

1. ANTICIPATE THE WIND

The first thing you need to do is figure out where the wind is coming from. Then visualize the course you are riding and where the wind is in relation to that course. Anticipate the wind and position yourself accordingly. In many instances, your position relative to the wind is far more important than your position front to back of the peleton. Use the corners to switch side of the field. Sometimes you may want to ride in the wind temporarily, if it means that you'll have shelter for a longer stretch exiting a turn.

2. RIDE PROPER PACELINES

You SAY you know how to, but I bet you don't. Here are the basics.

a) **Pull off INTO the wind** - This one drives me crazy. If done properly, as the relief rider (the one who just got done his turn) fades to the back of the field, he shields the riders moving up in the line. If done improperly, the riders moving up in the line are first pulling into the wind before their turn and then resting as they slow down . . . a total waste. It's very very simple . . . pull off into the wind. (I think that the reason that most new riders do this wrong is that they expect to be screwed by the next riders coming through. But more often than not, if you take the first step to pulling off the proper way, all other riders will follow)

b) DO NOT ACCELERATE - The lead rider should pull off or signal the second rider to come through and then the lead rider should SLIGHTLY decelerate. The second rider should **maintain pace**. It's the lead rider ending his turn who dictates the pace of rotation. The second rider should not storm past by sprinting or acceleration quickly. Speed is not what tires you out. Accelerating tires you out. The wind breaking efficiency of a paceline is ruined if energy is wasted with riders constantly changing pace. You can increase your speed 25% with the same effort with a steady paceline with subtle changes in speed.

c) Draft while going backwards - On a more leisurely ride, or when an attack first goes away, people can take long turns at the front. But once the group as settled in and is at speed, the paceline should be a **steady rotation**. This allows for maximum speed at minimum effort. The way this should work is that the pulling rider's turn (rider-A) should last just until he clears the wheel of the previous rider (rider-B). Then when rider-A pulls off and starts floating backwards, rider-B is drafting off him. Some folks thing that they are doing the group a favor by taking LONG, HARD pulls. The only things being accomplished with long hard pulls are that you break the rhythm of the paceline and you completely flick the guy who pulled before you as he has no one to draft off of and he has to sprint to get onto the back of the line.

d) If you follow the rules for pacelines as listed above, your paceline should NOT be a guy pulling super hard and then sitting up and going right to the back. What SHOULD be is guys making short efforts a hair above their limits then steady efforts a hair below their limits, riders drafting as they move up the line and as they move back in the line.

3. CONSERVE ENERGY

This is the most important thing that you can do, especially in longer races. You should play a game with yourself within each race to see how little of an effort you can make. Think of your body as a bank account with $100 where you have to pay $1 for every minute of riding. But you have to pay $5 for every minute of accelerating (sprinting). Typically, the guy who wins is the guy who has the most amount of money at the end of the race. Here are ways to conserve energy:

a) Look ahead - See what the riders at the front of the field are doing. If they are sitting up, then there's no reason to charge into the back of the field only to have to slam on your brakes. This is especially true out of corners. Conversely, if you see riders starting to attack and you know the pace is going to increase,

you can slowly start to increase your pace instead of having to rapidly react when you notice the rider in front of you take off.

b) Do not brake unless necessary - The previous instruction should help with this. Braking means that either you went too hard and now have to slow down or that you'll have to accelerate after whatever you're braking for. In either event braking usually means wasted energy of some sort.

c) Do not accelerate into dead air - Trite but true. If you need to sprint or move up etiher find a wheel to follow, or leave a spot between you and the wheel in front of you then accelerate into that wheel. Pullling out of line and then sprinting into a wall of air is a complete waste of energy.

d) Ride the wave - especially on circuit courses and criterium courses you'll find patterns of behavior. You'll see places where the field will accelerate and places where they sit up. Often out of corners they'll accelerate . . . then sit up. Figure out those patterns and take advantage of them. If you know of a section where the pace is to increase, try to position yourself toward the front before that acceleration then let yourself float back during the acceleration. You'll maintain a steady pace while everyone else is spending their $5 :) . Conversely, if you know there is a slow section, use that time as a chance to move your position in the field. This theory also works well with hills as you can start a climb in the front, make less of an effort as you float back and arrive at the top still in the field.

4. STOP PULLING FOR NO REASON

One of the main tactical difference between the cat 1/2 races and all other events is that no self respecting cat 2 would take a hard pull unless he had a good reason to. Conversely, cat 4 races usually play out with everyone sitting around until someone attacks, then everyone killing themselves to catch the poor guy, then everyone sitting around again. Save your big efforts to either attack or to bridge to a break. Don't make a big effort just to give all the other riders a free ride. Plus, if you want to ever be in a successful breakaway, you cannot contribute to the chase at any cost mentality. If there are knuckleheads who want to ride like lemmings, let them. Then when you counter attack, they'll be too tired to chase you. When you are racing, you are at war. Remember that there are more enemies in the pack (and on your wheel if you are pulling) then there are up the road. If someone is going to beat you, at least give a chance to the guys up the road making the effort and not to the passive riders taking advantage of your hard work.

