Pamela K. Keel

Eating Disorders

SECOND EDITION

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Preface

I wrote the first edition of *Eating Disorders* to address a problem I encountered while teaching an undergraduate course on this topic. After using a sourcebook of peer-reviewed journal articles for two years, I realized that before progressing to the primary literature, my students needed a text that summarized and synthesized the research base. The problem was that no such text existed. My solution was to write the book I needed and hope that it would be helpful to other instructors and students facing the same problem. This means that I am both the author of this book and an instructor who has used it in my courses. As time passed, I became increasingly (and at times uncomfortably) aware of the gap between what the first edition covered and what was now known about eating disorders. Initially this gap gave me an opportunity to present new and exciting findings in my lectures to supplement the reading. However, as more time passed and the gap grew wider, I knew that the only way to effectively teach my students was to write a second edition.

Since the publication of the first edition of this book in 2005, many new and important findings have emerged in the field. A new edition of the *Diagnostic and Statistical Manual of Mental Disorders* was published, altering the definitions of existing eating disorders and including new eating disorders. Indeed, my own program of research has focused heavily on defining and characterizing the new syndrome of purging disorder—a condition that was not even mentioned in this book's first edition! To reflect these and other advances in the field, information on purging disorder and night eating syndrome has been added to the second edition along with new case studies for each, and a new chapter has been included to address the related conditions of feeding disorders and obesity. The book has also been updated with new findings on the epidemiology of eating disorders to reflect the replication of the National Comorbidity Survey and several epidemiological studies focusing on eating disorders in racial and ethnic minority groups. Other changes include an increased focus on eating pathology in boys and men, more discussion of the influence of peers and social media on eating disorder risk, and descriptions of new findings from neuroimaging studies. One of the most exciting aspects of working on the second edition was the opportunity to

review new studies of prevention programs that reduce risk for the onset of eating disorders, as these studies have fundamentally altered conclusions regarding the success of prevention efforts in the field.

While the coverage of information on eating disorders has been fully updated for the second edition, the topic of eating disorders remains compelling for the same reasons as presented in the first edition. Eating disorders provide a perfect opportunity to examine the intersections of culture, mind, and body. To truly appreciate the causes and consequences of these disorders, one must be willing to consider topics that span the humanities (history, art, and literature), the social sciences (psychology, anthropology, women's studies, and economics), and the natural sciences (anatomy, physiology, pharmacology, and genetics). As a consequence, there is truly something for everyone in the study of eating disorders. Few topics of inquiry allow individuals from so many disciplines to make significant contributions.

Eating disorders are all around us. Almost anyone who picks up this book knows someone who has suffered from an eating disorder. Unlike numerous other topics in academia, eating disorders are often part of our personal lives. Even individuals who are fortunate enough to have never had an eating disorder or watched a loved one suffer from an eating disorder probably know someone who has.

Eating disorders are very topical. Many famous individuals have acknowledged the impact of these disorders on their lives. Thus even people who have not personally known someone with an eating disorder have a sense of familiarity with the problem. This topicality has two consequences. First, people probably know more about eating disorders than about many other subjects that might be covered by a textbook. Second, they probably have far more misinformation about eating disorders than they do about other textbook topics. Thus eating disorders can be both familiar and challenging (rather than more common combinations of familiar and boring or challenging and intimidating).

The field of eating disorders is still young. Sections of this book were difficult to write because there remains much that we simply do not know about these disorders. However, this limitation also represents an opportunity. Because there is so much left to learn, there are many ways that people can make a significant contribution to the knowledge base of these disorders. Young people have completed many fascinating and illuminating studies in the eating disorders field. This book includes many studies conducted by college undergraduates because of the important conclusions that can be drawn from them.

Case Studies

Like most textbooks on psychopathology, this one uses case studies to help bring its subject matter to life. Case studies are particularly important for illustrating eating disorders, since these disorders never exist in a vacuum but rather occur in the context of an individual's life. To balance the competing demands of breadth and depth, five case studies are followed throughout the book. Instead of presenting 25 different cases briefly, the book integrates the topics of different chapters into the lives of these five individuals, providing further insight into each one. For this reason, it is best if the chapters are covered in order. Reading Chapter 1 first would be worthwhile even for individuals who are intimately familiar with

the definitions of eating disorders, because that is where I introduce the cases that guide the reader throughout the text. Similarly, even if the time constraints that are present in any course mean that the final chapters are not part of the assigned reading, it is still worth reading them to learn more about how things turn out for the individuals introduced in Chapter 1.

Features for Students

Terms that may be new to students are defined within chapters and are included in a glossary at the end of the book. Glossary terms are presented in boldface type in the text and are listed at the end of the first chapter in which they play a key role. Italic type is used for other key terms to draw students' attention to important topics within chapters. Tables and figures are also included as study aids. While figures reinforce the information described in the text, tables often provide additional information. Thus figures are illustrative, and tables provide concise reviews of information often not presented elsewhere in the book. Each chapter includes a brief conclusion. The conclusion is not intended to serve as an abstract for the full chapter. Instead, it provides empirically supported take-home points regarding the topic of the chapter (when such conclusions are possible).

This book includes a chapter devoted to research methodology (Chapter 4), with examples from studies of eating disorders. This chapter is designed to enable students who have not completed prior coursework on research methods to critically evaluate the strengths and weaknesses of conclusions drawn from the empirical literature. The chapter also may serve as a refresher for students in advanced psychology courses who have already completed coursework on research methods.

Acknowledgments

I want to start by thanking the instructors and students who used the first edition of *Eating Disorders*. Writing is an important form of communication only when someone reads what you have written. Without those readers, there would be no second edition. I want to thank the reviewers of the chapter drafts, who provided exceptionally helpful feedback regarding opportunities to improve coverage of this growing field so that the book remains useful for a broad audience. I also want to thank the students who have taken my eating disorders class, because they have provided valuable feedback (both positive and negative) about aspects of the first edition which were most and least effective for their learning. Comparison of the first and second editions will reveal a dramatic decline in the number of detailed tables: Students talked, and I listened.

I am deeply indebted to the efforts of many members of the Oxford University Press staff, including Sarah Harrington and Andrea Zekus.

Finally, I want to thank you for your interest in this topic.

Pamela K. Keel

Eating Disorders

1

Introduction

What Are Eating Disorders?

Eating Disorders provides a thorough, research-based overview of current knowledge about eating disorders, including anorexia nervosa, bulimia nervosa, binge-eating disorder, and otherwise specified eating disorders. In addition, the book reviews disordered eating as the pathological end on a continuum with normal eating. Topics are treated from various perspectives to represent the different theoretical orientations in the field. Covered topics include who suffers from eating disorders, including historical and cross-cultural cases of eating pathology; biopsychosocial bases of eating disorders; and treatment and prevention of eating disorders. This exploration integrates findings from theoretical and empirical publications and journal review articles. The text also presents current understandings of the causes, correlates, and outcomes of eating pathology as well as covering the complexity and controversy surrounding these topics. Rather than pointing to one underlying cause for all eating disorders, this book strives to reveal how multiple factors conspire to produce these debilitating and sometimes deadly disorders.

Prior to embarking on this detailed exploration of eating disorders, it is important to define them—the subject of this chapter. Eating disorders are a form of mental disorder recognized in psychology, social work, nutrition, and medicine. Consistent with methods used to define other mental disorders, diagnostic criteria have been established for eating disorders and have been presented in widely used texts, such as the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*, published by the American Psychiatric Association) and the *International Classification of Diseases* (*ICD*, published by the World Health Organization). Three eating disorders—anorexia nervosa, bulimia nervosa, and binge-eating disorder—have received the majority of attention within research and clinical spheres. In addition, there are otherwise specified eating disorders, including purging disorder and night eating syndrome, that have received less study but are gaining greater attention. This chapter describes these syndromes and provides case histories that exemplify patients suffering from each of these disorders. The definitions of these disorders are not set in stone. They have and will continue to change as more is learned. Thus rather than

necessarily representing the discovery of a "natural order" of mental illness, the definitions that follow offer current descriptions of these disorders that allow them to be recognized in clinical settings and studied in research settings.

Anorexia Nervosa

Anorexia nervosa (AN) can be characterized as a self-starvation syndrome. The major sign of the syndrome is emaciation caused by deliberate restriction of food intake. In addition, there is an intense fear of becoming fat or gaining weight, which the AN sufferer may describe explicitly or may express implicitly as behavior aimed at preventing weight gain. Finally, there are body image disturbances, which may include a misperception of being overweight despite being emaciated, undue influence of weight or shape on self-evaluation, or poor recognition of the serious medical consequences of low weight. Diagnostic criteria for AN have been published in the fifth edition of the *DSM* (*DSM-5*; American Psychiatric Association, 2013) and the 10th edition of the *ICD* (*ICD-10*; World Health Organization, 1998). Table 1.1 highlights differences in how these two systems define the syndrome, demonstrating the extent to which definitions are neither universal nor set over time. Indeed, the *ICD-10* is currently being revised to be published as the *ICD-11*.

There is no specific duration criterion for a diagnosis of AN, and there is no universally accepted threshold for defining low weight. Instead, the clinician determines whether a patient has reached a low weight in consideration of the individual's age, gender, weight history, and medical function. The *DSM-5* gives a **body mass index** (BMI) less than 18.5 kg/m^2, as a guideline for recognizing low weight based on the World Health Organization's and Centers for Disease Control's definition of this threshold as the lower bound of a healthy

TABLE 1.1 Differences Between *DSM-5* and *ICD-10* Definitions of Anorexia Nervosa

	DSM-5	***ICD-10***
Weight criterion	Includes qualitative descriptor "significantly low weight" and a guideline of BMI < 18.5 kg/m^2 for assessing whether adult weight is below threshold.	Includes guidelines for body weight of "at least 15% below that expected" or BMI < 17.5 kg/m^2.
Behavior	Specifies restriction of food intake relative to the body's requirements.	Describes combination of avoiding fattening foods with purging (self-induced vomiting or use of laxatives or diuretics), excessive exercise, or use of appetite suppressants.
Endocrine function	Does not require loss of menstrual cycles (amenorrhea) for women.	Includes amenorrhea for women and loss of sexual interest or potency in men and notes other endocrinological disturbances that may be present (e.g., alterations in growth hormone, cortisol, thyroid hormone, and insulin).
Development	Makes no comment on developmental abnormalities	Notes that pubertal development is delayed or arrested if onset is prepubertal.

weight range for adults. (BMI is calculated by dividing an individual's weight by the square of his or her height.) For children and adolescents, a BMI that falls below the 5th percentile for age marks low weight under the *DSM-5* guidelines. However, an individual with a weight above these thresholds may still be considered to have a significantly low weight, depending on other indicators.

There are two subtypes of AN, the restricting type (ANR) and the binge-eating/purging type (ANBP), characterized by the current behavioral features of the illness. The restricting type of AN represents the illness as portrayed in most popular depictions. Individuals with ANR lose weight through restricted food intake, relying on their bodies rather than food to supply the fuel necessary to sustain their level of activity, which is often very high. As the name implies, individuals with ANBP interrupt their dietary restriction by engaging in binge eating, purging, or both behaviors. Although a person with ANBP may meet almost all diagnostic criteria for bulimia nervosa (BN), defined later in this chapter, the presence of AN precludes a diagnosis of BN in the *DSM-5*. Research supports the hierarchy of diagnosing ANBP rather than giving dual diagnoses of AN and BN. Women with ANBP demonstrate more similarities to women with ANR on treatment response and long-term outcome than to women with BN (see Chapters 9 and 11). Moreover, because patients with both subtypes of AN are characterized by medically low weight, treatments must address their medical needs.

Differences between the ANR and ANBP groups have also been reported. Compared with ANR, ANBP is associated with older age, greater impulsiveness, more substance use disorders, and more suicidal behavior. In general, these differences support the distinct behavioral presentations of the two subtypes. Individuals with the ANR subtype have more perfectionistic and constrained eating and other behaviors, while those with the ANBP subtype have more impulsive and out-of-control eating and other behaviors (see Chapter 7). However, longitudinal data (Eddy et al., 2002, 2008; Strober, Freeman, & Morrell, 1997) suggest that a high proportion of women with ANR develop binge-eating episodes later in the course of the illness. Thus, for many individuals, the two subtypes may reflect different stages of the same illness.

Anorexia nervosa predominantly affects women. Across studies the ratio of AN among those seeking treatment for the illness is approximately 10:1 between women and men. The percentage of women who have had AN over a 12-month period (**12-month prevalence**) is 0.4% (American Psychiatric Association, 2013). The proportion of women suffering from AN increased over the 20th century (Keel & Klump, 2003), with the greatest increases observed in adolescent girls. Anorexia nervosa usually develops during middle to late adolescence (ages 14–18 years).

Case Study: Emily

Emily, a 19-year-old sophomore at a large state university, bluntly stated that she had been "forced into treatment" by her school. Emily made it clear that she thoroughly resented the university's interference in her private life, since she had top grades in all classes and was clearly fine. Emily saw no reason to be in therapy or any sort of treatment. At 5 feet 10 inches tall and 109 pounds, Emily had a BMI of 15.7 kg/m^2, well below the 5th percentile for

someone the same age and in the severe range for an adult with AN. Emily's college roommates were extremely worried, because she had needed to be taken to the emergency room after fainting in the dining hall. When asked about the incident, Emily said that she had lost track of the time, hadn't eaten all day, and had become lightheaded after an afternoon run. However, Emily asserted that this was very unusual behavior and that she always had a high-energy snack before exercising. In fact, Emily said she always carried food because of a tendency to be hypoglycemic and, in contrast to what people thought, was eating all the time.

When asked what she would eat during a given day, Emily described having cereal for breakfast, snacking throughout the day, having a salad for lunch, snacking throughout the afternoon, and then eating a full dinner. On further questioning, Emily reported eating one packet of plain instant oatmeal made with spring water for breakfast in her room. The snacks consisted of celery sticks, carrot sticks, or sugar-free gum. Lunch was a "huge" plate of salad greens without dressing from the dining hall's salad bar. Dinner was the only meal that varied from one day to the next. Emily might eat a skinless chicken breast with half of a baked potato and a green vegetable. Occasionally, she ate half a cup of pasta with tomato sauce and vegetables added from the salad bar. On days when the dining hall served nothing she liked, Emily ate two slices of bread with cottage cheese spread over each slice and tomatoes on top—she likened this to pizza—along with a large salad. Based on this self-report, Emily's average daily caloric intake was estimated at less than 500 kilocalories (kcal). In comparison, average daily energy needs for a woman of the same age would fall between 1,500 and 1,800 kcal, depending on daily activity level.

Emily stated that she didn't eat red meat because she didn't like the idea of eating cute, furry animals. In fact, Emily didn't care for meat as a food group but made sure to always include protein in her diet because it was important for muscle development. Emily considered muscle development important because of feelings of constantly struggling with a "lopsided" body. Emily described herself as having a "classic pear shape," with shoulders and arms that were too thin and sticklike, and rotund hips, thighs, and buttocks. To improve her muscle definition, Emily exercised rigorously, running every afternoon between classes and dinner. On weekends, Emily added weight training. She had read that metabolism increased both during and after exercise and felt that this pattern increased the probability that dinner would fuel her body rather than being stored as fat. Emily was terrified of becoming fat.

Emily's concerns about weight had emerged during middle school. Always tall for her age, Emily felt like an "amazon" after entering puberty, because she towered over all of her classmates, including the boys on the football team. In an effort to fit in, she began to diet and lose weight. Emily's mother attributed the weight change to a loss of "baby fat," and friends expressed admiration of her self-control. Emily once was approached in the shopping mall, asked if she had ever considered becoming a model, and given the card of a modeling agency. Although flattered, Emily did not pursue this opportunity because she planned to go to a good college, then go to law school, and eventually become a judge. A career in modeling, Emily felt, would be a waste of her intellect because it required people to focus on superficial things like appearance.

Emily meets the *DSM-5* diagnostic criteria for ANR. She openly expresses a fear of gaining weight, deliberately restricts food intake, and exercises to maintain a weight that

is well below healthy limits for someone her age. The episode of fainting in the dining hall provides a clear indication of insufficient nutrition to maintain normal processes; the loss of consciousness might be explained by hypoglycemia, low blood pressure, or anemia.

Bulimia Nervosa

Bulimia nervosa is characterized by recurrent binge-eating episodes coupled with inappropriate compensatory behavior and undue influence of weight or shape on self-evaluation. Binge eating differs from normal eating in that it involves consuming a very large amount of food within a limited time (typically within two hours) and experiencing a loss of control over eating during the episode. Inappropriate compensatory behaviors include self-induced vomiting, laxative abuse, diuretic abuse, fasting, and excessive exercising. Of these methods, self-induced vomiting is the most common in BN. The first three compensatory methods are considered purging, because they all involve the forceful evacuation of matter from the body. Fasting and excessive exercise are considered nonpurging forms of compensatory behavior, because caloric intake during binge episodes is balanced by not allowing calories into the body or by engaging in strenuous physical activity so that the body burns calories at a higher rate, respectively. The *DSM-5* criteria for BN, unlike those for AN, include a minimum frequency and duration: Binge-eating episodes and inappropriate compensatory behavior must occur, on average, at least once a week over a three-month period. Table 1.2 presents key differences between the *DSM-5* and *ICD-10* diagnostic criteria for BN.

Like AN, BN predominantly affects women, with an estimated 10:1 ratio of women to men suffering from the illness. The overall percentage of women who have had BN over a 12-month period is approximately 1.0–1.5% (American Psychiatric Association, 2013). As with AN, the proportion of women suffering from BN increased in the second half of the 20th century (Keel & Klump, 2003). Bulimia nervosa usually develops during late adolescence to early adulthood. Thus individuals with BN tend to be older than individuals with AN, and approximately 30% of women with BN have a history of AN before developing BN.

TABLE 1.2 Differences Between *DSM-5* and *ICD-10* Definitions of Bulimia Nervosa

	DSM-5	***ICD-10***
Binge eating	Focuses on subjective experience of loss of control over eating.	Describes "preoccupation with eating" and "irresistible craving for food."
Body image disturbance	Focuses on undue influence of weight and shape on self-evaluation.	Describes a drive for an unhealthy low weight and a likely history of anorexia nervosa.
Behavioral frequency	Requires a minimum frequency of binge-eating episodes and inappropriate compensatory behaviors of once a week for three months.	Includes no minimum frequency or duration requirements.
Co-occurrence with anorexia nervosa	Precludes diagnosis of bulimia nervosa in individuals also meeting full criteria for anorexia nervosa.	Permits diagnosis of both bulimia nervosa and anorexia nervosa at the same time in the same individual.

Case Study: Jean

Jean was a 27-year-old secretary who lived with her boyfriend of two years. She was 5 feet 4 inches tall and weighed 138 pounds. Jean came in for treatment because of a return of eating problems that she thought had ended in college. In college, Jean had experienced binge-eating episodes and had engaged in self-induced vomiting. Jean spent a great deal of time trying to hide these behaviors from roommates and from family when home during breaks. However, her roommates confronted Jean after a particularly bad episode in which she had gone to the bathroom to vomit four times within a two-hour period.

Treatment had allowed Jean to stop binge eating and purging on a regular basis. Jean continued to have occasional slips—times when she felt she had eaten too much and purged to avoid weight gain. However, these occurred rarely, and sometimes Jean prevented herself from vomiting after "eating too much." About a year and a half ago, Jean had noticed that she was gaining weight and could no longer fit into the same size jeans she had worn since high school. Jean couldn't bring herself to buy larger jeans because she couldn't feel good about herself unless she fit into that specific size. She decided to diet and go to the gym more regularly. At first, the new fitness routine worked, and Jean lost approximately seven pounds. At a weight of 125 pounds, Jean felt great and was more likely to want to go out with friends and to flirt with and get attention from men.