5. ATTACK WHEN IT'S HARD

ANYONE can attack when it's easy. Anyone can attack downhill. Anyone can attack in a tailwind. If you feel fantastic and the pace is easy don't even bother attacking, because there are 99 other guys in the field who feel as great as you. **The key to a successful attack is to break the will of the other riders.** It's not easy to break the will of a guy whose HR is 130 and whose been coasting for the last 5 minutes. If you are fit, among the fittest of the riders in your group, then you want to attack when it's hard. If you're suffering at a level 8 (out of 10) and you're one of the strongest guys, then you have to suspect that the other riders are suffering at a 9 or a 10. That's when they're ripe for the cracking. These opportunities usually occur in crosswinds, on hills, and at the end of races.

A corollary to this rule is that it's often a good idea to attack when the pace is slow in order to make the race harder. I often attack at the beginning of races or when the pace slows, but I never make a full commitment to those efforts. Jumping and then cruising at 80% usually isn't that draining, but can often stir up the field and induce counter attacks and a period of hard racing. Being off the front when that happens also allows you to slip back into the front of the field and in good position for a counter attack with a real effort.

6. TEAMMATES

While Cippolini's red train is quite the site to see and Lance's armada can blow a tour stage open, team racing at the lower categories need not be so involved. Here are ways that I think teammates can most effectively assist each other.

a) Moving through the field - Don't move up alone. If you are going to make an effort, at least bring a teammate with you.

b) Counter attack each other - If your teammate is away in a break and that break gets caught and you do not counter attack, it's an INSULT. You're teammate's effort to get away caused the chasing riders to tire while you sat on. That's exactly the situation that you're looking for as a rider and as a team. Don't let that effort go in vain. Counter. Even if you get caught, the stage is set for him to recounter or for another of your teammates to hit the pack again.

c) Leadouts - mostly leadouts are a waste of time at the lower levels. If you have two guys strong enough to both be at the front, you're usually both best served by sprinting and getting two results. There are exceptions to that.
• If you're both out of position, one rider should sacrifice himself to get the other into position before the sprint.
• If it's a tricky or dangerous finish, you want to attack in tandem very early and conduct your leadout before the technical sections. This will get both riders clear of the chaos and should ensure victory for the rider being led out.
• If you're unsure about where to start the sprint and you have two guys in front, then have one guy start the sprint very early with a strong attack. If he's right and gets clear, he wins. If not, then the riders chasing him act as a leadout to the second rider. This gives you two chances to win.

7. SPRINTING

I'll try to keep this as simple as possible. Sprint early. Most new riders wait too long and find themselves in the middle of a mess. You're usually better off going early and avoiding the chaos. Even if you get passed and finish 5th, it's better than being trapped and finishing 12th or worse yet, ending up on the pavement. Also you'll never really know how far you can sprint until you've gone too early a couple of times. Once you've seen how far you can go before you blow, then you can make the necessary adjustments based on wind, terrain, and circumstances.

Lastly, gear selection is somewhat important. You are attempting to accelerate as quickly as possible and then hold your speed as long as possible. Unless you are Mark Light or Karl Woitas, you can do neither in the 12. Sprint in a gear that you can accelerate and then shift if you need to. Also, once you are up to speed, do not be afraid to sit down and spin. You're much more aero seated and in a long sprint you can oftern get a few more RPMs out of your gear while in the seated position.

CONCLUSION

There are some other things that I haven't touched on here and that I'll try to explain later. But hopefully this should provide you with enough of the basics to see some immediate improvement on your weekly training race.

16) CLIMBING FOR CYCLING

CLIMBING ELEMENT 1:
Focus On Developing Your Muscular Endurance (ME)

If there is one key ingredient to becoming a better climber it is your ability to maintain increasingly higher levels of power output for extended durations otherwise known as Muscular Endurance (ME).

The ability to maintain increasingly higher levels of power without passing too far into your lactate threshold takes practice and discipline and in my eyes is one of the most underused forms of training. Why? Because it's really *#$@# hard that's why.

Holding a steady pace of 400+ watts for 3 minutes or more can take a tremendous amount of self-discipline, physical ability, and most importantly muscular endurance and thus must be practiced repeatedly so that when we're climbing for performance we have a reserve of muscular endurance left to draw from.

Let's face it, climbing is something that any good cyclist worth his/her salt strives to improve. It's the pinnacle form of satisfaction to climb with greater confidence and fluidity. To do this you must be able to maintain a rhythmic cadence and have our muscles under constant duress without fatiguing. You must be able to control your effort and judge your abilities as you're climbing if you are to reach the top without losing steam.

You must master various levels of your Muscular Endurance! How can you develop this key ingredient for improved climbing? Let's briefly discuss two tactics both on-the-bike and off-the-bike that you can use to increase your muscular endurance.

1. ME Intervals on your indoor trainer or out on your bike with various levels of gradients on hills. The basic premise with ME Intervals is simple - repeatability! Climbing a hill or mountain once doesn't. In fact, to truly increase muscular endurance we must master various levels of hills for various lengths of time to improve performance. How many times do you think Lance practiced going up Alp Du'ez? I like intervals that range from 3 minutes at higher watts to as long as 15 minutes at moderate high watts. The key is to keep your cadence consistent (I prefer 80+ RPM - more on cadence in

element 5) and stay at or just below your lactate threshold (LT) / anaerobic threshold (AT) so that you can last the whole set.

Out on the bike my favorite ME repeats are done on a road with two different style climbs that I repeat 4-10 times each depending on my goals and training objectives. The first is short and steep and when performed well I can complete in about 90 seconds with a lower, more powerful gearing and spin at 65-80 RPM.

The other is longer and more gradual, but still really tough and takes me about 15 minutes to complete. I use lighter gearing and spin high (85+ RPM). I use these two style hills to generate two different forms of ME. One is shorter and more explosive and the other is longer and more controlling. My objective is still the same however - control my cadence and maintain my HR just at or below my AT.

Over time I will choose 1-3 intervals to focus on increasing cadence or lower the gearing to produce higher watts and help to condition my body to maintain higher levels of power output while maintaining my heart rate and exertion level, thus improving my ME.

During these specific intervals I will also try to keep my HR 4-10 beats **above** my lactate threshold to condition my body and hopefully create a new level of sustained muscular endurance at my AT.

2. One of my other tactics to increase your ME is to utilize Power Yoga. Out of all the various forms of training I use and teach to others, nothing is more challenging than Power Yoga. Don't let the pony tails and hairy chests mislead you, Power Yoga will challenge you more than your average Alp, I promise you that. In relation to improving ME, Power Yoga is perfect because it forces you to hold and maintain poses and positions that directly effect your climbing muscles, such as your low back, your core, your hamstrings, hip flexors, and quads for extended periods just as in climbing repeats.

Power Yoga will teach control and add a new layer of strength that classic weight training and even cycling can't match. I try to use it in my training at least once every other week and sometimes I use it just before or just after an ME Interval for added difficulty and a new challenge.

Combine these two muscular endurance challenging tactics together in as many ways as you can imagine and you will greatly improve not only your ME,

but your climbing skills and lactate threshold at the same time. Talk about a "zen moment"!

CLIMBING ELEMENT 2:
Improve Your Low Back Strength and Durability

The centerpiece for improved climbing begins and ends with the strength and durability of your low back. Climb any extended, steep hill or mountain and you'll know exactly what I mean. Focus on making it stronger and you will have the primary tool you need to improve your climbing -- Guaranteed!

The low back represents the centerpiece from which the large muscles of the hamstrings and glutes can create leverage from which to climb with. If the low back is weak, fatigued, rigid and inflexible, the entire body during a climb will begin to crack and break down and ultimately lead to a complete loss of power and control.

We all know instinctively that we need to condition and work on the physical conditioning of our low backs, but because it seems so simple and innocuous, many of us never do the little things to keep it strong and conditioned. And all the while we're working on other elements such as ME, Power, and Endurance, it is our low back durability that will determine long term improvement.

The funny thing is that the power, strength, and endurance of the low back is the net result of just about every other muscle and joint that revolves around it. From the alignment of the hips and spine to the strength and flexibility of the hamstrings and abdomen, the low back is truly part of a larger network that must all be well balanced and maintained if it is to improve. Let's discuss three issues one needs to address to develop a strong, fluid, stable low back for improved climbing skills.

1. You MUST keep the hamstrings flexible! One of the key issues for chronic low back fatigue isn't just a weak back, but tight hamstrings. Especially for the cyclist whose repeated actions never really places the knee through a full range of motion and as a result has a tendency for tight hamstrings. Unfortunately most people do a tug here and a pull there on these vital climbing muscles and leave it at that.

The hamstrings require deep, well-organized stretches in order to truly open

them and keep them strong and recovered. From using yoga positions to more classic stretching with a stability ball, one must have a well-put together hamstring stretching agenda to stay on top of the potential problem. Not only will it improve the strength of your low back, but it will help improve your climbing position and posture both in and out of the saddle. If you can't stay aero or in the drops very long your hamstrings are most definitely one of your limiters.

2. You must BALANCE strength and flexibility in all of the core muscles. Cyclists unfortunately have a "quad only" vantage point in relation to training. We overdue the training and focus on the quads and not enough on the hamstrings, hip flexors, glutes and low back. This imbalance can create a forward pitch in the posture of cyclists that will result in a loss of potential power on the bike while climbing.

A good low back conditioning routine is perfectly balanced with strength and flexibility exercise for both the front side as well as the backside off the body. You can't just do squats and hope to build low back and climbing strength. You must do the little things that make the low back complete.

3. You must CONSISTENTLY train and condition your low back OFF THE BIKE, not just while you're on it. Yes, you can train the muscles of the back to improve while on the bike by doing more climbing, but to maintain improvement and add the flexibility element, you must do more and you must do it consistently.

Many of us start a low back conditioning program, but either never do it enough or get caught in the same routine and never add new elements or keep it too simple and not place enough priority on low back training.

I guarantee that if you put just 15 minutes of direct strength conditioning and 15 minutes of flexibility for the low back and hamstrings into your regular routine, YEAR ROUND you will boost your climbing skills faster than any other training program there is.