However, when Jean and her boyfriend started living together, she had a hard time resisting the tempting foods he kept in the kitchen. While living alone, Jean had never had cookies, ice cream, or potato chips in the house, because these had been common triggers for binge-eating episodes. Now these foods were always around. At first, Jean simply resisted eating them because they were not part of her diet. However, one night, while her boyfriend was out with his friends, Jean ate an entire bag of potato chips and finished off a package of cookies and three-quarters of a gallon of ice cream. Disgusted with herself and in pain from consuming so much food, Jean made herself throw up. Afterward, she went to the store to replace the food. To hide the fact that she had bought new food, Jean used the garbage disposal to get rid of some of the new ice cream and cookies so that the packages looked as they did before. Jean vowed not to eat any more of these "dangerous foods" and told herself that this was just a slip. However, the next week, when Jean was alone in the apartment, the same cycle happened again. She would binge and purge only when alone, because that was when the impulse became irresistible.

Jean was now binging and purging several times a week, even leaving work early to get home to binge and purge before her boyfriend arrived. Jean had regained the lost weight and had found that her weight was creeping above its pre-diet level. She then redoubled her efforts at dieting, as well as using self-induced vomiting, to counteract the effects of the binge episodes. Jean even began vomiting when eating normal amounts of food, because it felt necessary to eat as little as possible to get rid of the unwanted weight. She felt disgusted with herself. As Jean's weight increased, she felt worthless and revolting.

Jean meets the *DSM-5* diagnostic criteria for BN. Her behavior appears to be a recurrence of the eating disorder from college. It is unclear whether Jean ever fully recovered from her earlier disorder, because she seemed to continue to base her self-evaluation on her weight and shape even after the binge eating and purging had gone into remission. In addition,

when living alone, Jean had avoided foods that she felt might trigger a binge-eating episode rather than feeling able to eat these foods in moderation.

Binge-Eating Disorder

Binge-eating disorder (BED) is characterized by recurrent binge-eating episodes in the absence of inappropriate compensatory behavior. Binge episodes in BED are defined as they are for BN and are also characterized by eating more rapidly than normal, eating until feeling uncomfortably full, eating large amounts of food when not hungry, eating alone because of feeling embarrassed by the amount eaten, and feeling disgusted, depressed, or very guilty after overeating. For a diagnosis of BED, three of these characteristic features are required; however, from the case study of Jean, it is clear that many of these factors also characterize binge episodes in BN. In addition, individuals must experience marked distress regarding their binge eating. As with BN, the *DSM-5* diagnostic criteria for BED include a minimum frequency and duration: Binge-eating episodes must occur on average at least once per week over a period of three months. Also as with BN, there is a diagnostic hierarchy such that a person who simultaneously meets criteria for AN and BED is given a diagnosis of ANBP only. Because a diagnosis of BED requires the absence of inappropriate compensatory behavior, it is also not possible to be diagnosed with both BN and BED. Binge-eating disorder was included as a provisional diagnostic category in the fourth edition of the *DSM* (*DSM-IV*; American Psychiatric Association, 1994) and added as an official diagnostic category in the *DSM-5*. Binge-eating disorder was not included in the *ICD-10* but may be added to the *ICD-11*.

Like AN and BN, BED is more common in women than in men (Hudson, Hiripi, Pope, & Kessler, 2007); however, the gender difference is not as dramatic. In US adults, 1.6% of women and 0.8% of men reported having BED over the past 12 months (American Psychiatric Association, 2013), suggesting a 2:1 ratio of women to men. The age of onset for BED may have a bimodal distribution, with many individuals reporting problems with overeating since childhood and others reporting significant problems beginning in late adolescence or early adulthood after a period of significant dieting (Manwaring et al., 2006; Spurrell, Wilfley, Tanofsky, & Brownell, 1997). Individuals with BED tend to be significantly overweight or obese. However, this is not a requirement for diagnosis, and it is not true for all individuals with BED (Keel, Holm-Denoma, & Crosby, 2011).

Case Study: Jamie

Jamie's problem was simply stated: "I eat too much. For some people, it's alcohol; for some it's cocaine. For me it's food." This had always been true; even when Jamie was a small child, a whole box of Twinkies was a single serving. For a junior high school bake sale, Jamie's mother had baked a cherry pie. The pie, Jamie's favorite dessert, was gone before the start of school that day. When the teacher asked for the dessert, Jamie lied to conceal the gluttony. There had been many times like this throughout childhood—episodes of eating all of something rather than just one serving. However, because Jamie was tall and athletic, that big appetite was often a source of pride rather than embarrassment. In fact, everyone on the athletic teams ate large amounts of food, so Jamie didn't feel unusual most of the time. Jamie

didn't realize there was any eating problem until the end of college, when job interviews started. It was the first time Jamie needed to buy a suit but couldn't fit into any of the sizes offered in the normal department store. Jamie was embarrassed by having to go to a special store that stocked larger sizes.

At 35 years of age, Jamie currently weighed 360 pounds, despite several diets and weight loss programs. Jamie was frequently able to lose some weight on these programs; the greatest loss had been 50 pounds, down from 280 to 230 pounds. However, as at all other times, the weight had come back—and more. Jamie denied eating when not hungry but acknowledged eating to the point of being uncomfortably full. Jamie felt that this was because, when hungry or with favorite foods, eating occurred at one rate: "as much and as quickly as possible." Jamie said it was like being a "food addict"; there was no way to stop until all of the food was gone. For example, Jamie would consume three "value meals" from the local fast-food restaurant in the car on the way home from work. Eating alone in the car, Jamie said, was "the best," because "I can just zone out." Terrible guilt followed these episodes, because Jamie knew that eating so much junk food contributed to the weight problem—and could lead to heart problems. However, Jamie didn't like salads, vegetables, or fruit because they were bland and boring. "I wish I felt about fast food the way that I feel about salads because then I would be thin as a rail." Recognizing the existence of an eating problem that occurred every day—often throughout the day—Jamie wanted to know if there was any medication that would cause weight loss or make it easier not to eat. Based on all of the TV advertisements for such products, Jamie felt like a good candidate for medication.

Jamie meets the *DSM-5* diagnostic criteria for BED. Although the distress over eating was a consequence of being overweight, the extent to which Jamie viewed the eating problems as contributing to the weight problems led Jamie to experience marked distress over the binge eating as well.

Other Specified Feeding or Eating Disorder

The *DSM-5* category of "other specified feeding or eating disorder" (OSFED) represents a diverse set of clinically significant disorders that do not meet the specific diagnostic criteria for AN, BN, or BED. Clinical significance is defined in terms of current distress and impairment due to disordered eating. Individuals who have trouble fulfilling major role obligations (e.g., missing or performing badly at work or school) or who experience social problems as a consequence of their disordered eating can be diagnosed with OSFED. Conditions within this category have sufficient evidence to be named and described, but more study of these diagnoses is needed to understand their clinical utility for predicting treatment response, course, and outcome.

Table 1.3 lists examples of OSFED from the *DSM-5* with brief descriptions. In some cases, the disorders are characterized as "subthreshold" because they resemble defined eating disorders but fall short of full diagnostic criteria (e.g., bulimia nervosa of low frequency and/or limited duration). Other disorders simply differ in clinical presentation from the defined eating disorders (e.g., night eating syndrome). While some cases of OSFED are less severe than their full-threshold counterparts (Garfinkel, Lin, et al.,

TABLE 1.3 Other Specified Feeding or Eating Disorder in *DSM-5*

Disorder	Description
Atypical anorexia nervosa	Individuals are not underweight despite the significant restriction of food intake, weight loss, and body image disturbance that characterize AN.
Bulimia nervosa of low frequency and/or limited duration	Individuals have episodes of binge eating and inappropriate compensatory behavior less than once a week, for less than three months, or both. Individuals also experience undue influence of weight and shape on self-evaluation.
Binge-eating disorder of low frequency and/or limited duration	Individuals have episodes of binge eating less than once a week, for less than three months, or both. Individuals report at least the 3 of 5 features that's a criterion for BED associated with their binge-eating episodes as well as marked distress over their binge eating.
Purging disorder	Individuals use self-induced vomiting or laxatives, diuretics, or other medications to purge following consumption of normal or small amounts of food but are not underweight.
Night eating syndrome	Individuals experience recurrent nocturnal eating episodes (waking from sleep to eat at night or excessive food intake following dinner) that they remember and that contribute to distress or functional impairment.

1995), this is not true for all forms of OSFED. For example, a person who purged multiple times a day for over a year without binge eating would have the OSFED of purging disorder, and no one would argue that this individual had a less severe eating disorder than someone who met the BN criterion of binge eating and purging once a week for three months.

In older editions of the *DSM*, OSFEDs fell within the broad category of "atypical eating disorder" (American Psychiatric Association. 1980) or "eating disorder not otherwise specified" (American Psychiatric Association, 1987; 1994). However, as is evident from Table 1.3, those labels had very little meaning, given the heterogeneity of clinical presentations that are defined by not being AN of BN. The decision to name (i.e., specify) these syndromes was intended to spur more research, given the tendency to "study what we define" (Walsh & Kahn, 1997).

Case Study: Valerie

Valerie wasn't sure if she had a "real" eating disorder. She never identified with the magazine stories about skeletal actresses terrified of becoming fat or with TV movies about desperate teens who seemed to have perfect lives but who secretly gorged on food to stuff their feelings down and then vomited to void their emotional pain. Valerie knew that what she did wasn't "normal" and understood that it might be dangerous—the blood in her vomit worried her – but she wasn't sure if she had an "eating disorder." The "aha" moment came when a link on "purging disorder" drew her attention while searching the Internet for information about vomiting blood. On reading the associated article and viewing a brief video clip, Valerie found that they matched what she had been doing for the last eight years. Valerie started scouring the Internet for more information, eventually finding an e-mail address for the person in the video clip and composing a message—a combination of affirmation that the disorder was real and a request for help. Valerie paused, wondering if she really wanted to

attach her name to an admission of what she had been doing and if the person in the clip would even read or respond to the message. Finally, Valerie hit "send," exhausted from eight years of trying to convince herself that if no one ever noticed or asked about the vomiting, it must not be that big of a deal.

This was the message Valerie sent:

> I just found an article on Purging Disorder and wanted to know if you needed any one for your research. I think I've had this Disorder for several years. I had a bad case of mono in secondary school and lost over 4 kilograms while I was sick. I didn't want to gain the weight back. So, I tried to eat only what I had been eating whilst sick—chicken noodle soup, saltine crackers, and water. That worked for a while, but then my parents were worried that something else was wrong, and I started trying to eat normally around them so they wouldn't worry. I remember the first meal I got rid of—it was my mum's Shepards Pie. I felt so sick afterwards, like I had eaten the whole dish. I felt bloated and gross and was convinced that after weeks of living on chicken noodle soup and crackers, I had ruined it all with one dinner. So, I went to the bathroom, locked the door, turned on the shower, and threw up in the toilet until my stomach felt completely empty and clean. I felt relieved and in control again. I don't throw up every time I eat. Sometimes I can eat and be fine. Sometimes even a small amount of food has to be gotten rid of. But I never have huge binges, and I've never starved myself, and I've never gotten very thin. I am the thinnest that I've ever been, and everyone tells me that I look great and that I shouldn't worry about my weight. Of course, they don't know what I do to keep the weight off. What frightens me most is that if I eat normally and don't purge, then I would get really fat. But I'm also frightened that I'll never be able to eat normally, and I don't know when I'll stop this. I'm hoping that you can tell me what to do.

Valerie's pattern of eating is consistent with a diagnosis of purging disorder. Although the eating disorder has kept her weight lower than it might otherwise be, it is within the healthy range, and Valerie has not lost substantial weight. Thus neither AN nor atypical anorexia nervosa fits the symptoms. Valerie does not experience episodes of large, out-of-control eating binges and thus cannot have BN. Instead, Valerie vomits after normal or small amounts of food. It is unclear how often she purges, but this behavior represents a recurrent pattern, causes distress, and causes medical problems.

Case Study: George

George originally sought help through a sleep clinic because of trouble sleeping. His snoring was so bad that he no longer was able to sleep in the same bed as his wife. George described spending half of the night sleeping on the couch and half of the night up and feeling exhausted the next day. Results of sleeping tests indicated that George suffered from sleep apnea, which was likely made worse by weight: at 45 years old, George was 5 feet 11 inches tall and weighed 260 pounds. He was given a special mask that would ensure sufficient oxygen flow while sleeping. In addition, George's doctor explained how even modest weight

loss and regular physical activity could improve sleep and quality of life. George felt like the sleep problems contributed to the weight problems, because frequently on waking up at night, he would get something to eat to help him feel sleepy. Once full, George would lie down on the couch in front of the TV and eventually fall asleep. The next morning, he didn't feel hungry at all.

The doctor asked more about George's eating patterns. George reported generally skipping breakfast, in order to get as much sleep as possible before going to work, and if he ended up getting into work late, skipping lunch as well to make up the time. George started feeling hungry around mid-afternoon. Thus he was always hungry for dinner and usually had a big meal. George joked that it was as if his whole body was on the night shift. More nights than not, George would have a second or third meal in the middle of the night, when everyone else in the house was asleep. He would eat the leftovers from dinner, finish with a bowl of cereal, and then, if still up and hungry, might make some scrambled eggs and toast. In George's mind this was like having breakfast before getting to sleep, since he wouldn't be hungry for it in the morning.

After listening to George describe this pattern of eating, the doctor suggested seeing a specialist in the eating disorders clinic of the hospital. George was skeptical about going, because he wasn't an underweight teenage girl. However, the doctor explained that his colleagues were studying a condition called night eating syndrome and might be able to help figure out a way to get George's eating on schedule while the team at the sleep clinic helped him get back to sleeping through the night.

George's eating patterns include **nocturnal eating**: eating at night after dinner, especially having to eat after waking from sleep in order to get back to sleep. The problems seem to stem from a disruption in circadian rhythm such that George's body is awake and wants food at night, when it should be asleep, and feels very tired but not hungry during the day. Although this pattern would be conducive to working the night shift, it is very disruptive for anyone with a nine-to-five job. Because George does not report a sense of loss of control over the night eating, the eating episodes do not meet criteria for binge eating, even if George consumes a larger amount of food than most people would eat under similar circumstances.

Like BED, night eating syndrome appears to be more common among overweight individuals. Not all individuals with night eating syndrome engage in nocturnal eating; some simply consume the majority of their calories in the later evening. It has been posited that digesting large quantities of food in the late evening may contribute to sleep problems. All of George's problems likely contribute to one another. Poor sleep contributes to the eating, which contributes to the weight, which contributes to the sleep apnea, which contributes to the poor sleep.

Unspecified Feeding or Eating Disorder

The final diagnostic category in the *DSM-5* is "unspecified feeding or eating disorder," which includes any significant disorder of eating that does not meet the criteria for AN, BN, or BED but for which the individual making the diagnosis does not specify the reasons. This diagnosis is often given when a clinician has determined that an eating disorder is present;

cannot fit the symptom profile to AN, BN, or BED; and does not have the time or resources to clarify on the nature of the problem but needs a diagnosis to make a referral to a specialist. This diagnosis also may be given for individuals who do not meet the full criteria for a feeding disorder (described in Chapter 2); feeding disorders (pica, avoidant/restrictive food intake disorder, and rumination) do not have named variants in the OSFED category.

That an unspecified category is needed reveals the extent to which observed eating problems exceed the number of conditions that have sufficient bases in research to be named and described in the *DSM-5*. Numerous labels have emerged (mostly in the popular literature) to capture these unspecified eating problems, including "orthorexia," "drunkorexia," and "female athlete triad," among many, many others. Briefly, orthorexia refers to individuals who feel compelled to follow rigid rules about eating "right." In orthorexia what counts as eating right tends to reflect concerns about health (e.g., eating organic foods) or being socially conscious (e.g., eating locally grown foods) but results in a highly restricted diet that interferes with the person's everyday life. Drunkorexia has been characterized in late adolescent and young adult individuals (e.g., high school and college students) who restrict their intake of food to "save calories" for excessive alcohol consumption. The female athlete triad involves (1) insufficient caloric intake relative to high caloric demands of athletic performance, (2) menstrual disturbances, including the loss of menstrual cycles (**amenorrhea**), and (3) bone loss (osteoporosis). The female athlete triad has a strong research base, with 239 citations emerging from a search of "female athlete triad" in the PubMed database (compared with 45 citations for "purging disorder"). It is unclear, however, whether the female athlete triad should be considered an eating disorder. It may best be considered a related condition that may or may not be accompanied by disordered eating. Chapter 2 considers other conditions that are related to eating disorders but are not themselves eating disorders, namely, feeding disorders and obesity.

Eating Disorders as a Continuum of Problems

Defining AN, BN, and BED and describing their prevalences represents a categorical approach to eating disorders; that is, an individual either does or does not have one of these disorders. Many experts prefer to think of eating disorders as existing on a continuum. One end of the continuum represents diagnosable eating disorders, and the other end represents healthy eating attitudes and behaviors. This perspective seems to match what is known about the presence of disordered eating attitudes and behaviors in the general population and in the course of individuals' lives. It also eliminates the need for separate categories for BN and bulimia nervosa of low frequency and/or limited duration and may even allow atypical anorexia nervosa to be placed on a continuum with its full-threshold counterpart.

Many people experience disordered eating at some point in their lives. Some may experiment with disordered eating behaviors and never develop any significant problems. Others, however, probably have genuine problems with eating and body image that do not fall within the narrow definitions given to the disorders characterized in the *DSM* or *ICD*. For example, body image disturbance may be present in many women who do not meet the

criteria for AN or BN. This problem may be coupled with severely restricted dieting and excessive exercise and may contribute to diminished self-esteem. Although such a pattern may not warrant the diagnosis of a mental disorder, it is likely worthy of both clinical attention and research. Understanding the factors that lead to disordered eating may help reveal the causes of AN, BN, BED, and related OSFED.

It is most likely that people with eating disorders (defined categorically) experienced disordered eating before developing full-threshold syndromes. For example, it seems highly unlikely that an adolescent girl would shift suddenly from completely healthy eating habits to full-threshold BN. Thus viewing eating disorders as a continuum helps us understand the progression of these problems within an individual's life.

Coinciding with the publication of the *DSM-5* by the American Psychiatric Association, the National Institute of Mental Health introduced an alternative approach to classifying mental disorders that focuses on dimensions of behavior, affect, and thought processes that might show stronger links to the biology of the brain than do the *DSM-5* categories. This alternative, called the Research Domain Criteria (RDoC), seeks to explain the full range of mental function from healthy to pathological (Cuthbert & Insel, 2013). The goal is to create new systems for classifying mental disorders that translate neurobiological and behavioral research into improved understanding and treatment of psychiatric illnesses. Toward this end, the RDoC approach accepts the concept of mental illness but largely ignores the diagnostic categories that have evolved from expert consensus and subsequent research.

The RDoC approach differs from the *DSM-5* in several key ways. First, it abandons the notion of mental disorders as discrete categories and instead views mental illness as residing on a continuum with healthy behavior, much as high blood pressure resides on a continuum with healthy blood pressure. Second, it emphasizes the function of the brain in contributing to mental illness and seeks to use modern neuroscience techniques to better understand psychopathology by identifying brain circuits linked to differences in how people think, feel, and behave. Third, the RDoC approach defines biobehavioral constructs (i.e., explanatory models) that connect the functions of brain circuits upward to the level of observable behaviors and self-reported symptoms and downward to the level of genes (see Figure 1.1).