If you know that your low back strength and hamstring flexibility is a limiter for you then you may be interested in my: *"Fix the Back, Stay in the Drops"* Workout Routine included in my Cyclo-ZEN Mental Toughness and Recovery Program. It's a simple, balanced and effective way to add new layers of low back strength and durability to your training program with no equipment and you can do it anywhere.

CLIMBING ELEMENT 3:
Methodically and Diligently Boost Your Climbing Cadence

There has been a complete paradigm shift in the thinking of climbing cadence due in large part to our friend Lance Armstrong. His unique ability to spin at higher RPMs of 100+ has allowed him to climb harder and faster than his body type should allow him to do.

It has been his dedication to developing the mental and physical skills needed to maintain high RPMs up mountains that has allowed him to stay stronger and fresher than the competition and is probably the single largest contributing factor to his 6 years of climbing domination.

If you want to be a better climber, you may want to learn some lessons from the higher climbing cadence techniques. Now, that's not to say that you need to spin at 90+ RPMs for climbing, but maybe find new techniques to increase your "natural" climbing cadence 3-10 RPM for improved efficiency and increased climbing speed and power for longer periods of time.

Let's discuss some ways of helping you increase your natural climbing RPM and improve your overall cadence and efficiency on the bike.

1. Add at least one to two higher cadence ME training sessions to you bike training schedule and focus on increasing your cadence by 1-3% to start. Don't make the mistake of jumping from 75 RPM to 100 RPM overnight. It takes time and discipline for your body to make this physiological adjustment and could result in overuse injuries and over-training if you're not careful.

Start with 3-5 minute intervals spaced out over a 75-90 minute ride and pick various gradient hills to practice the increased cadence. Now to accomplish this at first you're going to have to gear down to spin higher. Once you've focused and mastered the higher spin rate, work on gradually gearing back up at the same RPM for improved power and climbing speeds.

2. Utilize Power Yoga to help you to acquire contra-lateral reflexes to get more power out of your muscles and increase body control. What?? Simply put, teach your body to contract opposing muscles to create more power and stability. For instance learning to contract your right quad and your

left hamstring forcefully at the same time just as you need to on the bike, especially for climbing.

This is also known as agility or the ability to contract several opposing muscles simultaneously. Power Yoga is ideal for this discipline due to its necessity for your body to balance and oppose itself for stability. Several Power Yoga poses cannot be achieved without your body and your mind contracting opposite muscles. This adds new levels of muscular control that is vital to you increasing your cadence and efficiency on the bike.

3. Use high cadence hill repeats that average 5-10 RPMs over your ideal cadence that you're trying to achieve. For instance, if your natural climbing cadence is 80 RPM and you hope to increase it to 85 RPM for improved climbing speeds, then practice intervals of 3-10 minutes at 90-95 RPM at a slightly lower gearing. Repeat this 2-4 times in a workout every week to begin to overshoot your cadence marker and lay the foundation for improved climbing speed. This technique works really, really well and has helped me move my natural climbing cadence of 75 all the way up to 94 over the course of the past two years. As a result my climbing skills have improved 10 fold and I have turned my climbing into my greatest strength on the bike.

CLIMBING ELEMENT 4: Boost the Power and Lighten the Load

Climbing is all about gravity and mass. It's simple really. The more mass an object has the more work needs to be done to move that mass against the force of gravity. In our case as aspiring climbers we need to have less mass and more potential for work or power in this case.

We call it our strength to weight ratio (or power to weight ratio) and it is the direct determinant of how fast we can go up a hill or mountain. If we can lighten our mass and increase our power we will move faster and with less effort needed to produce the same outcome. This is good, because of any limitation due to genetics we can deal with this issue head on through several tactics and techniques both on and off the bike.

Let's discuss two elements needed to increase our power and lower our mass so that we may climb faster and with greater ease.

1. The first is obvious and probably the element we should focus on the

most - developing more power at the same weight or mass. If you're not big on losing weight or removing those sticky buns before a ride, then this is the tactic for you.

There are two kinds of power in relation to climbing, the first is one that we've already talked about in element one, ***muscular endurance*** or the ability to sustain consistent power for extended periods of time. We can use ME Intervals, hill repeats, and power yoga to help us develop this type of power as it is the most used power mode in climbing.

The second is ***explosive power*** or short bursts of high power for much shorter lengths of time. This type of power is actually very important as well as it addresses the issues of changes in incline on a hill or maybe an acceleration from a competitor. This explosive power is best worked on with shorter, steeper climbs that only last 60 seconds or less. You can also work on this explosive power with short, intense cross-training exercises such as Hindu squats, Hindu push-ups, plyometrics and other power provoking exercises performed off the bike.

You can also use longer duration ME intervals layered with short intense power bursts every few minutes to practice changes in power demand.

2. The second component of an improved power to weight ratio is to lose mass. Real brain trust on this one, but one that cannot be undersold. If you really want to improve your climbing the most profound way to do it is to lighten the load and keep the same or slightly higher power potential.

Right away most cyclists revert to calorie deprivation or excessive training to achieve this change in mass. Let me caution you, although it is tempting to drop calories and train more it can come back to hurt you in the end with fatigue and a lack of recoverability. Not only that, but restrictive diets and over-training do nothing more than lower your metabolism and catabolise muscle tissue which will ultimately reduce your power potential.