While acknowledging the limitations of arbitrary frequency or duration criteria for distinguishing eating disorders from normal eating, a purely "dimensional" approach would equate the difference between 500 and 501 lifetime binge-eating episodes with the

	Units of Analysis							Paradigm
Domains/Systems	Genes	Molecules	Cells	Circuits	Physiology	Behavior	Self-Report	
Negative								
Positive								
Cognitive								
Social								
Arousal/Regulatory								

FIGURE 1.1. Research Domain Criteria (RDoC) matrix. Adapted from http://www.nimh.nih.gov/research-priorities/rdoc/research-domain-criteria-matrix.shtml.

difference between 0 and 1 lifetime binge-eating episode, even though the latter case distinguishes someone who has never binged from someone who has binged.

Whether or not eating disorders lie on a continuum, many practical decisions require a categorical approach. For example, the decision whether or not to provide treatment is categorical. Whether or not to include a person in a research study is also a categorical decision. Further, a categorical approach occasionally allows us to detect differences that lead to improved understanding or treatment. For example, BN responds to treatment with selective serotonin reuptake inhibitors (see Chapter 9), but AN does not. If no distinction had been made between AN and BN in treatment studies, this finding might have eluded eating disorder researchers.

Conclusion

There are three currently recognized forms of eating disorders: AN, BN, and BED. In addition, a diverse set of conditions are included in the OSFED category. Anorexia nervosa is best summarized as a self-starvation syndrome. Bulimia nervosa is most often a binge–purge syndrome. Both AN and BN involve significant disturbance of body image. Binge-eating disorder involves recurrent binge-eating episodes in the absence of inappropriate compensatory behavior. It does not require body image disturbance but is frequently associated with significant overweight or obesity. The diagnostic category of OSFED includes purging disorder (purging in the absence of binge-eating episodes) and night eating syndrome (consuming a majority of food at night) as well as syndromes that resemble AN, BN, or BED but fall short of meeting the full diagnostic criteria. In addition to the individuals who meet criteria for a *DSM-5* eating disorder, many more experience disordered eating behaviors or harbor disordered eating attitudes but never develop a diagnosable disorder. These individuals may be conceptualized as falling along a continuum with those who suffer from recognized eating disorders.

Roughly a third of this book focuses on describing eating disorders—what they are, what they are not, who they affect, and their history. Another third is devoted to understanding the **etiology** of eating disorders: what causes these conditions to occur. The last third of the book concerns the treatment, prevention, and outcome of eating disorders.

An important aspect of understanding what eating disorders are is appreciating what they are not. Chapter 2 focuses on conditions of feeding disorders and obesity that are sometimes confused with eating disorders because of overlap in their behaviors (too much or too little food intake) and outcomes (too much or too little weight). The chapter reviews similarities to as well as distinctions from eating disorders to clarify the book's focus on eating pathology.

Popular media has promoted a particular association between eating disorders and White girls and women in modern Western culture. Chapter 3 examines the **epidemiology** of eating disorders, including differences between genders and ethnic and racial groups across cultures and history. Epidemiological investigations allow identification of when, where, and in whom the risk of eating disorders is highest. As such, they are crucial for unraveling the causes of eating disorders.

Chapter 4 introduces approaches to understanding these causes. It presents the logic of research methods used in studies that are reviewed in subsequent chapters. This chapter discusses some risk factors for eating disorders; Chapters 5 through 8 present more complete reviews of social, psychological, and biological risk factors.

Social risk factors for eating disorders include both societal ideals and the immediate family and peer environments. Chapter 5 discusses the societal idealization of thinness and denigration of fatness, gender roles, and the impact of societal messages on women's body image and the pursuit of thinness. The associations among body image, weight control behaviors, and eating disorders are reviewed in this chapter. Chapter 6 addresses the role of families and peers. Because the influence of families can be interpreted at social, psychological, and biological levels, this chapter introduces a biopsychosocial model of the causality of eating disorders, which is elaborated on in later chapters.

Chapter 7 reviews psychological factors that contribute to the risk of eating disorders in addition to the family influences explored in Chapter 6. These factors include personality and cognitive and affective processes. The extent to which these factors represent causes or consequences of disordered eating is also discussed.

Chapter 8 introduces biological factors that contribute to the risk of eating disorders. In addition, it reviews biological correlates and consequences of eating pathology. This chapter marks the transition from examining the etiology of eating disorders to discussing outcomes, including treatment and prevention outcomes.

Chapter 9 covers treatment of eating disorders. It explores the role of different theoretical models in shaping treatment approaches. The **efficacy** of interventions is reviewed as well.

Chapter 10 discusses theories of prevention and evidence of the impact of preventive measures on disordered eating and eating disorders. This chapter gives examples of prevention programs aimed at three levels of intervention: a general middle school population, a more specific population consisting of college women (who are at higher risk of eating disorders than college men) recruited from sororities, and the still more specific group of college-age women reporting high levels of body dissatisfaction. Results from studies of prevention programs are described, including promising findings from efforts to reduce risk for eating disorders and obesity in schoolchildren.

Chapter 11 reviews the outcomes associated with eating disorders. Statistics on mortality, recovery, relapse, and crossover are presented for AN, BN, and BED, as well as forms of OSFED where available. Predictors of outcomes are discussed, with particular attention to the impact of treatment on long-term outcome.

Chapter 12 concludes the book by summarizing information in the context of the case histories that were presented in this chapter. In addition, Chapter 12 introduces current debates surrounding various topics reviewed in the book. The chapter ends with a discussion of future research directions in the field of eating disorders.

Key Terms

12-month prevalence
Amenorrhea
Body mass index
Efficacy
Epidemiology
Etiology
Nocturnal eating

2

Related Conditions That Are Not Eating Disorders

Chapter 1 defined what eating disorders are. This chapter delves into related conditions that are not eating disorders, focusing on those most closely linked to eating disorders: feeding disorders and obesity. While AN, BN, and BED are eating disorders, pica, rumination disorder, and avoidant/restrictive food intake disorder are feeding disorders. As recently as the revision of the third edition of the *DSM* (*DSM-III-R*; American Psychiatric Association, 1987), AN and BN were grouped with pica and rumination disorder in the category of "disorders usually first evident in infancy, childhood, or adolescence," with no distinction demarcating eating from feeding disorders. In the *DSM-IV* (American Psychiatric Association, 1994), AN and BN were moved to their own chapter, and the eating disorders category was officially distinguished from feeding disorders. To acknowledge a lifespan developmental perspective in psychopathology, the *DSM-5* (American Psychiatric Association, 2013) eliminated the broad (and largely meaningless) category of "disorders usually first diagnosed in infancy, childhood, or adolescence" and moved diagnoses that had been in that section to the most closely related chapters. Thus feeding and eating disorders were reunited in one chapter of the *DSM-5*, but the distinction between them was retained.

The rationale for combining feeding and eating disorders into one chapter was that all of them can present across the life span (Bryant-Waugh, Markham, Kreipe, & Walsh, 2010). That is, children can be diagnosed with AN, and adults may suffer from pica. However, the feeding and eating disorders have historically been distinguished by the ages of the individuals they predominantly affect. As reflected in the *DSM-III-R's* placement of feeding disorders in the diagnostic category of disorders usually first diagnosed in infancy, childhood, or adolescence, they typically emerge in infancy and early childhood, when parents are primarily responsible for children's nutritional intake. Eating disorders, by contrast, typically emerge during adolescence as children begin to have control (or a lack of control) over their own food intake.

Moreover, feeding disorders lack the body image disturbance that characterizes AN and BN and the distress that characterizes BED. They emerge at a time when children do not have abstract concepts regarding what their bodies should look like in terms of weight or shape. Thus children generally do not see their feeding behavior as motivated by body image concerns or as particularly problematic. Instead, it is usually parents, teachers, or doctors who become concerned about the behavior and poor nutritional status of children with feeding disorders. When distress does accompany feeding disorders, it is often related to conflict between the child and a parent or other caregiver. Stress in the parent–child relationship can arise when a child experiences an irresistible compulsion to consume nonfood (as we will see in pica) or to regurgitate food (as in rumination disorder) or feels significant fear over the consequences of eating (as in avoidant/restrictive food intake disorder). The adult may then try to convince, cajole, or otherwise coerce the child into eating normally, making mealtimes stressful for everyone.

Thus feeding disorders, unlike eating disorders, are diagnosed primarily on the basis of observable eating behaviors and the absence of associated cognitions. What follows is a description of the definitions of the feeding disorders in the *DSM-5*, along with their definitions within the *ICD-10* (World Health Organization, 1998) when available.

Pica

The defining feature of pica in the *DSM-5* is the consumption of nonfood substances that lack nutritional value for at least one month. Typical items include paper, cloth, chalk, paint, clay, or ash. Although these items lack nutritional value, several of them may have qualities that are beneficial to an organism. For example, children with calcium deficiencies may be drawn to chalk in an effort to make up for what they lack. However, individuals with pica maintain consumption of nonfood items for reasons other than nutritional needs. Importantly, consuming nonfoods can endanger health when the substances eaten include toxins (as with lead-based paint) or bacteria (as may be found in dirt).

Pica is often observed in individuals with developmental delays characterized by intellectual disabilities, and the diagnosis should be made only when the severity of disturbed eating requires clinical attention. Because small children will often explore their environments by placing nonfood items in their mouths, pica is diagnosed only when the consumption of nonfood items is inappropriate to the child's age. Further, consuming nonfood items cannot fall within a culturally supported tradition. The *ICD-10* guidelines for pica differ from those in the *DSM-5* in recommending pica to be diagnosed only when consumption of nonfood items occurs as a "relatively isolated psychopathological behaviour" rather than when it occurs as part of another psychiatric disorder, such as autism.

Case Study: Toby

During Toby's annual wellness check, his mother, Angela, shared with the pediatrician her concern that Toby was eating things that he shouldn't. When asked what he was eating, she said she had seen Toby licking the bottoms of his shoes and had also caught him eating dirt when he was playing in the backyard of their home. She had told Toby to "stop it" and that

the bottom of his shoes were "dirty," but she wasn't sure that Toby understood, because it was difficult for him to grasp the difference between the fact that he was eating dirt (which was "dirty") and the concept of "dirty" meaning disgusting. She tried to watch him closely to make sure that he didn't hurt himself, but she estimated that he was probably consuming dirt more days than not during the week, especially when he was at preschool. Toby attended a special preschool for developmentally delayed children where the teacher-to-student ratio was better than in a preschool for typically developing children, but it still wasn't a one-to-one ratio. In addition, Angela perceived Toby as being "sneaky" about eating dirt. She felt that he understood enough to know that she didn't want him to eat dirt but not enough to understand why it upset her.

The doctor asked whether Toby was having normal bowel movements or had shown any signs of illness, such as fever or nausea. Angela said that Toby had been complaining of an upset stomach and did occasionally have difficulty with constipation. She attributed the latter problem partly to his reluctance to disengage from whatever he was doing to sit on the toilet. The doctor told Angela that the biggest health concerns about eating dirt included consuming bacteria and parasites that inhabit animal feces. In addition, if eaten in large quantities, dirt could contribute to constipation and abdominal pain as well as nausea, vomiting, and diarrhea. The doctor ordered a medical workup including blood tests and an X-ray to screen for anemia and toxins and to check for blockages in Toby's intestinal track. The doctor also provided a referral to a child psychologist who specialized in behavioral treatments with developmentally delayed children.

This case study illustrates several features that make pica quite distinct from the eating disorders described in Chapter 1. First, Toby may or may not have control over his behavior. Second, even if Toby does not feel in control of his behavior, he does not feel distressed by it. Finally, no aspect of Toby's behavior is related to concerns about his weight or shape; specifically, he is not eating dirt as a low-calorie substitute for food.

Rumination Disorder

The central feature of rumination disorder is regurgitation of food that has already been swallowed. This behavior occurs for no medical reason, such as gastroesophageal reflux, and occurs without nausea, retching, or disgust. Food may be partially digested, rechewed and reswallowed, or simply spit out. The diagnosis of rumination disorder is not given when another eating or feeding disorder is present, and if another psychiatric disorder, such as intellectual developmental disorder, is present, the diagnosis is made only if the regurgitation is determined to require separate clinical attention. Unlike pica, rumination disorder is not included as a separate diagnosis in the *ICD-10*. Instead, it is described within the broader category of disorder of infancy and childhood and is included as an associated feature of other conditions.

Rumination disorder is distinct from BN, in which individuals consume large quantities of food and feel a loss of control over eating before they bring food back up. Rumination disorder is also distinct from purging disorder, because individuals with purging disorder do not reswallow the food they bring up. In addition, individuals with BN and those with purging disorder experience significant body image concerns that are absent in rumination disorder.

Case Study: Sally

Sally had been sitting quietly in an Overeaters Anonymous group, wondering if she was in the right place. Everyone was describing problems with controlling how much food they ate and the vicious cycle they felt caught in: feeling unhappy, eating to soothe their emotional pain, feeling disgusted with themselves afterward, eating to deal with that feeling, and so on. Sally could relate to feeling self-disgust. She felt it every day. However, her problem did not involve eating too much food. Instead, she was caught in a pattern of re-eating the same food. From an early age, Sally would bring some of the food she had swallowed back up into her mouth to chew again before finally reswallowing it. She wasn't really sure why she did this, and it hadn't bothered her as a child. She sometimes wondered whether it would have been easier for her to stop if she had been able to get treatment at a young age. Now it felt abnormal to her to try to chew and swallow food without ruminating. Her rumination behavior caused her significant distress and interfered with her relationships. Specifically, Sally avoided eating with others because she was afraid that she might regurgitate her food without even realizing what she was doing. On the occasions when she did go out to eat with someone, she tried to stick to foods that did not require much chewing, like soup, and focus very hard on "eating like a normal person." She had decided to attend the OA meeting because it was anonymous and free and because she hoped she might gain a better understanding of what she was doing and how to stop. However, she found it very difficult to relate to what others were saying, making her feel more alone and isolated than she had felt before coming to the meeting.

Although Sally's case study focuses on her symptoms in adulthood, she began her feeding pattern in early childhood. In infants and very young children, rumination is often linked to an immature digestive tract that makes it easier for food to come back up the esophagus. It is unclear whether Sally had delays in the development of her digestive tract that put her on the path to rumination disorder. However, she will likely need retraining around eating to overcome her compulsion to ruminate.

Avoidant/Restrictive Food Intake Disorder

As the name indicates, the central feature of avoidant/restrictive food intake disorder (ARFID) is restriction of food intake or avoidance of food that results in insufficient nutrition through eating. The *DSM-5* specifies that the intake must be insufficient through "oral intake of food" (American Psychiatric Association, 2013, p. 334) to allow diagnosis of ARFID in children who receive dietary supplements through tube feeding (see Chapter 9). In contrast to pica and rumination disorder, which were both included in prior editions of the *DSM*, avoidant/restrictive food intake disorder was introduced in the *DSM-5*, in an attempt to standardize a heterogeneous group of conditions identified predominantly in children that had been categorized as "feeding disorders of infancy or early childhood" in the text revision of the *DSM-IV* (American Psychiatric Association, 2000) and in the *ICD-10* (Bryant-Waugh et al., 2010). The *DSM-5* eating disorders workgroup acknowledged that the literature base for ARFID was weak but attributed the weakness in part to the lack of a uniform set of criteria or definition with which to study clinically significant deficiencies in children's food intake.

To meet *DSM-5* criteria for ARFID, individuals must display inadequate food intake that relates to a lack of interest in food or eating, to avoidance of food because of specific sensory characteristics (e.g., taste, color, or texture), or to fears about negative consequences of food intake (e.g., choking or gagging). This last reason for food avoidance is most likely to occur following a traumatic incident of choking. Avoidant/restrictive food intake disorder also may emerge in children whose mothers have excessive concerns about choking (Bryant-Waugh et al., 2010). In these instances, mothers may continue to cut food into small pieces beyond the age at which children typically develop the oral–motor skills to chew food enough to allow safe swallowing. A lack of experience with biting and chewing foods can then contribute to young children's fears that they may not be able to handle some foods. Whatever the underlying cause, ARFID results in an inability to meet nutritional needs and is characterized by significant weight loss, nutritional deficiencies (e.g., calcium deficiency), a need for tube feeding or nutritional supplements (e.g., liquid meals), or marked interference with social relationships.

ARFID cannot be diagnosed in individuals who meet diagnostic criteria for AN or BN, nor in individuals who experience the body image disturbance observed in AN or BN. As with pica and rumination disorder, if another psychiatric disorder is present, the diagnosis of ARFID requires that the avoidance of food be severe enough to warrant separate clinical attention. Finally, there must be no cultural or medical explanation for the food avoidance.

Case Study: Jackson

Kimberly brought her 3-year-old son, Jackson, to the doctor because she was worried about his eating and weight. Jackson had been small for his age since birth, but recently he had developed very rigid rules about what he would and would not eat. Kimberly understood that many 3-year-olds were "picky eaters," but Jackson's behavior seemed to go far beyond that. He would eat chicken nuggets, but only if they came from McDonald's, not if they came from Wendy's or any other restaurant. He would eat bananas, but only if they were the perfect level of ripeness and if they were sliced just right. He refused anything that was green or orange, anything "sticky" or "pointy," or anything he considered difficult to chew. As Kimberly tried to describe to the doctor all the rules she was trying to follow, she realized that doing so was impossible, because it felt like a food that had been acceptable one day was suddenly on the "No!" list. At first, Kimberly and her husband had agreed that they would not cater to Jackson's whims. However, as Jackson started losing weight, Kimberly found herself making repeated trips to McDonald's to try to make sure that there was some protein in his diet. When the doctor asked Jackson why he wouldn't eat orange slices, he simply responded: "I don't like them. They're orange and sticky."

Although Jackson may seem like a typical "picky eater," his behaviors have become a medical concern because of his significant weight loss. In addition, his rigid rules are distressing to his parents and create conflict in their relationship with Jackson. Repeated conflict over food reinforces that eating is challenging rather than that eating is healthy and can be a source of pleasure.

Obesity

Unlike both eating disorders and feeding disorders, obesity is diagnosed on the basis of body weight in relation to height without regard to behaviors, cognitions, or emotions. For adults (ages 19 years and up), obesity has been defined as having a BMI ≥ 30 kg/m^2, with a BMI ≥ 40 kg/m^2 representing severe or morbid obesity (http://www.cdc.gov/obesity/adult/defining.html). For children and adolescents (aged 2–19 years), obesity is defined as having a BMI at or above the 95th percentile for the individual's age and sex (http://www.cdc.gov/obesity/childhood/basics.html), that is, being in the top 5 in BMI out of 100 children of the same age and sex. According to these definitions, in 2011–2012, 34.9% of (or 78.6 million) U.S. adults and 16.9% of (or 2.7 million) children and adolescents were obese (Ogden, Carroll, Kit, & Flegal, 2014). Body weights in the United States and worldwide increased steadily over the 20th century, leading to an increasing number of individuals whose weights fell in the obese range. This trend appears to have abated over the past decade: The most recent study of the U.S. population found no significant increase in obesity from 2003–2004 to 2011–2012 (Ogden et al., 2014). However, patterns within specific subgroups diverged from this overall trend. Specifically, the percentage of children 2–5 years old who were obese decreased significantly from 2003–2004 to 2011–2012, while the percentage of women 60 years old or older who were obese increased significantly in the same period. It is unclear what accounts for these patterns.

The use of BMI to define excess weight does not take into consideration body composition (the extent to which weight is due to fat, muscle, bone, etc.). Some elite athletes in fields that require very high muscle mass would be identified as obese according to this approach. As an alternative to relying solely on BMI, waist circumference has been used as a measure of abdominal fat among individuals with a BMI between 30 and 35 kg/m^2. Circumferences of 102 cm (40 in.) for men and 88 cm (35 in.) for women have been identified as thresholds above which the risk of disease increases (Expert Panel on the Identification, Evaluation, and Treatment of Overweight in Adults, 1998).