That being said we can lower calories slightly over time (no more than 250/day) and gradually increase our mileage to produce a leaner, lighter frame. The best advice is to eat high energy, low weight foods like lean meats and fish, beans, lentils, and high fiber cereals and grains. Avoid all the crap and artificial foods that rob us of our natural energy.

The best scenario is to focus on both and let them work together.

Focus on improving your sustainable power and explosive power while watching what you eat and controlling your portions to lighten your mass. Put them both together and you'll be topping 3000 foot climbs before with confidence and ease before you know it!

CLIMBING ELEMENT 5: Bolster Your Mental Toughness and Focus Your Breath

Watch any good climber and you will see someone with a stone face and a mastery of breathe and body control. No energy is wasted and no breath is underutilized. To them it's not a physical obstacle, but a mental one. A test of mettle and mental toughness to endure extreme discomfort.

Lance is a great example of someone who inherently is not built to be a great climber, but has turned it into one of his most powerful strengths as a result of harnessing his mental toughness and focusing on his breath and body control. He has practiced and mastered the ability to deal with the pain. Granted, overcoming cancer was probably his single greatest teacher. We can learn a lot from this lesson of mental toughness and focus.

Most of us aren't built to be great climbers, but many of us can be if we can toughen our resolve and learn how to get the maximum out of our bodies while conserving our energy. We can improve our climbing and our tolerance of pain by practicing, focusing and developing our mental toughness both on and off the bike.

How can you toughen up and teach yourself to deal with the discomfort of climbing a challenging hill or mountain on your bike? Let's discuss three things you can do to practice developing your mental toughness:

1. Incorporate power yoga into your training routine frequently. Yes, I already mentioned this in secret 1 as we discussed muscular endurance. I think you will recall that I described muscular endurance as a true test of mental toughness. So, by using power yoga as a tool to teach your body to overcome discomfort and pain while maintaining breath and focus is pivotal to increasing your climbing skills and performance.

Hold a yoga chair position for 90 seconds or maybe repeat an asana (several yoga poses in a row) 4 to 5 times without break and tell me if it doesn't test your mental toughness and breath just like a hard steep climb.

2. Pick one or two workouts a week on or off the bike that you really don't enjoy. Yes, that's right, don't enjoy! Maybe it's a route with endless difficult climbs or strong headwinds or maybe continuous gradual up hills with no reprieves. Pick sections and really hammer it out and don't stop. Teach your body to look past the physical and absolve the pain. Teach it to overcome fear and trepidation and push yourself past what you think is possible even if it's only for a few seconds.

Use this technique sparingly to avoid over-training and most importantly go into these workouts with the sole intent to push mentally. You must recognize and embrace the mental pain if you are to improve your mental toughness and resolve. Focus on your breath and teach yourself to channel your energy efficiently to the pedals.

3. Incorporate a "mantra" or "positive affirmation" along with the tactics above to get through the pain and the fear. You must align yourself with something strong and magical if you want to break barriers of physical and mental performance. Pick a word, saying, or image or moment in time that will help propel you past your mountain of pain. It may sound corny, but it may be the single most important part of developing your mental toughness and ultimately your climbing skills.

Developing A "Cottage of Wattage" Watching the BIG TT specialists yesterday in the prologue I loved catching a glimpse of the riders from behind. And yes, I was looking at their butts! Talk about power from behind! Their hamstrings, calves, and glutes look more like forged steel than human flesh! That's where the power is coming from my friends. That's what we need to focus on!

In my recent interview with former PRO cyclist, Chris McGovern, (part of Cyclo-SPEED due out in late July) he termed this area as the "cottage of wattage" or the definitive place where the power comes from. Below are some keys that Chris shared with me to help you get more power for your "*cottage of wattage"*:

"*Cottage of Wattage*" Training Tips:

1. Strength Training on the bike with high gear & low cadence interval sessions like my overspin/overpower intervals once a week. Get it at http://cyclo-zen.com/combo/SPEED.html

2. More emphasis on core and low back conditioning at least once per week to create a more stable platform for your cottage of wattage.

3. More hamstring strength training and flexibility work! At least every other day for the flexibility work (Cyclo-CORE stretching routine after hard rides & Power Yoga once a week)

4. More POWER from cross-training! Use the Cyclo-ZEN 8 minute Power Routine (as many rounds as you can stomach) once per week to skyrocket your cottage of wattage! Serious stuff, so stay at your own level and look to advance week to week.

All these tips and suggestions will help you focus direct on your cottage of wattage and help your power, climbing speed, and performance on the bike improve dramatically.

SUFFERING

I woke up this morning at 6 am to watch the exciting stage 9 of the Tour de France. Why? Because today is really the first day of mountain suffering!

Why do we love to watch other riders suffer anyway? Are we sadistic? Do we enjoy other people's pain? Are we sick?

Well . . . YES. In any other venue this would be disturbing, but being a cyclist we have the right to enjoy watching the pain because if you've been riding long enough you know what it means to suffer!

You can't improve as a cyclist UNLESS you can suffer! The good news is . . . you can TRAIN to suffer. I have some suggestions for you to learn how to suffer better.