Obesity has been identified as a "disease" by the American Medical Association and the World Health Organization. This decision reflects evidence of associations between obesity and elevated risks of heart disease, stroke, type 2 diabetes, and some forms of cancer in adults (http://www.cdc.gov/obesity/adult/defining.html), as well as of elevated blood pressure, type 2 diabetes, asthma, and sleep apnea in children (http://www.cdc.gov/obesity/childhood/basics.html). Because of the proportion of the U.S. population affected by obesity and the associated health consequences, a great deal of research has been devoted to understanding factors that contribute to excess weight; some of this research is reviewed in Chapter 5.

Prior to publication of the *DSM-5*, there was considerable attention to whether or not obesity should be included as a mental disorder, and if so, whether it might represent a form of addiction or an eating disorder (Marcus & Wildes, 2009). The *DSM-5* eating disorders workgroup advocated for including obesity in their deliberations because of the members' expertise on dysregulated ingestive behavior and weight. Indeed, many eating disorders researchers also study obesity.

Is Obesity a Food Addiction?

Some researchers have argued that obesity shares features with drug addiction, only with food taking the place of drugs. Support for this position comes from evidence that brain regions showing increased activity after drug use respond similarly to food consumption (Avena, Bocarsly, Hoebel, & Gold, 2011; Volkow, Wang, Tomasi, & Baler, 2013). Some obese individuals describe themselves as feeling addicted to food (Davis et al., 2011). Further, in a subset of obese individuals who describe themselves as addicted to food, self-reported craving in response to food cues (such as advertisements on TV) predicts increased food consumption even when these individuals are not hungry (Veilleux & Skinner, 2015).

Overeaters Anonymous, a self-help group based on the tenets of Alcoholics Anonymous, uses food addiction as the explanatory model for its members' behavior. The key challenge of this approach is that people can easily survive without drugs and alcohol; thus a model in which abstinence provides freedom from addiction is potentially viable for the treatment of drug and alcohol abuse but not for problems with food. Overeaters Anonymous has therefore adapted the approach of focusing on specific aspects of food that make it "addictive," including the presence of refined sugar and flour. Simple carbohydrates found in those substances have higher caloric density and influence blood glucose more rapidly, stimulating insulin release, than do complex carbohydrates. Overconsumption of refined sugar and flour can therefore more easily contribute to weight gain.

From an evolutionary perspective, it makes sense that people would be drawn to overconsume foods that facilitate weight gain. Our bodies evolved when food was scarce. Thus hardwired biological mechanisms that drove our ancestors to prefer and consume calorically dense food would have increased the likelihood of survival and reproduction. Reflecting this drive, food is a natural reinforcer—it is inherently rewarding and activates regions of the brain that help us to seek out and obtain rewards. However, there are also negative feedback loops within this system such that the reward value of food tends to diminish during food consumption. A simple example is the extent to which the first bite of a chocolate bar is more delicious and rewarding than the fifth bite. Advocates of this evolutionary perspective posit that obese individuals experience less reward from food and increase their food consumption in pursuit of a sense of satisfaction that they cannot get with normal amounts of food (Stice, Spoor, Bohon, & Small, 2008). This behavior is akin to a pattern of drug tolerance in which larger and larger quantities are needed to get the desired effect.

Despite the compelling nature of such models and supporting data from animal and human studies, all findings indicate that the addiction model fits only a relatively small proportion of obese individuals. Indeed, most people who meet criteria for obesity do not report pathological eating responses to food. Instead, it appears that a relatively modest elevation in food intake relative to energy needs contributes to obesity in a majority of those who are obese. For example, consuming 100 additional calories above caloric needs (e.g., 10 more potato chips than needed) every day for one year would produce a 10-pound weight gain. Considering all of the societal factors encouraging excessive food intake (e.g., Taco Bell's FourthMeal campaign), a major question is whether current trends in obesity should be attributed to individual behavior or to the environment, or at least to the combination of the two. An argument can be made that the same tendencies that helped humans survive in

a historical environment in which food was scarce (discussed above) have become detrimental in our modern environment in which food is all too plentiful.

Is Obesity an Eating Disorder?

As noted above, the *DSM-5* eating disorders workgroup was assigned the task of determining whether or not obesity should be defined as an eating disorder. Because eating disorders are forms of mental disorders (see Chapter 1), the first question the group needed to address was whether obesity represented a mental disorder. Marcus and Wildes (2009) provided a comprehensive review of deliberations on this topic, focusing on two key issues in defining a mental disorder: First, does obesity cause harm? Second, does obesity result from a dysfunction of mental processes—that is, does it reflect problems in how individuals think, feel, or behave?

With regard to harm, Marcus and Wildes (2009) concluded that obesity does confer harm on the individuals it affects, by increasing the risk of physical diseases and distress. They noted the link between obesity and increased risk for cardiovascular disease, diabetes, hypertension, sleep apnea, and colon, breast, and pancreatic cancer, among other forms of cancer. In addition, obesity has been linked to increased risk for psychiatric disorders such as mood, anxiety, and personality disorders. Finally, obesity increases the risk of premature death.

With regard to whether obesity emerges from mental dysfunction, Marcus and Wildes (2009) concluded that it may be linked to dysregulated food intake in a subset of obese individuals. In particular, individuals who suffer from BED are more likely to be overweight and obese. However, BED is already included in the *DSM-5* as an eating disorder. Moreover, multiple factors give rise to obesity, most of which do not represent aberrant thoughts, feelings, or behaviors. Marcus and Wildes concluded that while certain subtypes of obesity may be linked to mental dysfunction, most obese individuals are mentally healthy.

The following case study exemplifies how obesity does not, in and of itself, represent a mental illness, even though it can be associated with considerable health risks and distress.

Case Study: Joanne

Joanne, a 10-year old girl in the fifth grade, had always been heavier than her friends as a little girl. On hot summer days she preferred staying inside watching TV with her mom, snacking on Wheat Thins and Cheez Whiz, over running around outside. Her mom enjoyed her company too, and they would go on mother–daughter "dates" to the movies, where they would share a large buttered popcorn, Cherry Slushies, and a box of Mike and Ike candies. After the movie, they would go to the food court in the mall and, after walking the court and considering all of their options, come back to Panda Express and order the orange chicken, fried rice, and egg roll combo, washing it down with a large Mountain Dew. Joanne always liked the Mountain Dew commercials because they made "doing the Dew" seem exciting—like she was climbing a mountain, skateboarding, or something else daring and adventurous rather than sitting in the air-conditioned food court of the local mall with her mom.

In contrast to her imagined love for mountain climbing, Joanne did not enjoy exercising, and she particularly hated everything about physical education class. She hated having to

change clothes in the locker room in front of the other girls, because she felt self-conscious about the extra roll of fat around her middle that other girls didn't have. She hated the way her thighs chafed from rubbing together when she ran. She hated how out of breath she got when she ran. Worst of all, she hated when the class had to try to climb a rope and ring a bell at the top. She would grasp onto the end of the rope, straining to lift herself up, but would ultimately just swing there until her arms gave out or her physical education teacher blew the whistle to signal that her turn was over. She could tell that the PE teacher felt frustrated by her lack of progress, even though he never actually said anything other than: "Keep trying. Don't give up." She also hated being chosen nearly last when the class played team sports like kickball or dodgeball.

One time her friend Sara tried to convince a team captain to pick Joanne earlier because Joanne was having a birthday party and had invited Sara for a sleepover. But the captain refused, on the basis that Joanne made too easy a dodgeball target because she was so big and couldn't move fast enough. Early in that game, to escape proving the team captain right, Joanne faked a sprained ankle and hobbled to the nurse's office for a bandage and ice that she didn't need. Afterward, Joanne asked her mom for a note to get out of PE because of her injury. Joanne's mom dutifully wrote the note, and days out of PE stretched to almost two weeks, when her mom commented that if Joanne wasn't able to move around comfortably, then the sleepover might not be a good idea. Joanne faced a difficult dilemma: Should she allow herself to make a "full recovery" and return to the daily humiliation that was PE class, or should she just forgo the sleepover? In the end, she decided to have the sleepover, because she knew she couldn't get out of PE forever—but she was looking forward to the day when she wouldn't have to take PE ever again.

Joanne's case illustrates what happens to many children who get caught in a vicious cycle of consuming more calories and getting less exercise than their bodies need. She does not suffer from a mental disorder, because her actions are under her control—she eats as she does because she wants to, and she avoids exercise because she hates it. These behaviors are not optimally healthy, but people are allowed to engage in suboptimal behaviors without being labeled with a mental disorder. For example, people routinely begin crossing an intersection when the red "Don't Walk" sign starts flashing. Though this behavior increases the risk of getting hit by a car, it does not (by itself) indicate a mental disorder. Moreover, for a set of thoughts, feelings, or behaviors to represent a mental disorder, they must be associated with present distress or impairment. Arguably, Joanne is distressed by her inability to do as well in PE class as other children and by her body's appearance. However, it is normal for social comparison with peers to lead to feelings of inadequacy. While Joanne feels self-conscious about her roll of fat, another girl likely feels uneasy about her flat chest, and still another feels awkward because her breasts are too large. Similarly, Joanne's friends who struggle with math likely dread having to go up to the board to solve a problem in front of the class. This does not mean that they have a mental disorder.

Obesity Is a Physical Condition with Heterogeneous Causes

Ultimately, obesity is a physical condition. It may be a sign of an eating disorder, as it is frequently observed in individuals suffering from BED. However, making obesity an eating disorder unto itself would be comparable to making emaciation an eating disorder without considering the

various causes that could lead to that physical state. Certainly, an individual who is unable to sustain a minimally healthy weight while undergoing chemotherapy treatment for cancer would not be diagnosed with an eating disorder. Similarly, an individual who is unable to maintain a healthy weight because of hyperthyroidism (which leads to weight loss) or hypothyroidism (which leads to weight gain) would not be considered to have an eating disorder. Too many factors contribute to obesity to equate the physical condition with a mental disorder.

Beyond the lack of evidence that obesity is linked to a mental dysfunction (Marcus & Wildes, 2009), the current prevalence of obesity in the United States suggests that it represents an increasingly normative consequence of widespread lifestyle changes. As a nation (and a world), we live in a period of mass production and marketing of inexpensive and highly palatable foods. Food used to represent a much higher proportion of a household's expenses (Pomeranz, 2000). As a consequence, it was not used as a form of entertainment, as it is today. Along with the increased access and encouragement to consume large quantities of food we now experience, lives are far less physically demanding today than they once were. Machines have taken over many forms of physical labor once required to function at home and work. For example, a shift from agricultural labor without machines to modern agricultural conditions reduces daily caloric needs by 33–50% per hour of work (Pomeranz, 2000). Recalling the earlier example of the effect of consuming just 100 additional calories in relation to physical needs each day for a year, one can see how this dramatic decrease in physical needs contributes to the increased prevalence of obesity.

Health at Every Size

Although clear evidence links obesity to increased risk of medical problems, some researchers have argued that the changes in lifestyle associated with obesity, rather than obesity itself, are to blame for health risks. Several psychosocial factors related to stigmatization of obesity appear to exaggerate these risks (a topic discussed more in Chapter 5).

The Health at Every Size movement has emerged to emphasize that individuals can follow a healthy lifestyle at a range of body weights and that individual decisions about nutritional intake and activity level predict health more strongly than does a number on a scale. For example, although metabolic abnormalities are more common among the overweight (47.1%) and obese (67.7%) than among those within a healthy weight range (25.6%) (Wildman et al., 2008), this still means that a third of obese individuals are metabolically healthy, while a quarter of individuals within a healthy weight range have metabolic risk factors for disease. Wildman et al. (2008) reported that among people in the healthy weight group, a larger waist circumference, older age, and less physical activity contribute to increased risk. Similar predictors emerged among those in the overweight/obese weight group. These results support the idea that age and lifestyle factors, in addition to BMI and waist circumference, contribute to health status.

Conclusion

Feeding disorders and obesity are related to eating disorders. However, they are not eating disorders in and of themselves. Although feeding disorders are mental disorders, they lack

the body image disturbance and distress that characterize eating disorders. In addition, they affect a different segment of the population from that typically seen for eating disorders (discussed in more detail in Chapter 3). Obesity is a physical state, not a mental disorder. Although obesity is associated with the presence of BED, a diagnosis of BED captures the mental disorder that is present in these individuals. Moreover, many individuals who are obese do not engage in behaviors that are drastically different from those of their healthy-weight peers. Instead, they may have genetic makeups that make them particularly susceptible to living in an environment that promotes obesity.

3

Who Suffers from Eating Disorders?

Chapter 1 presented five case studies of individuals with eating disorders. Emily suffered from AN, Jean suffered from BN, Jamie suffered from BED, Valerie suffered from purging disorder, and George suffered from night eating syndrome. In reading the case studies, you might have formed a mental image of these people. You might have imagined that everyone was White, even though no specific information was given concerning their race or ethnicity. You might also have imagined that aside from George, everyone was female, even though no specific information concerning gender was given for Jamie. If you did automatically assign race and gender to these individuals in this fashion, it likely related to what is generally believed concerning who suffers from eating disorders—White girls. Beyond representing a general stereotype, this idea has been presented as fact by experts in eating disorders (e.g., Bruch, 1966; Crisp, Palmer, & Kalucy, 1976; Garfinkel & Garner, 1982).

But is the stereotype correct? This chapter examines who actually suffers from eating disorders using data from clinical samples, population-based epidemiological studies, and case studies.

Eating Disorders in Treatment Samples

The belief that eating disorders predominantly affect White females matches observations from treatment-seeking samples. In 2010, less than 65% of the U.S. population was White and of non-Hispanic origin, and approximately half were female. In contrast with these national statistics, the demographic characteristics of eating disorder patients in the United States have shown a preponderance of White women. Many controlled treatment studies of eating disorders include only women (e.g., Carter et al., 2011; Fichter et al., 2012; Ter Huurne, Postel, de Haan, & Dejong, 2013; Walsh et al., 1997). However, even in studies recruiting both genders, more than 90% of participants have been female (Goldstein, Wilson, Thompson, Potvin, & Rampey, 1995; Gowers et al., 2010; le Grange, Crosby, Rathouz, &

Leventhal, 2007; Lock et al. 2010; Schmidt et al., 2007). This pattern contributes to estimates of a 10:1 ratio of women to men affected by AN and BN in the *DSM-5* (American Psychiatric Association, 2013).

No treatment study restricts admission on the basis of racial or ethnic background. Indeed, before funding a treatment study, the National Institutes of Health require a specific plan for recruiting ethnic and racial minority participants to ensure a sample that is representative of the U.S. population. Nonetheless, in several independent studies of treatment-seeking individuals, over 90% of participants have been White (Carter et al., 2011; Goldstein et al., 1995; Schmidt et al., 2007; Strober, Freeman, & Morrell, 1997; Sullivan, Bulik, & Kendler, 1998). Some of this seemingly disproportionate representation appears to be associated with the ethnic diversity of the locations in which studies have been conducted (Lock et al., 2010). More diverse samples have emerged from locations with more diverse populations. For example, in a recent multisite treatment trial for AN approximately 25% of participants were from ethnic or racial minority groups, with a more diverse sample recruited in the San Francisco Bay area than in the greater Chicago area (Lock et al., 2010). A recent study of BN included 36% minority participants recruited from the Chicago area (le Grange et al., 2007). Even so, these samples were less diverse than the populations from which participants were drawn.

These data indicate that patients with eating disorders are more likely to be female and White than the general population, suggesting that something about being female and White may increase risk for developing an eating disorder. However, these data best represent *patients* with eating disorders. For someone to become a patient, he or she must seek treatment, and a healthcare professional must recognize that the person has an eating disorder. Thus there are two possible points at which individuals who suffer from eating disorders may not receive treatment for their disorder. Because both gender and ethnicity may influence who seeks treatment for an eating disorder and in whom an eating disorder is recognized, community-based samples can provide less biased estimates of gender and ethnic minority representation among individuals who actually suffer from eating disorders.

Eating Disorders in Community-Based Samples

The epidemiology of eating disorders addresses the central question of this chapter by evaluating who in the population has these disorders. **Lifetime prevalence** of a disorder is the statistic representing the proportion (or percentage) of the population who ever had that disorder in their lifetime. The largest and most recent epidemiological study of mental illness in U.S. adults is the National Comorbidity Survey—Replication (Kessler et al., 2004). This study surveyed 9,282 English-speaking adults 18 years and older who were sampled to be representative of the general U.S. population with regard to gender, race, ethnicity, income, and education level. Of these participants, 2,980 were interviewed to examine whether they had eating disorders, including AN, BN, and BED (Hudson, Hiripi, Pope, & Kessler, 2007). The National Comorbidity Survey—Replication Adolescent Supplement extended interviews to 10,123 adolescents between the ages of 13 and 18 years, all of whom were assessed

for eating disorders (Swanson, Crow, le Grange, Swendsen, & Merikangas, 2011). Neither purging disorder nor night eating syndrome was assessed in either study. Questions about purging were asked only if participants reported engaging in binge eating, and the interviews included no questions about night eating. Preliminary information on the epidemiology of these OSFEDs is reviewed later in this chapter.

In adult women, lifetime prevalence was 0.9% for AN, 1.5% for BN, and 3.5% for BED. In adult men, lifetime prevalence was 0.3% for AN, 0.5% for BN, and 2.0% for BED (Hudson et al., 2007). In adolescent girls, lifetime prevalence was 0.3% for AN, 1.3% for BN, and 2.3% for BED. In adolescent boys, lifetime prevalence was 0.3% for AN, 0.5% for BN, and 0.8% for BED (Swanson et al., 2011). Although all three eating disorders were significantly more common in women than in men, and both BN and BED were more common in adolescent girls than in adolescent boys, the ratios are far less skewed than those from treatment studies. These results suggest that males with eating disorders are underrepresented in treatment.

Case Study: Jamie

Jamie was embarrassed about beginning treatment for an eating disorder, because he had always been such a "guy's guy." He had been an athlete throughout school, and he worked in a male-dominated business environment. When a counselor raised the possibility of Jamie's entering group treatment, he immediately rejected it. He felt that he would stand out like a sore thumb as the only man in the group. The counselor told Jamie that the group included both men and women but acknowledged that there were far more women. Jamie repeated that he was interested in the possibility of using medication to help him adhere to a weight loss program. He felt it would be hard to explain to his male friends where he was going every Monday night if he entered group treatment. In addition, attending the Monday night group meant missing Monday night football. As far as Jamie was concerned, if the group was really meant to include men, it would not meet on Monday nights during football season.

Eating Disorders and Men

As captured by Jamie's case, men with an eating disorder may feel marginalized with respect to their gender ("real" men don't suffer from eating disorders) and with respect to others who suffer from eating disorders ("real" eating disorders don't happen in men). These feelings may reduce men's likelihood of seeking treatment and participating in studies of eating disorders, even when studies recruit both women and men. Reflecting this self-selection bias, men were significantly less likely than women to participate across each wave of an epidemiological study of eating disorders (Keel, Heatherton, Dorer, Joiner, & Zalta, 2006). If eating disorders occur less frequently in men and men are less likely to seek treatment and to participate in studies of eating disorders, then a self-perpetuating cycle develops in which men are either excluded from or severely underrepresented in eating disorders research. For example, in the January 2016 issue of the *International Journal of Eating Disorders,* eight reports were restricted to female patients, men made up 2% of patients in one treatment study,

and a meta-analysis of internet-based interventions included 17 studies restricted to female patients compared to only 3 studies including both male and female patients.

The underrepresentation of men in the study of eating disorders raises an important question: Do men have problems with eating and body image that go unrecognized because the problems do not match the patterns exhibited by women? This question essentially reopens the question addressed in the first chapter, "What are eating disorders?" Thus far this chapter has reported on eating disorders in men as they have been defined in women. This assumes that eating disorders are expressed in men as they are in women.