SUFFERING Tip 1: Don't let the pain take hold of your mind. You can burn, you can hurt. You can sweat and bleed, but NEVER, EVER allow your mind to get negative on the pain. It's so easy to lose your confidence once you let the pain set in mentally. You must find other ways to channel the pain out. (suggestions below)

SUFFERING Tip 2: Set a suffering tempo and rhythm. 1..2..3..4..1... The fastest way to let your pain set in is to lose a sustainable tempo and rhythm

YOU can hold. Each of us have natural rhythms that we can keep while climbing, time trialing, and overall suffering. It's imperative that we maintain this tempo as often as we can. If we lose tempo to catch a break or to make a break, we must develop the discipline to regain our tempo & FAST.

SUFFERING Tip 3: Stay calm and collected when tempo breaks. It's inevitable that during a hard ride, especially one with others, that you will lose your tempo. That's OK. Don't panic! Tell yourself to calm down, breathe, relax, and set back into your tempo and hope to catch up on the other side. Panic and you'll redline your heart and energy systems. Yoga helps to practice.

SUFFERING Tip 4: Don't clinch and pinch! When you suffer it's so easy to clinch down into your shoulder blades and close off your lungs and diaphragm. It's so easy to pinch into the handlebars and over-grip. Both of these natural human tendencies under stress decrease oxygen flow, increase energy demands and sets a cue to your brain that you've accepted the pain.

SUFFERING Tip 5: You must scan your body every 10 to 20 sec while you suffer to remain a relaxed and fluid posture. Repeat the following body scan, or something like it while you suffer and you will keep the pain at bay.
strong legs . . . open lungs . . relaxed palms . . loose face . . fluid pace . .
strong legs . . . open lungs . . relaxed palms . . loose face . . fluid pace . .
strong legs . . . open lungs . . relaxed palms . . loose face . . fluid pace . .

SUFFERING Tip 6: Your suffering is relative! Never forget that if you're suffering, more than likely everyone around you is suffering as well. Also, try to think of other things in your life that have challenged you and your mettle and how you overcame that suffering. Sometimes the pain of climbing a big hill or a grueling TT is minor in comparison to a life altering stress situation. Look what suffering through cancer has done for Lance. Makes climbing an Alp seem pretty insignificant doesn't it? Suffering isn't a bad thing. It lets us know that we're working for progress. It's a marker of our courage and need to improve.

Recovery Rules for Mortals:

1. ALWAYS take at least 1 DAY OFF (passive recovery) each and every week. NO activity.

2. Take 1 to 2 ACTIVE recovery days each week during hard training

weeks. This means that you can be out on your bike with a light spin, little terrain, and a low HR. This helps to speed the removal of waste products of hard training and keeps your legs in tempo. Active recovery can also be spent off the bike with hiking, walking, light flexibility work, and my recovery yoga from Cyclo-ZEN (preferred and most beneficial active recovery).

3. ALWAYS do a post-ride stretching routine (5 to 15 minutes) after ALL hard rides to speed recovery and further remove waste products that cause fatigue and soreness. I recommend Cyclo-CORE 15 minute flexibility routine OR Cyclo-ZEN low back flexibility routine. Quick and effective.

4. Do cross training once a week to give your body a new stimulus and allow for recovery of your 'cycling system'. This is not really a recovery session, but a change of scenery that can sometimes be just as beneficial. I do Cyclo-CORE body-weight conditioning OR functional training OR the Cyclo-ZEN 8 minute power routine OR 30 minute Power Yoga. Careful NOT to place their workouts to close to a hard ride and these are best done the day before an OFF training day.

That's a start for recovery. It can be active or passive. Bottom line -- You have to recover enough each week to allow your body to progress. **There is no faster way to kill your fitness than to NOT have recovery days.**

The 3 Keys to Low Back Longevity:

KEY #1: Diligently Maintain & Challenge Your Flexibility!

More Than Just the Low Back Strength: The MOST important part of low back durability is keeping the hamstrings, glutes, IT band, and hip flexors loose, open, and flexible! I can't stress this enough.

Most people think it's only about core strength and or low back strength. This is NOT true. In fact people that do only crunches and the occasional low back strength exercises without adding in the proper flexibility work could be makin their situation worse!

How Much Flexibility Work and When:

I recommend doing a bare minimum of three post-ride flexibility and low back recovery sessions every week for tight individuals. You know who you are! If

you hope to make your back stronger and more powerful, this is your first place to start.

I recommend to my customers, either the 20 minute thorough full body flexibility routine from Cyclo-CORE once a week and then the 'fix the back, stray in the drops' STRETCHING routine exclusively for the low back complex and surrounding muscles twice a week. This has worked miracles for hundreds of my customers with low back problems. Learn more. . .

Using Yoga as a Tool for More Progress:

If you are in really rough shape and want to see even more improvement I recommend something like my 30 minute POWER Yoga Routine or the 30 Minute RECOVERY Yoga routine from Cyclo-ZEN to really get into your low back and hamstrings deeply. I would recommend adding one of these workouts once every other week along with your riding schedule. Great to do in the morning.