Muscle Dysmorphia—A Predominantly Male Eating Disorder?

There has been speculation that men may be at risk for a type of eating disorder not seen in women. In the field of eating disorders, this disorder has been described as the "Adonis complex" (Anderson, 1984) or **reverse anorexia** (Pope, Katz, & Hudson, 1993) and is officially recognized as a form of body dysmorphic disorder in the *DSM-5*—**muscle dysmorphia** (American Psychiatric Association, 2013). Instead of viewing their bodies as much larger than they really are (as can happen in AN), men with muscle dysmorphia view their bodies as puny despite their efforts and success at bodybuilding (American Psychiatric Association, 2013; Pope et al., 1993). This distorted perception contributes to extreme efforts to increase lean-muscle mass and overall body size. Such extreme measures include excessive exercise, dietary manipulations that include extremely high protein intake, and use of anabolic steroids. To the extent that this clinical pattern involves altered eating patterns, the use of extreme weight control behaviors, and body image disturbance, many researchers have argued that it represents an eating disorder (Andersen, 1984; McCabe & Ricciardelli, 2001; Murray, Rieger, Hildebrandt, et al., 2012; Pope et al., 1993), albeit one rarely seen in women.

Just as there is a cultural idealization of thinness for women, there is a cultural idealization of muscularity for men. Pope, Olivardia, Gruber, and Borowiecki (1999) examined changes over time in the proportions of male action figures, including G.I. Joe and toys representing the *Star Wars* characters Luke Skywalker and Han Solo (see Figure 3.1). In recent decades, the shoulder-to-waist ratio has increased dramatically. Current action figures represent a distortion of bodily proportions for men on par with the distortions presented by Barbie dolls for women. According to Pope et al., the large, bulging, well-defined muscles of these action figures are obtainable in real life only with professional weight training and the use of anabolic steroids. Changing body ideals for men are also evident in the physiques of actors portraying superheroes such as Batman and Superman.

Males who want to lose weight are more likely to be overweight than are women who feel the same way, resulting in a gender difference in who wants to lose weight (Calzo et al., 2012; Keel, Baxter, Heatherton, & Joiner, 2007). However, studies demonstrating this gender difference (Calzo et al., 2012; Cohn et al., 1987; Fallon & Rozin, 1985) find that underweight boys and men report body dissatisfaction due to the desire to be bigger. Thus the characterization of women as having more body dissatisfaction than men is more appropriately framed as women having greater dissatisfaction with being too large.

FIGURE 3.1 Luke Skywalker and Han Solo, 1978 (left); Luke Skywalker and Han Solo, 1998 (right). Source: Pope, H. G., Olivardia, R., Gruber, A. and Borowiecki, J. (1999), Evolving ideals of male body image as seen through action toys. *International Journal of Eating Disorders*, 26, 65–72, p. 70. © 1999 by John Wiley & Sons, Inc. Reprinted with permission by John Wiley & Sons, Inc.

Mirroring this difference, adolescent boys are significantly more likely to engage in anabolic steroid use and related behaviors to increase muscle mass than are adolescent girls (Bahrke, Yesalis, & Brower, 1998; Drewnowski, Kurth, & Krahn, 1995; Eaton et al., 2006; Eisenberg, Wall, & Neumark-Sztainer, 2012; Field et al., 2005; Irving, Wall, Story, & Neumark-Sztainer, 2002; Johnston, O'Malley, Bachman, & Schulenberg, 2005; Neumark-Sztainer, Story, Falkner, Beuhring, & Resnick, 1999). Across studies, rates of anabolic steroid use range from 1.3% to 12% among adolescent boys and 1.0% to 5.7% among adolescent girls. Given the effects of anabolic steroids on liver, heart, and reproductive function, these rates are alarmingly high. Recent research suggests that anabolic steroid use may be becoming less common among adolescents but may be more likely to occur in younger than older adolescents (van den Berg, Neumark-Sztainer, Cafri, & Wall, 2007), with approximately 5% of 13-year-olds reporting use of steroids to promote weight gain, compared with approximately 1% of 20-year-olds. This shift may reflect developmental changes in body size.

As girls enter puberty, their percentage of body fat increases; this body fat is distributed in the development of breasts and increases in the size of hips and thighs. Thus puberty takes girls further from the feminine ideal portrayed in popular media (see Chapter 4). Conversely, as boys enter puberty, their percentage of lean-muscle mass increases. Both boys and girls become taller during puberty, but sexual differences in height emerge, causing boys to be taller than girls. Further, boys experience an increase in the width of their shoulders relative to their hips. Thus puberty brings boys closer to the masculine ideal portrayed in popular media.

However, the masculine ideal within a weight-obsessed culture may provide men with two potentially opposing ideals to trigger extreme concerns with their weight and shape: one focused on leanness, which might push some men toward AN, and one focused on muscularity, which might push others toward muscle dysmorphia (Field et al., 2014). In a study

comparing 24 men with AN, 21 men with muscle dysmorphia, and 15 healthy men who used the gym regularly, Murray et al. (2012) found that despite having different body ideals, men with AN and those with muscle dysmorphia had similar levels of body dissatisfaction, weight/shape preoccupation, disordered eating, and exercise behavior. Furthermore, severity of muscle dysmorphia was significantly associated with severity of disordered eating.

More research is required to understand the relationship between eating disorders and muscle dysmorphia. Currently, muscle dysmorphia is included as a specifier within the *DSM-5* diagnostic criteria for body dysmorphic disorder rather than as a form of eating disorder (American Psychiatric Association, 2013). In contrast, the *DSM-5* considers concern about being fat in an individual with AN a symptom of the eating disorder and not a form of body dysmorphic disorder.

Summary of Eating Disorders in Men

Eating disorders are more common in women than men. This has been confirmed in population-based epidemiological studies, treatment studies, and school-based investigations of body dissatisfaction, disordered eating, and full-threshold eating disorders. That said, eating disorders occur more often in men than most people realize. Men make up at least 10% of individuals diagnosed with AN or BN, and approximately 36% of BED occurs in men (Hudson et al., 2007). Comparisons with estimates of eating disorders in population-based epidemiological studies show that men have been underrepresented in eating disorders research.

Part of this underrepresentation reflects a self-selection bias in which men do not volunteer to participate in studies (and perhaps treatment) of a disorder that has been strongly associated with females. Another part may reflect that eating disorders have been conceptualized largely based on their appearance in women. As a result, the features that have come to define eating disorders (e.g., fear of gaining weight or becoming fat) may produce greater gender disparity in eating disorder prevalence than would exist if a more inclusive definition of eating pathology were used.

Moreover, the American Psychiatric Association identifies a syndrome characterized by preoccupation with being too fat and by extreme behaviors to address this concern as an eating disorder, but it identifies a preoccupation with being too small and extreme behaviors to address this concern as not an eating disorder. This approach to defining what an eating disorder is (and is not) may cause apparent gender differences in who suffers from eating disorders. Further work is needed on clinical patterns that may characterize eating disorders seen more often in men than in women: an intense desire to attain a larger, more muscular physique, which has been linked to an undue influence of body weight or shape on self-evaluation, and extreme, unhealthy eating and weight control behaviors aimed at reaching this masculine ideal.

Eating Disorders and Race/Ethnicity

Research supports the idea that stereotypes of who suffers from an eating disorder may reduce recognition of eating disorders in those who do not match the stereotype, even when there are no differences in symptoms. In a study of college students, Gordon, Perez, and

Joiner (2002) found that race significantly influenced the likelihood that an eating disorder will be recognized. Participants in the study were given a written summary of a 16-year-old girl's eating and other activities over a five-day period, which were indicative of an eating disorder. When "Mary" was depicted as White, 93% of participants concluded that she had an eating disorder. When she was depicted as either African American or Hispanic, only 79% of participants recognized her eating disorder. This effect did not depend on the race of the participant. If healthcare providers, like college students, are less likely to appreciate the presence of an eating disorder when it occurs in a non-White or Hispanic woman, this could contribute to a difference in referral for treatment of the disorder.

Among 5,787 college students who participated in the National Eating Disorders Screen and subsequently met with a counselor, those who were Latino or Native American were significantly less likely to receive a referral for eating disorders treatment than were White students, and these differences were not explained by differences in symptom severity (Becker, Franko, Speck, & Herzog, 2003). In addition, among students in the same study who were concerned about their eating, doctors were approximately half as likely to ask ethnic minority students about their eating as they were White students. Given these data, it seems clear that treatment samples may better reflect who we expect to suffer from eating disorders than who actually suffers from those disorders.

Because ethnic minority groups cannot be assumed to be alike on eating pathology the following sections examine evidence of eating disorders from community-based epidemiological studies separately for three ethnic minority groups: African American/Black, Asian, and Hispanic–Latino. Differences among people with specific cultural backgrounds within these groups are examined where possible, as well as information on whether acculturation to dominant White culture is linked to eating disorder prevalence in these groups.

Eating Disorders in African American or Black Samples

Cases of AN and BN have been recognized in Black individuals for some time (Dolan & Ford, 1991; Gray, Ford, & Kelly, 1987; Johnson, Lewis, Love, Lewis, & Stuckey, 1984; Pate, Pumariega, Hester, & Garner, 1992). Indeed, Lawlor, Burket, and Hodgin (1987) described a Black male adolescent hospitalized for an eating disorder over 25 years ago. However, early cases were recognized with the caveat that they were "rare." It was generally believed that Black individuals were "protected" from developing eating disorders because the ideal of beauty was heavier and more voluptuous for Black women than it was for White women (Williamson, 1998). In support of this belief, several studies found that African American women reported less body dissatisfaction, disordered eating, and were less like likely to suffer from BN in comparison to White American women (Abrams, Allen, & Gray, 1993; Chandler, Abood, Lee, Cleveland, & Daly, 1994; Edwards-Hewitt & Gray, 1993; Gray et al., 1987; Rhea, 1999; E. F. Rosen et al., 1991). A study of eating disorders in 985 White and 1,061 Black women found that AN affected 1.5% of White women but no Black women (Striegel-Moore et al., 2003). Bulimia nervosa was also more common in White than in Black women, affecting 2.3% of the former and 0.4% of the latter. Binge-eating disorder showed the least dramatic difference, affecting 2.7% of White women and 1.4% of Black women.

More recent data, however, have indicated that Black and African American individuals are not protected from the development of eating disorders. In the National Survey of American Life, Taylor, Caldwell, Baser, Faison, and Jackson (2007) interviewed 5,191 Black adults: 3,570 African Americans and 1,621 Black Americans of Caribbean origin. Lifetime prevalence of eating disorders in women was 0.1% for AN, 1.9% for BN, and 2.4% for BED. In men, lifetime prevalence was 0.2% for AN, 1.0% for BN, and 0.8% for BED. No cases of AN were found among Caribbean Blacks; however, there were no differences between African American and Black Caribbean individuals in risk for BN or BED.

Marques et al. (2010) pooled data from the National Comorbidity Survey—Replication and the National Survey of American Life to evaluate whether race was associated with prevalence of eating disorders. Overall, African Americans were significantly *more* likely to have a lifetime history of BN than were non-Hispanic White participants. When the data were broken down by gender, no significant differences for lifetime prevalence of AN, BN, or BED were found between White and Black women or between White and Black men. In the National Comorbidity Survey—Replication Adolescent Supplement, Swanson and colleagues (2011) found no significant differences between non-Hispanic Black and White teenagers in prevalence of AN, BN, or BED.

Marques et al. (2011) found that although African American adults were not less likely than White adults to have suffered from an eating disorder, they were less likely to have ever sought mental health treatment. Combined with the lower likelihood of African Americans being asked about problems with eating or being referred for eating disorder treatment (Becker et al., 2003), a decreased likelihood of seeking mental health treatment would reduce representation of African Americans among individuals in treatment for an eating disorder.

Eating Disorders in Asian Samples

The National Latino and Asian American Study evaluated eating disorder prevalence in 2,095 Asian American adults, including participants of Chinese, Filipino, Vietnamese, and "other" Asian (Japanese, Korean, and Asian Indian) descent (Nicdao, Hong, & Takeuchi, 2007). In women, lifetime prevalence for eating disorders was 0.1% for AN, 1.4% for BN, and 2.7% for BED. In men, lifetime prevalence was 0.1% for AN, 0.7% for BN, and 1.4% for BED. Eating disorder prevalence did not differ among Asian subgroups. In addition, there was no association between acculturation to U.S. culture and eating disorder prevalence in this sample.

Comparing findings for non-Hispanic White and Asian American participants from the National Comorbidity Survey—Replication and the National Latino and Asian American Study, respectively, Marques et al. (2011) found no significant difference between the two groups for lifetime prevalence of AN, BN, or BED. However, Asian individuals with a lifetime eating disorder were significantly less likely to have ever sought mental health treatment than were non-Hispanic White participants.

Several studies have failed to find a significant association between acculturation to Western culture and body dissatisfaction or disordered eating among Asian American women (Gowen, Hayward, Killen, Robinson, & Taylor, 1999; Haudek, Rorty, & Henker, 1999; Jackson, Keel, & Lee, 2006; Marques et al., 2011; Yoshimura, 1995). These results

suggest that cultural factors that contribute to eating disorders may be native to some Asian cultures. Rieger, Touyz, Swain, and Beumont (2001) have argued that traditional values and practices in some Asian cultures idealize thinness. The virtues of fasting to the point of emaciation are included in the Taoist text *Sandong Zhunang* (Rieger et al., 2001). Asian cultures may emphasize the importance of gaining approval from family members and friends (Mukai, Kambara, & Sasaki, 1998; Mukai & McCloskey, 1996), who may encourage dieting even among underweight girls (Mukai, Crago, & Shisslak, 1994).

Case Study: Jean

Jean (see Chapter 1 for description of BN in this case) dreaded going home for vacation, because she knew her mother would complain about two things—Jean's living with her boyfriend before marriage and Jean's weight. What was more, Jean's mother would tie the two complaints together into one theme. She would warn Jean that if she did not lose weight, she would never receive a wedding proposal. Jean's mother was born in Korea and had met Jean's father when he was in Korea visiting his grandparents. Despite spending most of her adult life in the United States, Jean's mother held traditional values for her daughter that included the importance of Jean's attracting a good husband—preferably a Korean husband. To reach this goal, Jean's mother felt Jean needed to remain thin and avoid revealing her age. Most of all, she needed to break up with her boyfriend. It was clear to Jean's mother that the boyfriend did not have honorable intentions.

As Jean's case study reveals, rather than all cultural contributions to eating pathology originating in White Western culture, there may also be non-Western cultural factors that increase the risk of disordered eating and eating disorders. Jackson et al. (2006) examined disordered eating attitudes and behaviors in three groups of Korean women: women born and living in Korea (native Korean), women born in Korea who had moved to the United States (Korean immigrants), and women who were born in the United States to Korean parents who had been born in Korea (second-generation women). If U.S. culture was the primary source of risk for eating disorders, disordered eating levels should be highest for women who had spent their entire lives in the United States and lowest for those born in and living in Korea. If a conflict between traditional Korean and American cultural values, referred to as **acculturative stress**, increased risk for eating disorders, then disordered eating should be highest in Korean immigrants.

In contrast to both these hypotheses, disordered eating levels were lowest in Korean women who had been born in the United States and highest in native Korean and Korean immigrant women—that is, in women who had been born in Korea, regardless of whether they had remained there. Furthermore, there was no association between acculturation to Western culture and disordered eating levels among the Korean women living in the United States, whether they had been born there or immigrated. Thus Jean's eating disorder may emerge from factors confronting all women in the United States as well as factors unique to those from Asian backgrounds.

Eating Disorders in Hispanic–Latino Samples

A key limitation of the National Comorbidity Survey—Replication study was its requirement that all participants speak English, because not all Hispanic–Latino individuals in

the United States are English speakers. The National Latino and Asian American Study addressed this limitation by interviewing 2,554 English- and Spanish-speaking Latino adults in the United States (Alegria et al., 2007). Of these, 868 were of Mexican descent, 495 Puerto Rican, 577 Cuban, and 614 "other" Latino. In women, lifetime prevalence was 0.1% for AN, 1.9% for BN, and 2.3% for BED. Lifetime prevalence in men was 0.03% for AN, 1.3% for BN, and 1.6% for BED. Lifetime prevalence of eating disorders did not differ across the Latino subgroups. Being born in the United States, spending a higher proportion of one's life in the United States, and having a higher BMI increased risk for BN.

Marques et al. (2011) provided direct comparisons of lifetime prevalence of eating disorders for Latino participants and non-Latino White participants by pooling data from the National Latino and Asian American Study and the National Comorbidity Survey—Replication. Latino men were significantly more likely to suffer from BN over their lifetimes than were non-Latino White men. No other differences were found in eating disorder prevalence between Latino and non-Latino White adults. In the National Comorbidity Survey—Replication Adolescent Supplement, Swanson and colleagues (2011) found that Hispanic adolescents had a significantly higher lifetime prevalence of BN than did non-Hispanic White teens. Even though Latino individuals were as or more likely to suffer from an eating disorder over their lifetimes than were non-Latino White participants, they were significantly less likely to have ever sought mental health treatment (Marques et al., 2011).

Several studies have reported that acculturation to U.S. values is associated with higher levels of eating pathology among Hispanic individuals (Chamorro & Flores-Ortiz, 2000; Gowen et al., 1999; Pumariega, 1986). In particular, widespread adoption of U.S. beauty ideals has been associated with greater eating pathology (R. Lester & Petrie, 1995). Gowen et al. (1999) reported that among more acculturated Hispanic girls, 13.6% could be diagnosed with an eating disorder, compared with 0% of less acculturated Hispanic girls.

For Latino individuals, increasing representation of Latino women among female pop artists such as Selena Gomez, Demi Lovato, and Christina Aguilera may provide a bridge between ethnic identity and dominant Western ideals of beauty, fame, and wealth. Indeed, both Ms. Aguilera and Ms. Lovato have received intense public scrutiny of their weight, and Ms. Lovato has acknowledged publicly her struggles with an eating disorder.

Summary of Eating Disorders in Racial/Ethnic Minorities

The belief that White individuals are at particularly high risk for developing eating disorders appears to reflect a stereotype rather than reality. The apparent underrepresentation of ethnic or racial minorities among individuals with eating disorders seems to be related more to biases in who seeks and receives treatment for these disorders than to any protection from eating pathology afforded by ethnic or racial minority status. Women of color demonstrate the same overall risk of developing eating disorders as White women, and some men and women of color may have greater risk for developing BN than their White counterparts.

The role of acculturation or adoption of the values of the majority culture in increasing the risk of disordered eating among ethnic or racial minorities is unclear. There is some support for the view that girls who attempt to conform to dominant cultural ideals are

more likely to fall prey to eating disorders; this evidence is especially strong for Latino girls (Chamorro & Flores-Ortiz, 2000; Gowen et al., 1999; Lester & Petrie, 1995; Pumariega, 1986). However, risk factors for eating disorders may be inherent in certain cultural values of ethnic minority groups that emerge from non-Western traditions (Rieger et al., 2001). The following section examines evidence of eating disorders in non-Western cultures as an avenue to better understand not only who suffers from these illnesses but why they may do so.

Eating Disorders in Non-Western Cultures

Popular accounts of eating disorders, such as those presented in *People* or *Glamour* magazine, draw prominent connections between eating disorders and the idealization of thinness in Western culture. In addition, some scholars (e.g., Prince, 1985) have argued that AN represents a culture-bound syndrome, because some studies have suggested that AN does not occur outside of Western cultures. The impact of Western ideals on risk for eating disorders will be explored in Chapter 5. However, as described above, some non-Western cultures may promote values that increase risk of eating disorders. Understanding the effects of these cultural values could significantly affect our understanding of who suffers from eating disorders.

Anorexia Nervosa in Non-Western Cultures

Attempts to examine evidence of AN across cultures have been marked by debates concerning the definition of the illness. While there is universal agreement that AN represents a disorder marked by self-starvation, some experts (Beumont, 1988; Habermas, 1989) have argued that fear of fat (weight phobia) is a necessary motivating force behind food refusal in AN. Other experts (Banks, 1992, 1994; M. A. Katzman & Lee, 1997; S. Lee, 1995), however, have argued that weight phobia is not a core feature of the disorder. These experts have argued that culture shapes sufferers experience and understanding of their disease. Recognizing cultural diversity in how patients with AN describe their experiences, the definition of AN was changed in the *DSM-5* to permit its diagnosis in individuals who do not report a fear of gaining weight but who engage in behaviors to prevent weight gain. Because definitions of AN change with each edition of the *DSM*, differ between the *DSM* and *ICD*, and continue to be contested, this section considers evidence from studies using various definitions of AN and notes how these definitions differ from the *DSM-5* definition (presented in Chapter 1).

Numerous case reports have revealed the presence of self-starvation syndromes around the world. Cases of AN have been described in South Africa, Nigeria, Zimbabwe, Egypt, the United Arab Emirates, Iran, China, Japan, South Korea, Russia, India, Pakistan, and Malaysia (Keel & Klump, 2003). In many of these cases, there is evidence of exposure to Western culture. For example, Buchan and Gregory (1984) described AN in a Black Zimbabwean woman who had lived in England for many years as a child and recalled being ridiculed about her weight while living there. However, there also are examples of AN in individuals with no apparent exposure to Western ideals. Abou-Saleh, Younis, and Karim (1998) described AN in a woman who was a nomad from the Empty Quarter of the United Arab Emirates. This patient had no previous contact with Western culture and refused to

eat for reasons unrelated to her weight. Many other non-Western cases of AN also report no weight phobia.

Evidence of a self-starvation syndrome predominantly affecting young women existed in China prior to the recently increased influence of Western culture. In 1993, S. Lee, Ho, and Hsu suggested that Westernization and industrialization, while not necessary to produce AN, bring about certain aspects of the disorder. Thus they predicted that increasing Western influence in China would increase both the prevalence of AN in China and the occurrence of AN with weight phobia. Consistent with this prediction, S. Lee and A. M. Lee found in 2000 that weight concerns were greatest in highly Westernized Hong Kong and least prominent in the largely agricultural Hunan province, suggesting an association between Westernization and body image disturbance. Further, S. Lee (2000) and Lai (2000) both described an increasing prevalence of AN characterized by body dissatisfaction in the East. Finally, comparing patient records in Hong Kong from 1987–97 and 1998–2007, S. Lee, Ng, Kwok, and Fung (2010) found that the number of eating disorder patients had doubled in the later sample, with a significant increase in the proportion of AN patients who reported having weight phobia. Thus increased Western influence was linked to increased prevalence of AN and increased weight phobia among those suffering from AN. However, cases of AN, particularly those without weight phobia, pre-date significant Western influence in China.

Anorexia nervosa has been found in certain non-Western cultures at the same or even higher frequencies than in Western culture (Tong et al., 2013). C. K. Lee et al. (1987) found no difference in the lifetime prevalence of AN between a nationwide epidemiological study in Korea and the Epidemiological Catchment Area study in New Haven, Connecticut, or St. Louis, Missouri. Nobakht and Dezhkam (2000) reported the lifetime prevalence of AN to be 0.9% in Iranian schoolgirls, a value higher than the lifetime prevalence in the United States reported in the *DSM-5* (American Psychiatric Association, 2013). S. Lee, Chiu, and Chen (1989) reported that AN in Hong Kong occurred with an incidence (a statistic we discuss in greater detail later in this chapter) of just under 0.4 cases per 100,000 person-years, similar to incidence rates that have been reported in Western nations (e.g., Kendell, Hall, Hailey, & Babigian, 1973; Møller-Madsen & Nystrup, 1992; Theander, 1970; Willi & Grossmann, 1983). The percentage of psychiatric patients who are treated for AN has been observed to be similar in non-Western nations such as Malaysia (Buhrich, 1981; Goh, Ong, & Subramaniam, 1993) and Egypt (Okasha, 1977) and in Western nations such as Norway (Götestam, Eriksen, Heggestad, & Nielsen, 1998).

Bulimia Nervosa in Non-Western Cultures

Unlike cases of AN, for which severe emaciation is likely to gain medical attention regardless of awareness of eating disorders, cases of BN can remain hidden from medical attention. For example, Whitehouse, Cooper, Vize, Hill, and Vogel (1992) found that 50% of individuals with BN as determined by a community survey in England were unknown by their general practitioners to have the disorder, even though half of these hidden cases had obtained referrals for treatment of complications of bulimic pathology. The same study found no hidden cases of AN, supporting the idea that low weight makes AN easier to detect.

Because there is no outward sign of BN, characterizing its presence outside of a Western context has proven challenging. In several cross-cultural reviews of eating disorders (Davis & Yager, 1992; Dolan & Ford, 1991; Keel & Klump, 2003; M. N. Miller & Pumariega, 2001; Pate, Pumariega, Hester, & Garner, 1992; Tsai, 2000), references to AN far outnumbered references to BN. In a comprehensive review of eating disorders by Keel and Klump in 2003, reports of AN were found in five of five non-Western regions of the world, whereas BN was reported in only three of the five non-Western regions. Subsequent to this review, one case of BN was reported in Tanzania, Africa (Eddy, Hennessey, & Thompson-Brenner, 2007), and another was described in New Delhi, India (Mandal, Arumuganathan, Sagar, & Srivasta, 2013). A 2012 survey of 66 psychiatrists practicing in Bangalore, India, found reports of 12 patients who had been treated for BN (Chandra, Abbas, & Palmer, 2012). Thus, like AN, cases of BN now have been reported in all non-Western regions of the world. In all reports, including those reviewed by Keel and Klump and reports published since that review, individuals with BN uniformly expressed weight concerns, and no studies identified BN in an individual who lacked exposure to Western ideals.

As with the prevalence of AN, degree of Westernization appears to account for a good deal of the variance in BN prevalence estimates across non-Western regions of the world (Keel & Klump, 2003). In comparing patient records in Hong Kong from 1987–97 and 1998–2007, S. Lee et al. (2010) found a significant increase in the number of patients seeking treatment for BN in the later, more Westernized sample. Examining findings from studies published in the 21st century, lifetime prevalence of BN in Wuhan, China, was comparable to estimates reported in Finland (Tong et al., 2013), and prevalence estimates for BN in Iran, Japan, and the United States are similar to each other (Keel & Klump, 2003).

Binge-Eating Disorder in Non-Western Cultures

Cases of BED or subthreshold BED have been reported in China (Chen & Tao, 2010; Tong et al., 2013), Malaysia (ZamZam, Thambu, Midin, Omar, & Kaur, 2009), India (Chandra et al., 2012), Jordan (Mousa, Al-Domi, Mashal, & Jibril, 2010), Iran (Garrusi & Baneshi, 2012), Egypt (Senna, Ahmad, & Fathi, 2013), and Tunisia (Ketata, Aloulou, Charfi, Abid, & Amami, 2009), covering all five non-Western regions originally reviewed by Keel and Klump (2003). These reports are recent, ranging from 2009 to 2013, suggesting that cases may have emerged following exposure to Western influences.

Summary of Eating Disorders in in Non-Western Cultures

It is clear that AN is not restricted to Western cultures any more than it is restricted to White women in the United States. Indeed, studies suggest that it is as common in some non-Western cultures as in the West. Although Westernization appears to have contributed to increased rates of AN, exposure to Western ideals is not necessary for development of AN.

BN and BED also have been observed in non-Western cultures. However, evidence for these disorders in non-Western cultures is more sparse than it is for AN. Western culture may be necessary for the emergence of BN and BED given that there is no evidence of these disorders in individuals with no exposure to Western culture. In addition to promoting a

thin ideal for beauty (discussed more in Chapter 5), exposure to Western ideals may be accompanied by other cultural shifts that are more crucial for the emergence of BN and BED, including increased industrialization and urbanization. Both have been linked to prevalence of BN (van Son, van Hoeken, Bartelds, van Furth, & Hoek, 2006) and BED (Favaro, Ferrara, & Santonastaso, 2003), potentially because of their influence on food availability. A key feature of both BN and BED is recurrent binge-eating episodes, which require availability of large quantities of readily edible food. In many regions around the world, there simply is not enough food for binge-eating episodes to occur. Thus modernization rather than Westernization per se may influence who suffers from eating disorders around the world. The following section explores this question from a historical perspective by attempting to understand when in history eating disorders emerged and whether more people are suffering from eating disorders now than ever before.

History of Eating Disorders

Identifying when eating disorders became defined syndromes—when they were first named in the medical literature, and when these names were made formal diagnostic categories—provides one approach for understanding when these disorders emerged in history. However, this approach represents only when an eating disorder was recognized as a mental illness, not when it first plagued people's lives. Thus this section presents historical accounts of syndromes that resemble each *DSM-5* eating disorder described in Chapter 1 that predate their official recognition. As will be seen, the various eating disorders described in the *DSM-5* have each had very distinct historical trajectories.

Historical Accounts of Self-Starvation in Adolescent Girls

Early Christianity offers a possible case of AN from the late fourth century A.D. in *Blessila,* an account in which a 20-year-old woman died from self-starvation (Bemporad, 1996). Cases of self-starvation attributed to demonic possession and purportedly cured by exorcism were documented during the fifth and eighth centuries (Bemporad, 1996). Sometime in the eighth or ninth century, St. Wilgefortis allegedly engaged in self-starvation resulting in her becoming emaciated and developing lanugo (growth of fine, downy hair all over the body) (Bemporad, 1996; Lacey, 1982). However, these early cases are controversial (Bynum, 1987), and details for the oldest historical cases are extremely limited. Starvation could have been caused by physical or psychological conditions unrelated to AN. A key challenge to interpreting the nature of the illnesses in these cases is lack of insight into the motivations of the affected individuals.

Among possible AN cases from the 12th to the 17th centuries for which insight into the deliberate nature of self-starvation is available, probably the most has been written about fasting medieval religious **ascetics.** Bell (1985) reported that approximately 90 saints living on the Italian peninsula from A.D. 1200 onward suffered from "holy anorexia." Holy anorexia, like AN, involved food refusal resulting in emaciation but was interpreted as an act of God by those afflicted. The following case studies of St. Catherine of Siena and St. Veronica are summarized from Bell (1985).

Case Study: St. Catherine of Siena

The story of St. Catherine of Siena (Figure 3.2) begins with a "robust, happy, obedient child" (Bell, 1985, p. 52) who goes on to seek spiritual perfection by conquering all of her bodily drives (hunger, fatigue, and sexual desire). She began fasting at around 16 years of age. At various stages of her life, her diet was restricted to raw vegetables, bread, and water; raw vegetables and water; and water and the sacrament. She engaged in vigorous physical activities, including long, fast-paced walks. She constantly chewed on bitter herbs and spat out the juice and saliva. St. Catherine also was known to self-induce vomiting. The following comment on her life is attributed to one of St. Catherine's contemporaries, Raymond of Capua: "She was constrained every day to vomit what she had eaten. To do this she regularly and with great pain inserted stalks of fennel and other plants into her stomach, otherwise being unable to vomit" (Bell, 1985, p. 28). According to Bell (1985, p. 24), when she was "warned that by such eating habits she was bringing about her own death, Catherine shot back that eating would kill her anyway so she might as well die of starvation, and do as she wished in the meantime." St. Catherine died from starvation in 1380, at approximately 32 years of age.

St. Catherine had many characteristics resembling those of modern-day patients with AN. She engaged in food refusal, excessive exercising, and self-induced vomiting. Her constant chewing of bitter herbs may be analogous to the modern use of sugar-free gum to satisfy the urge to eat without actually consuming food. Both Catherine's age of onset and eventual death from starvation are characteristic of modern-day AN patients.

FIGURE 3.2 Catherine of Siena refused to eat despite repeated pleas and commands from Church superiors. Source: Online collection of Brooklyn Museum; Photo: Brooklyn Museum. Frank L. Babbott Fund, Frank Sherman Benson Fund, Carll H. de Silver Fund, A. Augustus Healy Fund, Caroline A. L. Pratt Fund, Charles Stewart Smith Memorial Fund, and the Ella C. Woodward Memorial Fund.

Case Study: St. Veronica

St. Veronica (Veronica Giuliani) began a pattern of self-starvation at age 18. Like St. Catherine, she ate little (e.g., bread, water, "five orange seeds") or nothing, slept little, and was very active. When forced to eat, St. Veronica also vomited (although without obviously inducing the episodes). St. Veronica, who was a nun, was in conflict with her fellow sisters: "Most of the time she reasoned that she was in a race against all the other novices to show who loved God the most. She was losing, and so despite all the sleepless nights spent crying over she knew not what, Veronica carried more water and chopped more wood than anyone else" (Bell, 1985, p. 71). The sisters observed Veronica to have episodes in which she gorged on food, which were particularly likely to occur when she believed herself to be alone. According to an account by Abbess Ceoli, "the sisters sometimes found Sister Veronica in the kitchen, the refectory, or the dispensary, where she ate everything there was" (Bell, 1985, p. 75). Veronica was also accused of having intercourse with her confessor. St. Veronica's life was filled with contests of will between her and her superiors. Early in her monastic career, she openly defied orders. In response to a confessor's commanding her to provide a genuine confession versus confessing to trivial errors, she replied, "Father, what do you want that we should do, since we are always silent and cloistered, or to say it more clearly, imprisoned?" (Bell, 1985, p. 71). St. Veronica ultimately recovered from her disordered behavior. This happened sometime between her 30s and 50s, and she lived until the age of 67, dying in 1727.

Although St. Veronica's case differs significantly from that of St. Catherine, she resembles patients with AN who engage in self-starvation, excessive exercise, and binge-eating episodes. In addition, she demonstrated a competitive drive to be better than her fellow novices and asserted her own will when pressured to conform to the expectations of others. As will be discussed in Chapter 7, striving for achievement is a psychological feature often observed in patients with AN and thought to contribute to risk for developing the illness.

For the 17th and 18th centuries, Bliss and Branch (1960) found nine dissertations on apparent anorexia written between 1685 and 1770. In 1694, Morton provided the first medical account of a self-starvation syndrome that described the condition as an illness. Morton described the teenage daughter of Mr. Duke as having "nervous atrophy" or "a Nervous Consumption" characterized by loss of appetite, extreme emaciation, amenorrhea, overactivity, and indifference to the condition.

Case Study: Mr. Duke's Daughter

Mr. Duke's 18-year-old daughter experienced amenorrhea and significant weight loss that could not be attributed to tuberculosis or chlorosis (a form of anemia), appearing "like a Skeleton only clad with skin" (Morton, 1694, p. 9). The patient engaged in "continual poring upon Books, to expose her self both Day and Night to the injuries of the Air" (p. 8)—meaning that she demonstrated a high level of outdoor activity throughout the day and night. After initial compliance with treatment, she "quickly tired with Medicines, she beg'd that the whole Affair might be committed again to Nature, whereupon consuming every day more and more, she was after three Months taken with a Fainting Fit and dyed" (p. 9).

The cause of Miss Duke's death is unclear. However, one plausible explanation seems to be **refeeding syndrome,** which is caused by reintroducing too much food too quickly to a person in a state of starvation. (See J. M. Walsh, Wheat, & Freund, 2000, for a review of treatment complications in AN.)

At around the same time as the case of Miss Duke, "Miraculous Maids" (predominantly girls between the ages of 14 and 20) who engaged in self-imposed fasts emerged throughout the European countryside; they were considered "miraculous" for their ability to eat nothing yet remain completely healthy, including maintaining a healthy weight (Bemporad, 1996). Throughout the 18th and 19th centuries fasting girls gained great attention in popular and scholarly media throughout America and Europe (Bemporad, 1996; Brumberg, 1989; Vandereycken & van Deth, 1994). Typically, these cases involved adolescent girls who abruptly stopped eating. They created a mixture of religious awe and medical concern. Unlike the Miraculous Maids, other fasting girls became extremely emaciated, in some cases dying. Among them were Lina Finch [1886], Kate Smulsey [1885], and Lenora Eaton [1881], who all reportedly died of starvation before the age of 22 as a result of their food refusal (Brumberg, 1989). (Square brackets indicate the years of reported cases that have been described within cited references.) Sarah Jacobs [1869], the "Welsh Fasting Girl," represents a particularly tragic case of self-starvation leading to death (Bemporad, 1996; Brumberg, 1989; Vandereycken & van Deth, 1994).

Case Study: Sarah Jacobs

Sarah Jacobs began to fast in 1867, at the age of 12. Her parents publicized her behavior with the support of a local clergyman, who confirmed the authenticity of the claims. She became a tourist attraction for the curious and a source of inspiration for religious pilgrims. As with other fasting girls of her time, Sarah's fame attracted concern and skepticism from the medical profession. To resolve the ongoing debate over her case, nurses from Guy's Hospital were dispatched to watch the girl. As a condition of the watch, her parents insisted that she not be offered unsolicited food. Over the course of the week of observation, Sarah grew feeble and lost the ability to regulate her body temperature. The nurses and supervising doctors attempted to end the watch and recommended refeeding, but her parents refused to end or modify the conditions of the test, and Sarah never requested food. After 10 days, she died of starvation.

According to Brumberg (1989), Anglo-American girls during the Victorian era were well acquainted with the religious fasting of medieval saints, and St. Catherine of Siena's biography was included in inspirational books for girls. Indeed, the actions of Sarah Jacobs's parents seem to reflect a deep faith in a divine source of their daughter's food refusal. However, the growing field of psychiatry viewed these girls as suffering from "nervous" conditions.

Introduction of Anorexia Nervosa as a Named Syndrome

Whereas the fasting girls of the 18th and 19th centuries embraced the continuity between the religious medieval fasting of saints and their own behaviors, psychiatrists instead saw

a continuity between the extreme fasts leading to death among adolescent girls in the United States, England, France, and Germany and the newly identified syndrome of AN. William Gull introduced the term *anorexia nervosa* in the medical literature in 1874 to describe four adolescent girls with deliberate weight loss, three of whom went on to achieve full weight recovery. Around the time of Gull's publication, independent descriptions of a similar syndrome appeared under the labels *l'anorexie hysterique* in France and *anoressia* in Italy (Keel & Klump, 2003). A paper read at a meeting of the South Australian branch of the British Medical Association in 1882 documented two cases of anorexia in young women in Australia (Vandereycken & Beaumont, 1990), and in 1894 Kissyel (as reported in DiNicola, 1990b) described a case of severe *hysterical anorexia* in an 11-year-old girl in Russia.

Just before the turn of the 20th century, American physicians began to differentiate anorexia as a syndrome distinct from the larger category of hysteria (Vandereycken & Lowenkopf, 1990), and German physicians distinguished AN from the larger category of **neurasthenic disorders** (Vandereycken, Habermas, van Deth, & Meermann, 1991). Anorexia nervosa has been included in official diagnostic classifications of mental disorders since publication of the first edition of the *DSM* in 1952 (American Psychiatric Association, 1952).

Despite the sudden psychiatric attention given to fasting girls around the world in the latter part of the 19th century, it was Gull's characterization of AN that gained the most prominence. Gull's (1874, 1888) descriptions provide the first accounts of the disorder as it is recognized and defined today. They also are strikingly similar to the accounts of fasting girls, nervous consumption, and holy anorexia of earlier periods.