Intermittent Flexibility Sessions:

One of my personal secrets to keeping the hamstring-calf- low back complex open and functional is stretching all day long as often as I can. I get up from the desk every 60 to 90 minutes and stretch my hamstrings and calves on the stairs. I stretch in the morning in the shower by doing forward bends, standing twists and other stretches from my Cyclo-ZEN workouts. Learn more . . .

The more places you find to keep the muscles open, the more progress you will see.

Consistency is Everything!

You must do this whole flexibility process on an ongoing basis! It's a use-it-or-lose-it scenario with flexibility. Few of us are naturally flexible and must constantly work at it for it to maintain and improve.

If you truly want to improve and solve your low back issues, then you must put in your time and do the work. It won't just come to you. It's really not that hard if you stay on top of it.

CORE Conditioning IS NOT About a 'Six Pack':

Core Conditioning is often misunderstood by the masses. To most people it means simple crunches, abdominal training, and developing a washboard stomach. Although this may be a sideline benefit to doing core training along with proper diet it IS NOT what core training is about. It's much deeper than that!

When it comes to your low back longevity, core training plays a much more 'underlying role'. I mean that as literally as it sounds. Instead of thinking about your core as a group of muscles you can see, think of the core as an underlying network of support beams holding and positioning your body in correct alignment, like a building.

Are You Wearing Your Girdle?

The CORE is actually several muscles working together to create a stable platform for your spine. However, the most important muscle in that network is the one we will focus on today. It's called the Transverse Abdominal Plane (TAP).

I like to think about the TAP as our body's natural girdle. Kind of like what women used to wear in colonial times to pinch in their waist and lift up their chest.

The Transverse Abdominal Plane (TAP) and Cycling:

For cyclists, it's kind of the same idea. When well conditioned and engaged, the TAP does several key things to help us ride stronger:

1. The TAP creates internal pressure from 360 degrees around the body that stabilizes the spine, hips, and chest cavity to optimize our posture that aligns our kinetic chain (our natural flow of energy).

2. The TAP, when engaged offers a rigid and stable platform for our legs to drive force down into the pedals. Without an engaged TAP, the force production of the legs is diminished.

3. As the TAP engages the spine, it elevates the chest cavity, opening the breathing pathways for improved oxygen uptake. This is probably one of the most important aspects of a strong TAP. More air is more oxygen. More oxygen is more energy production.

Working the TAP:

Anyone that has used my CORE & ZEN Programs is definitely aware of mow much attention I give to the Transverse Plane of the CORE. I consider it the focal point of all my workouts and I'd say that just about every exercise I offer engages the TAP.

A strong TAP means a well positioned hip girdle and spinal column. This is how TAP training plays such an integral role in your low back longevity. Most people don't just have a weak low back, they have a greatly underdeveloped TAP. This is why they are out of alignment and have chronic low back pain and fatigue and are more susceptible to low back conditions.

Engaging the TAP Through Functional Movements:

There are several ways to learn to engage the TAP. Everything from static holds to functional movements with light weights. I also incorporate a stability ball which is great for engaging the TAP if used correctly. There are hundreds of exercises that one can use. I choose the one's that have the greatest impact in the TAP first.

The TAP and Yoga:

One of the most powerful ways to engage and enliven the TAP is with Yoga and several of the poses that I chose specifically from my experience with yoga.

The TAP and Gravity:

My greatest secret for engaging the TAP is to utilize gravity and my body-weight. No other form of training can help you capitalize on your core strength better than body weight conditioning. No weights. No fancy equipment. No Gyms. Just old fashioned, natural movements that engage the core by making you balance, adjust, and align your body. Not easy stuff and as anyone that has done my workouts can attest...they are very challenging exercises.

Opposite Style Climbing Intervals:

OBJECTIVES: Teach your body 7 mind discipline, even when it's not logical to maintain tempo or climbing style. Promote more mental toughness and skills that can be used when riding with others. Develop strength in both in and out of the saddle riding to add to your riding arsenal. Mix things up and add fun challenges to otherwise familiar and boring rides you've done a hundred times before!

1. Pick a 30 to 60 mile course that you frequent on your bike and know very well. Make sure it has as many hills, and as many types of hills as you can get in that one ride, even if they are short and steep or long and barely headed up.

2. Ride tempo (moderate hard pace) to the base of the first hill (only if you have completed a light 5 mile warm-up or so) and pick the opposite climbing style that you would use for tha hill. So if it's long and gradual, get OUT of the saddle and quick step for the ENTIRE length of the hill! YES, this is NOT how you would typically approach this hill type and not recommended each time you do it. HOWEVER, by changing the style you force yourself to change in dynamics and capacity. PLUS, if the hill takes longer than one to two minutes to climb and you're out of the saddle . . . OUCH! That's going to really challenge your core and hip flexors AND your mental toughness!

NOTE: I understand that gradients change within a hill climb and usually you would change position throughout. In this case pick the OPPOSITE of the GENERAL climbing position and STICK it out all the way. This is the DISCIPLINE portion that is so critical! Think of these intervals as illogical ways of accomplishing logical results.

3. Once to the top, continue on to the next interval climb. between each climb you're riding a strong TEMPO. This is NOT light spinning. Consider the entire ride other than warm-up and cool-down a FITNESS ride and one that will challenge you the entire time.