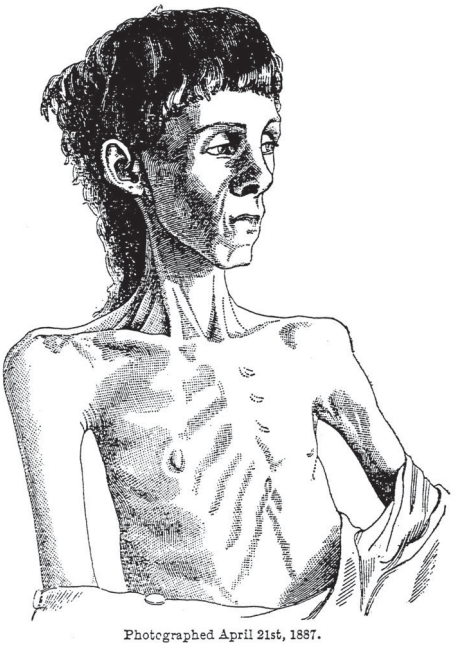
Case Study: Miss K. R.

The following case study of Miss K. R., a 14-year-old girl, is presented verbatim as described by Gull (1888):

> The patient, who was a plump, healthy girl until the beginning of last year (1887), began early in February, without apparent cause, to evince repugnance to food; and soon afterwards declined to take any whatever, except half a cup of tea or coffee. On March 13th, she traveled from the north of England, and visited me on April 20th. She was then extremely emaciated, and persisted in walking through the streets to my house, though an object of remark to the passers-by. [. . .] Patient expressed herself as quite well. A nurse was obtained from Guy's, and light food ordered every few hours. In six weeks Dr. Leachman reported her condition to be fairly good; and on July 27th the mother wrote: "K. is nearly well. I have no trouble now about her eating. Nurse has been away three weeks."

Figure 3.3 shows Miss K. R.'s emaciated appearance in late April 1887, and Figure 3.4 depicts her notable improvement in mid-June 1887.

Summing up what the historical record tells us, it is clear that a syndrome of self-starvation existed before the modern era. Like AN, this syndrome predominantly affected



Photographed April 21st, 1887.

FIGURE 3.3 Miss K. R. at age 14 years, height 5 feet 4 inches, weight 63 pounds, diagnosed with anorexia nervosa. (Photographed April 21, 1887.) Source: *The Lancet*, 1888, Vol. 1, pp. 516–517. Reprinted with permission from Elsevier.

Photographed June 14th, 1887.

FIGURE 3.4 Miss K. R. following successful treatment for anorexia nervosa (Photographed June 14, 1887). Source: *The Lancet*, 1888, Vol. 1, pp. 516–517. Reprinted with permission from Elsevier.

adolescent girls and young adult women. Like AN, cases could end in either full recovery or death. Unlike AN in the modern era, starvation was not attributed to a fear of becoming fat. Reasons for self-starvation included pursuit of moral superiority, attention seeking, and fear about the danger food posed to the body—all features that are often seen in modern-day AN.

The absence of weight/shape concerns in most (though not all) historical cases may reflect the importance of modern cultural ideals in producing body image disturbance. However, the similarities between historical and modern cases appear to outweigh this difference—particularly because a *DSM-5* diagnosis of AN does not require fear of gaining weight or of becoming fat, undue influences of weight or shape on self-evaluation, or misperception of weight or shape. Instead, an individual can be diagnosed with AN if he or she deliberately consumes less food than the body requires, engages in behaviors that prevent weight gain, and seems indifferent to the medical consequences of severe weight loss. Thus these historical cases could all meet *DSM-5* criteria for AN.

Historical Accounts of Binge, Purge, and Binge–Purge Syndromes

Several authors have reviewed historical reports of syndromes characterized by binge eating, purging, or both prior to the formal recognition of BN, BED, and purging disorder (Habermas, 1989; Parry-Jones, 1991; Parry-Jones & Parry-Jones, 1991; D. M. Stein & Laakso, 1988; Ziolko, 1996). Although many of these cases involved the coupling of binge eating with self-induced vomiting, it is unknown whether those behaviors were accompanied by preoccupations with weight or shape. Like arguments asserting the necessity of weight phobia for defining AN, concerns about weight and shape have been claimed to be core features of BN (Russell, 1979). However, like definitions of AN, definitions of BN have not always required body image disturbance. For example, body image disturbance was not a required feature of bulimia when it was introduced in the *DSM-III* (American Psychiatric Association, 1980). In addition, body image disturbance has never been required for a diagnosis of BED. Thus this section focuses on historical descriptions of the key behaviors that constitute BN, BED, and purging disorder.

Crichton (1996) speculated on whether the Roman emperors Claudius (A.D. 41–54) and Vitellius (A.D. 69) represented cases of BN in the early part of the first millennium A.D. Descriptions of Vitellius suggest that the emperor's excessive food intake was not under his control: "He was a man of not only such extreme and impulsive, but also disgusting, gluttony that he could not even curb it during a sacrifice or on a journey" (Suetonius, quoted in Crichton, 1996, p. 204). Loss of control is currently a criterion for identifying binge-eating episodes within BN and BED. Crichton stated that vomiting distinguished Claudius and Vitellius from historical peers who experienced a binge-only syndrome, comparable to BED. However, binge eating and purging may have represented a common behavioral pattern among the elite in the Roman Empire; Seneca is known to have commented, "Vomunt ut edant, edunt ut vomant" ("They vomit so that they may eat and eat so that they may vomit"). At least in the case of Vitellius, this pattern seems not to have been entirely volitional.

Awareness of a disorder characterized by overeating and self-induced vomiting in Arabic medicine dates as far back as the eighth century A.D., when the philosopher and physician Avicenna prescribed self-induced vomiting to undo the ill effects of overeating. However, he warned:

> To procure emesis (vomiting) to an undue degree is injurious for the stomach. It is also prejudicial to the thorax and to the teeth. The custom of some people of eating to excess and then procuring emesis . . . is one of the things which ends in chronic disorders. (Gruner, 1930, p. 498)

From the 12th to the 17th centuries many of the fasting saints were reported to have engaged in binge eating (e.g., St. Veronica) and self-induced vomiting (e.g., St. Catherine) (Bell, 1985; Rampling, 1985). These cases appear to fall within the *DSM-5* diagnosis of ANBP. It is unclear whether there were cases of binge eating and purging among normal-weight women of this time. If cases of the latter sort were common in convents, there is little reason to think that they would have gone unnoticed, given the religious significance of women's eating during medieval times (Bynum, 1987). In addition, nuns living within convents shared a common kitchen, decreasing the opportunity to consume large quantities of food inconspicously.

In the 17th century, a 50-year-old man experienced uncontrollable eating followed by vomiting for 20 days each year. Following the 20-day binge–purge cycle, the man fasted for 20 days and then resumed normal eating for the remainder of the year (Parry-Jones & Parry-Jones, 1991; Ziolko, 1996). Robert Whytt [1764] provided a description of *fames canina,* or "canine appetite," originally observed by Dr. Richard Lower in the 17th century (Silverman, 1987). According to Whytt [1764], Lower observed "an uncommon hunger" among hypochondriac and hysteric patients that produced "a great craving for food" (Silverman, 1987, p. 145). However, Whytt reported, Lower found that "in other cases, . . . the morbid matter affecting the nerves of the stomach in hypochondriac and hysteric patients, sometimes occasions a want of appetite and a *nausea*" (Silverman, 1987, p. 145). According to Ziolko (1996), Forestus [1602] described a nun afflicted with *kynorexia,* another form of canine appetite, who was miraculously cured after several unsuccessful medicinal treatments by physicians. Like *fames canina, kynorexia* was defined by insatiable appetite, out-of-control eating, and compulsive vomiting as a result of excessive food intake (Stein & Laakso, 1988; Ziolko, 1996).

Parry-Jones and Parry-Jones (1991) reviewed 12 potential cases of BN from the 17th to the 19th centuries. Of these, five cases partially resembled BN, and three cases involved binge eating without inappropriate compensatory behavior, suggestive of BED.

Case Study: Dr. Samuel Johnson

Parry-Jones (1992) detailed how the case of the famed English writer Dr. Samuel Johnson [1784]—one the five BN-like cases noted by Parry-Jones and Parry-Jones (1991)—meets *DSM-III-R* criteria for BN (American Psychiatric Association, 1987). Johnson was known for his gluttonous behavior. In an autobiography, he reported that his eating patterns began in

childhood and elicited comments from relatives when he was 9 years old. At one particularly notorious meal, Dr. Johnson consumed large portions of venison, fowl, pork, goose, and dessert in one sitting. To control his weight, he engaged in fasting and used senna as a purging agent. He was very concerned about his excessive weight and his health in general. According to accounts of the time, Dr. Johnson appeared to lose awareness of his surroundings while eating and would tear though food with his bare hands, sweating profusely, until no food remained.

Like modern-day BN patients, Dr. Johnson suffered from binge-eating episodes and engaged in inappropriate compensatory behavior to control his weight. Unlike typical modern-day BN patients, he was an obese middle-aged man whose disorder began with binge eating alone in childhood. This clinical picture is more characteristic of BED.

Habermas's (1989) review of Briquet [1859] revealed a case of apparent BN in a woman who "ate well" but vomited up everything she had eaten and maintained a normal weight (p. 267). Van Deth and Vandereycken (1995) reviewed cases of *hysterical vomiting* and noted that some of them occurred in individuals of normal weight who also engaged in binge-eating and fasting behaviors. Notably, most of these cases occurred in female adolescents. However, because binge-eating episodes were absent in several other cases, van Deth & Vandereycken equated them more with a modern-day **conversion disorder, psychogenic vomiting**. Historical cases characterized by purging in the absence of binge eating among individuals of normal weight may represent forerunners to purging disorder. Rosenvinge and Vandereycken (1994) reviewed a case of *hysteria* described by Selmer in which a 12-year-old girl refused to eat but maintained normal weight. This apparent contradiction was explained one night when the girl's mother observed her "'eating butter, herrings, potatoes and all the food she was able to find in the house'" (Selmer, 1892, quoted in Rosenvinge & Vandereycken, 1994, p. 280). Thus this girl appeared to fast during the day and binge eat at night, a behavioral pattern that may represent BN, BED, or, potentially, night eating syndrome.

Turning now to the first half of the 20th century, Pope, Hudson, and Mialet (1985) reviewed a case that involved the combination of binge eating with purging. A 17-year-old boy, "Ron," experienced episodes of "'voracious' appetite," "never felt satiety," and engaged in self-induced vomiting after periods of heavy food consumption (Pope et al., 1985, p. 741). Habermas (1991) presented Ludwig Binswanger's description of Irma, published in 1909, as "the first known report on a case of bulimia nervosa at normal body weight" (p. 361). This 22-year-old patient engaged in recurrent binge eating and fasting and experienced fear of becoming overweight. The case histories of Ellen West [Binswanger, 1944] and Laura [Lindner, 1940] also have been interpreted as examples of BN in the first half of the 20th century (Stein & Laakso, 1988). However, several experts in eating disorders have reviewed Ellen West as representing AN, and it is unclear whether she ever engaged in binge eating and purging at normal weight (DiNicola, 1990a). Cases described in the German psychiatric literature by Wulff [1932], Bergmann [1932], and Feuchtinger [1942] also were characterized by binge eating and purging (Habermas, 1989, 1992; Stunkard, 1990).

According to Habermas (1989), Wulff's [1932] cases included four women and one man, all of whom experienced periods of compulsive eating at normal weight. In three cases, onset occurred during puberty. In two cases, vomiting was present in addition to dieting. Finally, in three cases, body image disturbance was recorded. However, a different picture

emerges from Stunkard's (1990) translation of Wulff's [1932] work. First, Wulff described marked obesity in two of four cases (Cases A and C). Further, in three of the four cases (Cases A, B, and C), binge eating and fasting or purging occurred in distinct phases, suggesting that these patients may have shifted between BED and purging disorder. Only Case D experienced a binge–purge cycle within one period; these periods alternated with short periods of abstinence and fasting.

In summary, although the historical accounts offer clear evidence of syndromes characterized by binge eating, purging, and both binge eating and purging well before the recognition of BED, purging disorder, and BN in the present day, this evidence is quite sparse compared with that for a self-starvation syndrome. Moreover, unlike historical descriptions of deliberate self-starvation, the earliest accounts of binge–purge syndromes do not seem to have occurred preponderantly in adolescent girls. In fact, prior to the 19th century, cases characterized by binge eating involved mostly adult men. Some of these historical cases, particularly those of middle-aged men who were obese and may not have been purging, resemble BED. There are hints that purging disorder and night eating syndrome may have existed in adolescent and young adult women long before their official recognition; however, it is difficult to make a differential diagnosis between purging disorder and psychogenic vomiting.

Introduction of Night Eating Syndrome, Binge-Eating Disorder, Bulimia Nervosa, and Purging Disorder as Named Syndromes

In 1955 Albert Stunkard, a psychiatrist working at the University of Pennsylvania Medical School, introduced the term *night eating syndrome* to the medical literature to name a subtype of obesity in a series of 25 patients uniquely characterized by a disrupted circadian rhythm in which the majority of food intake occurred at night (Stunkard, Grace, & Wolff, 1955). These patients described an absence of hunger in the morning and increased feelings of depression in the evening. Four years later, Stunkard (1959) introduced the term *binge eating syndrome,* later referred to as *binge eating disorder* by Spitzer (1991), as another behavioral subtype of obesity, characterized by recurrent episodes of binge eating.

Despite these early characterizations of two illnesses characterized by excessive food intake among the obese, BED was only first included in the *DSM-IV* in 1994, as a provisional eating disorder diagnosis. Following an accumulation of research on BED (a search of PubMed identifies over 750 articles with "binge-eating disorder" in the title since 1994), it was formally included as an official eating disorder, on equal footing with AN and BN, in the *DSM-5* in 2013. In contrast, night eating syndrome has been relatively ignored in research (a PubMed search identifies 106 articles on "night eating syndrome" since 1994) and in diagnostic systems; it was included as a named condition among OSFED in the 2013 publication of the *DSM-5*.

Bulimia nervosa was first named as a distinct disorder approximately a century after the recognition of AN and more than two decades after the descriptions of night eating syndrome and BED. Gerald Russell used the term in 1979 to name a binge–purge disorder in a series of 30 patients seen over a period of 6.5 years. In stark contrast to the delays between introductions of night eating syndrome and BED as named syndromes to the literature and their inclusion in diagnostic schemes, BN was included under the name *bulimia*

in the *DSM-III* in 1980 and then renamed *bulimia nervosa* with the 1987 publication of the *DSM-III-R*. This rapid adoption of BN as an official eating disorder reflected a growing sense that the illness had become epidemic among young women, with cases of BN far outnumbering cases of AN in eating disorder treatment programs.

Finally, Pamela Keel introduced the term *purging disorder* to the medical literature in 2005 (Keel, Haedt, & Edler, 2005) to describe individuals who recurrently purged by self-induced vomiting or by laxative or diuretic abuse following consumption of normal or small amounts of food and who were overconcerned with their weight and shape but were neither underweight nor experienced large binge-eating episodes. Individuals with this clinical presentation had previously been described by Mitchell in a case series of atypical eating disorders—that is, disorders that did not meet diagnostic criteria for AN or BN (Mitchell, Pyle, Hatsukami, & Eckert, 1986)—and described using other terms in other reports (Keel & Striegel-Moore, 2009). Like night eating syndrome, purging disorder was added as a named condition within the broader category of OSFED in the *DSM-5* in 2013, because of evidence of its prevalence and clinical significance.

Modern Trends in Incidence and Prevalence of Eating Disorders

An important benefit of naming and defining eating disorders is that it permits examination of disorder epidemiology. This section discusses epidemiological data on whether specific eating disorders are affecting an increasing portion of the population. When evaluating such changes, it is important to link the identification of cases with time. Lifetime prevalence is not always useful for this purpose, because an illness may occur at any time over an individual's life; as a result its lifetime prevalence tends to increase with age. In contrast to this pattern, eating disorders have demonstrated a cohort effect for lifetime prevalence such that they are actually more common among younger cohorts than among older cohorts (Hudson et al., 2007). This effect suggests that eating disorders have become increasingly common in modern times. It is also possible, however, that older individuals have forgotten eating patterns from earlier in their lives or deny a history of mental health problems because of fears of stigmatization (Moffitt et al., 2010).

Using **point prevalence** avoids this problem of retrospective recall. This statistic evaluates the percentage of a population that has a disorder at a given time. Point prevalence can be compared across cohorts of the same population (e.g., college students in 1982, 1992, and 2002; Keel et al., 2006) to evaluate whether an illness is becoming more common. However, point prevalence combines people who have a disorder without regard for when they developed it, whether years ago or shortly before the time of evaluation. Thus point prevalence over time is influenced by two factors—the proportion of the population affected by an illness and the chronicity of that illness.

A third approach to evaluating changing rates of eating disorders over time is to examine their **incidence**. Incidence represents the number of new cases of an illness per 100,000 people per year. Thus if there were 360 new cases of a disorder during a year in a population of 2.5 million, the incidence would be 14.4 per 100,000 population per year. Because incidence counts only new cases and identifies these cases by the year in which their onset was

recognized, examining incidence rates over time provides a precise examination of whether eating disorders are affecting more people now than ever before.

A **meta-analysis** (Keel & Klump, 2003) and a systematic review (Hoek & van Hoeken, 2003) found statistically significant increases in AN incidence over the 20th century. However, this increase had a modest effect size (Keel & Klump, 2003), indicating that while the increase in incidence was reliable, it was not dramatic. Further, Hoek and van Hoeken (2003) reported that the increase was evident until the 1970s, after which incidence rates demonstrated relative stability. Two more recently published epidemiological studies support the stability of AN incidence rates from 1994 to 2000 in the United Kingdom (Currin, Schmidt, Treasure, & Jick, 2005) and from 1985–1989 to 1995–1999 in the Netherlands (van Son et al., 2006). Although overall incidence for AN appears to have leveled off, evidence suggests increasing rates in the highest-risk group, that of females aged 15–19 years, with incidence nearly doubling from 56.4 to 109.2 per 100,000 person-years in the Netherlands study (van Son et al., 2006). These results echo findings from the United States (Lucas, Crowson, O'Fallon, & Melton, 1999) in which incidence rates showed a linear increase from 1935 to 1989 in females aged 15–24 years. Thus popular media portrayals of an AN "epidemic" in young women have some basis in truth, because rates of the illness have increased over time, particularly among adolescent and young adult females.

A meta-analysis of BN incidence studies (Keel & Klump, 2003) supported a statistically significant and large increase in BN during the latter half of the 20th century; this increase was particularly evident from studies that tracked patient records from the 1970s to the mid- to late 1980s. There are no incidence data for BN prior to 1970, supporting Russell's (1997) assertion that BN is a new disorder. Although there appears to have been a sudden appearance and rapid increase in BN over a brief time in the 20th century, more recent epidemiological data collected in England, the Netherlands, and the United States suggest that BN may have peaked in the 1980s in Western nations and gradually subsided to current prevalence estimates (Smink, van Hoeken, & Hoek, 2013). Currin et al. (2005) reported an increase in BN incidence in English women aged 10–39 years from less than 25 per 100,000 person-years in 1988 to more than 50 per 100,000 person-years in 1996, but this was followed by a nearly 40% decrease in incidence by 2000. Van Son et al. (2006) reported a 29% decline in Dutch BN incidence from 1985–1989 to 1995–1999; this decrease became statistically significant when BN incidence from 2005–2009 was added (Smink et al., 2013). Finally, Keel et al. (2006) reported a significant decline (nearly 60%) in the point prevalence of BN across three U.S. college cohorts assessed in 1982, 1992, and 2002. In contrast, Crowther, Armey, Luce, Dalton, and Leahey (2008) reported no significant changes in BN point prevalence across college cohorts from 1990 to 2004 in the U.S.; however, their study might have observed higher rates from the early 1980s had it included such a cohort.