4. At the next climb, follow protocol and do the OPPOSITE climbing position to the top that you normally would and HOLD it no matter what. So, even if you're in the saddle and it pitches WAY UP, you stay in the saddle and grind it out. THIS is very important and you must stay mentally strong here!

5. Get to the top and continue strong tempo onward to the next interval site. This may continue for the entire ride for as long as 60 miles if you like. Keep

in mind however, that this is a tough sort of ride and one that requires good rest before and after to allow for improvements in fitness.

NOTE: If you do not have any hills near or around you, meaning you're a flat lander, you may do this exact same interval pattern into stiff headwinds and/or get out of the saddle for 2 to 5 minutes and jog to simulate the out of the saddle climbing and mix that with a tough gearing (15 and below) seated, low cadence grind fest to simulate seated climbing. Mix these with tempo riding in-between. VERY HARD and great for everyone as a way to mix it up.

NOTE: If you have VERY long climbs that usually take over 10 minutes, you may alternate climbing position throughout the climb, but try to stay out of the saddle for at least 3 minutes each time. This is tough at first, but boy does it work! If you can stay out of the saddle longer, give it a go.

POST-RIDE RECOVERY: Because this workout is so different and unconventional from every other sort of ride I STRONGLY suggest you follow my advice and perform a post-ride recovery session to prevent fatigue and soreness from prolonged out of the saddle climbing. Use the Cyclo-CORE 15 minute full body flexibility routine when you get home OR you can use the Cyclo-ZEN 'Fix the Back, Stay in the Drops' STRETCHING routine as the low back got one heck of a workout on the intervals.

FINAL NOTE: I would only use this sort of interval workout once every month on a day that you are dreading the same old boring ride and want to shake it up a bit. This is really fun and challenging and will make you a more disciplined and well rounded cyclist.

Ride Hard. Do the Opposite of Normal. Have Fun & Mix it Up!

Mind/Body/Soul Preparation Tips:

Technique 1: Psyche Yourself DOWN!

Most people think that the solution to doing well in an event, race or otherwise is to psyche themselves UP! Do you know what that really means? STRESS!!!!

The added stress of trying to excite your body to perform, especially when you do it for days beforehand will rob you of precious energy you need for the ride or race.

Psyching yourself up is something you do for a fight or maybe a 100 meter sprint, but not a road race or cycling event. You must conserve your energy, relax your mind, replenish your mental state.

A good cyclist is a relaxed cyclist with a clear and open mind. Don't clutter it with “I should have done more intervals” or “I wish I had just one more rest day” or “I know that climb up Appalachian Gap is going to pop me”.

These negative or anxiety driven mental states will become self-fulfilling prophecies. Squash them now!

POSITIVE AFFIRMATIONS:

You must convince yourself of your strength and energize your mind and body with positive affirmations. This is a sort of active, ongoing form of meditation or mantra.

“I will remain calm, focused, and relaxed while I ride.”
“I will float and soar over each hill / mountain I encounter.”
“I will ride my own race and search for enjoyment while I ride.”
“My legs are strong, powerful, and swift.”

Pick whatever you want to reaffirm your place and state of positive influence before, during, and even after you ride. It works!!!! I will be repeating things like this today as I drive my family towards VT. I will breathe and open my mind, not cloud it with anxiety and fear, and trust me, I should be afraid of this one!

ACTIVE AFFIRMATIONS:

I will also be using these positive affirmations in my final physical preparations for the ride. I'll be stretching with my CORE stretching routine after a light spin today to open and release stress from my body. Tomorrow, one day before the ride I will also do some stretching and a little bit of my yoga from ZEN to work on my breath and relax and open my mind and body. Works great! Make Your Persistence Pay Off!

Anyway, this is not about my persistence, but about yours. I want to encourage you to be persistent at everything you care about and have passion for. Cycling or otherwise.

Persistence is the ONE thing that you have complete control over regardless of talent, genetics, and ability.

One of the things I've learned from working with thousands of people over the years is that the ones that succeeded are usually the ones that failed the most! Yes, you read that correctly.

It was their persistence to get back up and try again and again and again that made them successful. Do you know how many attempts it took Thomas Edison to invent the light bulb? Over two years of failed attempts! 6,000 different carbonized plant fibers that didn't work! See . . . persistence pays off!

Whether you choose to ride a bike to lose 20 pounds and reduce your cholesterol and blood pressure or you choose to ride for the conquest of miles traveled or you ride for the competition to move one day from CAT 4 to CAT 1 racing. Do it with persistence! Do it with unrelenting determination!

Never stop. Never stop believing in yourself. You can do anything you set your mind to. More importantly . . . set your heart to.

I don't care if my persistence actually makes you buy= something from me or not. I'm glad just to have you here and learn from you along the way.

What I do hope is that you learn from my experiences, failures, and triumphs and you recognize that it's my persistence that makes me successful. Use me as a launching pad to motivate yourself to pursue your dreams and ambitions. Use me as a persistent reminder that no matter what the obstacles, there is always a way to get over, under, or around them.

If you want to live your dream, you have to get off your butt and go and get it. Expect to fail! In fact . . . expect to fail a lot! I did! BUT, there is no other way to achieve your dreams and accomplish your goals than to try. Especially when you try with persistence!