Although some studies have evaluated the prevalence and incidence of BED, none have examined time trends using point prevalence or incidence (Keel, 2010; Smink et al., 2013), making it difficult to evaluate whether the disorder has become more common. However, the lifetime prevalence of BED in the United States increased over successive birth cohorts from 1944 to 1985 (Hudson et al., 2007), providing indirect evidence that BED has become more common over recent decades.

Studies of the prevalence of night eating syndrome have been restricted to single cohorts, and there are no studies of night eating syndrome incidence over time. In a small sample of 68 overweight or obese individuals diagnosed with a serious mental illness and seeking weight loss, 25% met proposed research criteria for night eating syndrome (Lundgren, Rempfer, Brown, Goetz, & Hamera, 2010). In a sample of 395 families in Canada in which one parent was obese, night eating syndrome was found to affect 0.0% of children, 0.5% of mothers, and 0.3% of fathers (Lundgren et al., 2012). In a multisite study of 845 patients diagnosed with type 2 diabetes, 3.8% were diagnosed with night eating syndrome (Allison et al., 2007). Differences in sample size and inclusion criteria are no doubt responsible for this very large range of prevalence estimates. Thus more research is needed to understand who suffers from night eating syndrome. A challenge for past studies has been that the criteria for night eating syndrome differ qualitatively from those for defined eating disorders, making data from most epidemiological studies of eating disorders irrelevant.

As with night eating syndrome, studies of the incidence of purging disorder have been restricted to single cohorts with no evaluation of population-based changes over time. Estimates of lifetime prevalence for purging disorder in women have ranged from 1.1% (Favaro et al., 2003) to 5.3% (Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006). These estimates do not include individuals with a lifetime history of AN, BN, or BED. In adolescents, lifetime prevalence has ranged from 2.0% (Field et al., 2012) to 3.4% (Stice et al., 2013), with a reported incidence of 447 per 100,000 person-years (Stice et al., 2013). The reported point prevalence of purging disorder has ranged from 0.3% to 1.0% in women (Crowther et al., 2008; Gauvin, Steiger, & Brodeur, 2009; Haedt & Keel, 2010; Hay, Mond, Buttner, & Darby, 2008). Most studies have examined purging disorder in women only, but one study reported that this disorder is significantly more common in women (0.9%) than in men (0.1%) (Haedt & Keel, 2010). Although no studies have examined purging disorder incidence over time, studies of successive cohorts of college students have found no significant changes in point prevalence over time (Crowther et al., 2008; Haedt & Keel, 2010). No data have been published regarding the prevalence of purging disorder prior to 1982. Thus it remains unclear whether purging disorder became increasingly common during the period leading up to its recognition in the 21st century or whether purging disorder was simply overlooked because no one thought to ask about purging in the absence of low weight or binge eating (Swanson, Brown, Crosby, & Keel, 2014).

Conclusion

The answer to the question "Who suffers from eating disorders?" is that *anyone* can suffer from these disorders. Although eating disorders affect more women than men, men are not immune to developing AN, BN, BED, night eating syndrome, or purging disorder. In addition, men may be at increased risk for eating disorders that are not currently recognized as such and that are characterized by a desire for a larger physique and use of unhealthy, extreme behaviors to achieve these ideals. In contrast to the stereotype that eating disorders affect only White girls, epidemiological data indicate that individuals from ethnic and racial minority groups are as likely overall to suffer from AN, BN, and BED as are non-Hispanic

White individuals and that some ethnic minority groups may actually be at greater risk for BN than are their White counterparts.

While acculturation to dominant Western culture has been associated with increased risk for eating disorders in Latino individuals, there appears to be no association between acculturation to Western ideals and risk for eating disorders among Asian individuals. The latter finding mirrors evidence that AN exists in non-Western cultures without exposure to Western values. Conversely, while BN and BED have been reported in all non-Western regions of the world, their appearance seems to be closely tied to Westernization. This finding may reflect the importance of the thin ideal for developing distress related to binge eating or for responding to binge episodes with purging. Alternatively, it may reflect the extent to which increasing exposure to Western ideals has co-occurred with increased industrialization and economic shifts in access to food—both of which may contribute to observed cross-cultural and historical patterns of who suffers from eating disorders characterized by binge eating.

Unlike self-starvation, which can occur in any context, binge eating requires large quantities of readily edible food. Thus cases of BN and BED may be limited to individuals with access to abundant food. In places and times not marked by mass production of food, use of preservatives, and refrigeration, individuals may not have had access to enough food to supply recurrent binge episodes. Similarly, purging may be hampered in individuals who do not have access to modern plumbing. The ability to flush away evidence of self-induced vomiting or of laxative or diuretic abuse in the privacy of one's indoor bathroom greatly facilitates secretive purging in a way that outhouses and chamber pots do not. Thus the relative dearth of evidence for BN, BED, and purging disorder outside of their current cultural context may be due to their restriction to individuals with access to modern conveniences.

Lawlor and colleagues (1987) characterized their observation of an eating disorder in a Black adolescent boy as "rare." In retrospect, perhaps what was rare was the appreciation that the disorder could exist in a patient who differed so much from the expected norm. Future research on eating disorders would benefit from challenging stereotypes to promote the recognition of eating disorders and increase access to care regardless of gender, ethnicity, or race.

Key Terms

Acculturative stress
Ascetics
Conversion disorder
Incidence
Lifetime prevalence
Meta-analysis
Muscle dysmorphia
Neurasthenic disorders
Point prevalence
Psychogenic vomiting
Refeeding syndrome
Reverse anorexia

4

Finding the Causes of Eating Disorders

Going Beyond Descriptive Methods

Before examining the causes of eating disorders in Chapters 5–8, this chapter describes how research studies are designed to reveal causal factors. A common misperception is that statistics can be made to show anything. This is not true. Although people have believed that eating disorders are limited to White girls in the United States, epidemiological data do not support this perception, as shown in Chapter 3. However, epidemiological data are fairly straightforward. The number of patients with AN in Rochester, Minnesota, in 1944 or the percentages of White, Hispanic, and Asian women with eating disorders are descriptive statistics that do not require much interpretation. Even though statistics cannot be made to show anything, when researchers move from descriptive statistics to inferential statistics, interpretation of results can lead to very different, and sometimes erroneous, conclusions.

This chapter reviews the logic behind different research methods used to examine risk factors in eating disorders and the limits to conclusions about actual causes of eating disorders that can be drawn from different kinds of studies. The chapter thereby provides a working knowledge of the kinds of studies discussed in this book, and later chapters will refer back to it.

Hypotheses

Just as there are common perceptions of who suffers from eating disorders, there are common understandings of the causes of these disorders. One common explanatory model attributes increased rates of eating disorders to increasing societal idealization of thinness. Another model attributes development of an eating disorder to an individual's need for control or perfectionistic strivings. These models often reflect a combination of actual observations (of an individual or a group) and inferences about the meaning of these observations. Thus they are best described as hypotheses. Research studies are specifically designed to test hypotheses.

Several research methodologies can be used to test these and other hypotheses concerning eating disorders. Because it would be unethical to cause an eating disorder in a person, these research designs operate by a combination of deduction and inference. Deduction occurs when a premise is stated and then specific consequences of this premise are drawn. For example, if the stated premise is "Dieting causes binge eating" (Polivy & Herman, 1985), then a consequence that follows is "Individuals who diet should be more likely to report binge eating than individuals who do not diet." Using deduction, the accuracy of the consequence is tested. The particular consequence just mentioned can be tested with a simple comparison of binge eating in dieters and nondieters. If the predicted difference is found, it is inferred that dieting may cause binge eating—although this **correlation** does not actually prove the original premise, because there are alternative explanations for the association between binge eating and dieting. For example, binge eating may cause weight gain, which in turn causes dieting. Individuals who binge would then be more likely to diet than individuals who do not binge. For this alternative explanation to be true, however, dieting would need to follow binge eating, not vice versa. **Longitudinal studies** are used to test temporal relationships between a proposed risk factor and the onset of an eating disorder as a first step in inferring a causal association. The logic is simply that for A to cause B, A must precede B in time.

Longitudinal Studies

There are two basic types of longitudinal studies: retrospective, follow-back investigations and prospective, follow-up investigations. Each has its benefits and limitations. The basic design features of each type of study are described below, along with findings that have emerged in eating disorders research using these approaches.

Retrospective, Follow-Back Design

A retrospective, follow-back investigation begins with individuals whose eating disorder status is known. A case–control design would include individuals with eating disorders (cases) and demographically matched individuals who have never suffered from an eating disorder (controls). For example, a person with an eating disorder could be matched to an individual with no lifetime history of an eating disorder on factors such as sex, age, ethnic/racial background, and **socioeconomic status**. The purpose of matching across these factors would be to ensure that differences found between the eating disorder group and the control group can be attributed to the presence of the eating disorder rather than other factors that might differ between the groups. Matching participants on these variables assumes that the study is not concerned with investigating these variables' role in the eating disorder. A matched variable may not be of interest because it is already known to be a risk factor (e.g., gender) and does not require further investigation. Alternatively, a factor may not be of interest because it is not thought to be related to eating disorder risk (e.g., socioeconomic status) but could operate as a **nuisance variable** if it happened to differ between comparison groups. A nuisance variable is anything that creates differences but that is irrelevant for understanding the problem under investigation.

A retrospective, follow-back study employing a case–control design uses the onset of the eating disorder as a starting point. The study then examines potential differences between groups on historical variables from before this starting point. Fairburn and colleagues completed a series of studies using this approach to understand risk factors in the development of AN (Fairburn, Cooper, Doll, & Welch, 1999), BN (Fairburn, Welch, Doll, Davies, & O'Connor, 1997), and BED (Fairburn et al., 1998). In each study they identified a group of individuals with the eating disorder under investigation, a group of individuals with other forms of psychopathology (general psychiatric controls), and individuals who were free of eating and general psychopathology (healthy controls). This approach allowed them to differentiate between factors that appeared to increase the risk for psychopathology in general (factors that were elevated in the eating disorder group relative to healthy controls but not general psychiatric controls) and factors that were specific to increasing the risk for an eating disorder (factors that were elevated in the eating disorder group relative to both the healthy and general psychiatric controls).

Box 4.1 lists the factors examined in these studies. Fairburn and colleagues organized risk factors into three domains of vulnerability: environmental, personal, and

BOX 4.1 Potential Risk Factors for Eating Disorders Examined by Fairburn et al. (1997, 1998, 1999)

Environmental Vulnerability Domain

Subdomain 1: Parental problems
- Low parental contact
- Separation from parents
- Parental arguments
- Parental criticism
- Parental high expectations
- Parental overinvolvement
- Parental underinvolvement
- Parental minimal affection
- Maternal low care and high overprotection
- Paternal low care and high overprotection

Subdomain 2: Disruptive events
- Parental death
- Change of parent figure
- Parental chronic illness
- Frequent house moves
- Severe personal health problems

Subdomain 3: Parental psychiatric disorder (prior to age of onset)
- Parental depression
- Parental alcoholism
- Parental drug abuse

Subdomain 4: Teasing and bullying
- Teasing (not related to weight, shape, appearance, or eating)
- Bullying

Subdomain 5: Sexual and physical abuse
- Sexual abuse
- Repeated severe sexual abuse
- Physical abuse
- Repeated severe physical abuse
- Repeated severe physical or sexual abuse

Personal Vulnerability Domain

Subdomain 1: Childhood characteristics
- Negative self-evaluation
- Shyness (not included in Fairburn et al., 1999)
- Perfectionism
- Extreme compliance
- No close friends
- School absence through anxiety

Subdomain 2: Premorbid psychiatric disorder
- Major depression
- Drug abuse
- Alcohol abuse

Subdomain 3: Behavioral problems
- Marked conduct problems
- School absence (truancy)
- Deliberate self-harm

Subdomain 4: Parental psychiatric disorder (ever)[a]
- Parental depression
- Parental alcoholism
- Parental drug abuse

Dieting Vulnerability Domain

Subdomain 1: Dieting risk
- Family member dieting for any reason
- Family member dieting for shape or weight
- Family criticisms of body or eating
- Repeated comments by others about body or eating
- Teasing about body, appearance, or eating
- Parental obesity
- Childhood obesity
- Parental history of AN or BN (prior to age of onset)

Subdomain 2: Obesity risk
- Childhood obesity
- Parental obesity (ever)[a]

Subdomain 3: Parental eating disorder
- Parental history of AN or BN (ever)[a]

[a]"Ever" denotes before or after the age of onset of an eating disorder.

dieting. Environmental vulnerability factors are characteristics of the individual's environment that may increase the risk of an eating disorder, such as parental criticism. Personal vulnerability factors are characteristics of the individual that may increase risk, such as perfectionism. Dieting vulnerability factors are characteristics of the environment or the individual that would have increased the likelihood of dieting, such as childhood obesity.

Table 4.1 presents specific risk factors identified from studies using this design for AN (Fairburn et al., 1999; Pike et al., 2008), BN (Fairburn et al., 1997), and BED (Fairburn et al., 1998; Striegel-Moore et al., 2005). More risk factors were identified for BN than for AN or BED. Low self-esteem and perfectionism increased the risk of developing both AN and BN. Interestingly, factors that would contribute to body image concerns and dieting were elevated in BN but not AN (Fairburn et al., 1999; Pike et al., 2008). For both BN and BED, difficult family environments and weight problems were risk factors (Fairburn et al., 1998; Striegel-Moore et al., 2005). Finally, elevated parental expectations may be a transdiagnostic risk factor for AN, BN, and BED (Fairburn et al., 1997, 1998; Pike et al., 2008; Striegel-Moore et al., 2005).

TABLE 4.1 Specific Risk Factors for Eating Disorders

Vulnerability domain	Anorexia nervosa[a]	Bulimia nervosa[b]	Binge-eating disorder[c]
Environmental	Parental high expectations	Parental high expectations	Parental high expectations
		Low parental contact	Low parental contact
		Parental arguments	Severe personal health problems
		Parental criticism	
		Parental alcoholism	
Personal	Negative self-evaluation	Negative self-evaluation	
	Perfectionism	Perfectionism	
		Parental alcoholism (ever)	
Dieting		Childhood obesity	Childhood obesity
		Family member dieting for any reason	
		Repeated comments by others about shape or weight	
		Parental obesity (ever)	
			Family overeating or binge eating
			Family criticisms of body or eating

Note: Table shows risk factors from Box 4.1 on which individuals with the indicated eating disorders differed significantly from general psychiatric controls.
[a] Risk factors from Fairburn et al. (1999) and Pike et al. (2008).
[b] Risk factors from Fairburn et al. (1997).
[c] Risk factors from Fairburn et al. (1998) and Striegel-Moore et al. (2005).

A benefit of the retrospective, follow-back design is the ability to compare a large number of affected individuals with matched controls. Large samples increase the statistical power to detect differences, and the use of controls eliminates the need to control statistically for potential nuisance variables. A drawback of the follow-back design is the potential influence of **retrospective recall bias.** For example, individuals who have an eating disorder may be more likely to recall criticism about their weight or higher parental expectations than individuals who do not have an eating disorder when no true difference occurred. One means of avoiding recall bias is to use objective historical records (e.g., medical or school records). However, the variables that can be obtained from such records tend to be limited. For example, few medical records include valid or reliable measures of perfectionism or parental expectations. Because of these drawbacks, prospective, follow-up designs have been used in risk factor research.

Prospective, Follow-Up Design

A prospective, follow-up design begins with a large group of unaffected individuals and collects data both before and after the period of risk for onset of an eating disorder. Individuals who develop eating disorders over the course of follow-up are compared with individuals who do not develop eating disorders on factors measured before the disorder emerged. No matter how young participants are at baseline (i.e., when first assessed), it is crucial to measure disordered eating at baseline as well as at follow-up, because some individuals may have developed eating problems at an early age. If that is the case, associations found between predictors and outcomes may not reflect a true prospective association. Several groups have conducted prospective investigations, and much of this work will be reviewed in later chapters. What follows is a review of findings from major prospective studies of eating disorder risk factors in girls and boys.

Leon, Fulkerson, Perry, Keel, and Klump (1999) conducted a longitudinal study of the development of disordered eating in over 1,400 girls and boys in the 7th through 10th grades. Each adolescent cohort was followed prospectively until the students graduated or until the end of the five-year study. Students completed self-report assessments of *DSM-III-R* (American Psychiatric Association, 1987) eating disorder symptoms, mood disturbances (e.g., depression), self-esteem, dieting, exercise, body image, personality features (e.g., perfectionism), other risky behaviors (e.g., alcohol use), and pubertal status.

Box 4.2 shows how the investigators arranged the variables into vulnerability domains. Unlike Fairburn et al.'s (1997, 1998, 1999) arrangement of factors by hypothesized associations, Leon et al. (1999) defined domains according to how the risk factors were associated with each other using statistical methods. Thus smoking cigarettes, drinking alcohol, and constraint (a personality measure of impulsiveness) were highly related to each other and formed a category of related factors: Substance-Related Impulsivity. Not surprisingly, puberty and grade in school were highly correlated; these factors constituted the Development domain. Finally, body dissatisfaction, depression, negative emotionality (a tendency to feel unhappy and anxious), feeling ineffective, and low

BOX 4.2 Potential Risk Factors for Disordered Eating Examined by Leon et al. (1999)

Substance-Related Impulsivity

Smoking cigarettes
Drinking alcohol
Constraint (personality measure of impulse control)

Development

Puberty
Grade in school

Negative Affect/Attitudes

Body dissatisfaction (in girls, this factor also loaded on the Development domain)
Depression
Negative emotionality (personality measure of tendency to feel unhappy and anxious)
Ineffectiveness
Interoceptive awareness (ability to recognize and separate one's own feelings and internal sensations)

interoceptive awareness (ability to recognize one's own feelings) were interrelated as a third factor: Negative Affect/Attitudes.

Over the course of follow-up, the Negative Affect/Attitudes factor was a significant predictor of new onset of disordered eating attitudes and behaviors in both girls and boys (Leon et al., 1999). As a **diathesis,** negative affectivity is likely to be associated with negative attitudes toward the self, leading to low self-esteem and dissatisfaction with many aspects of the self, including physical appearance and weight. Leon et al. (1999) posited that negative affectivity is a nonspecific diathesis for the development of psychopathology and that cultural factors, such as an idealization of thinness for women, shape the expression of this diathesis as an eating disorder.

Pearson, Combs, Zapolski, and Smith (2012) examined predictors of binge-eating onset during the transition from elementary school to middle school in adolescent girls and boys by assessing 1,906 children during the spring of sixth grade, the beginning of seventh grade (first year of middle school), and the spring of seventh grade. Key findings from this investigation were that negative urgency (a personality feature that represents the tendency to act rashly in response to negative emotions) predicted increases in the expectation that eating would alleviate negative emotions, and that this expectation in turn predicted increases in binge eating. These findings were similar for boys and girls and for White and African American students.

Neumark-Sztainer, Paxton, Hannan, Haines, and Story (2006) examined prospective predictors of binge eating and extreme weight control behaviors (use of diet pills, laxatives,

diuretics, or self-induced vomiting) in 2,516 adolescents first assessed in middle and high school and evaluated again at five-year follow-up. For both girls and boys, greater body dissatisfaction predicted both binge eating and extreme weight control behaviors at follow-up. However, after controlling for BMI, body dissatisfaction no longer predicted binge eating in girls and no longer predicted extreme weight control behaviors in boys. Higher body dissatisfaction did not increase use of healthy behaviors, such as increased physical activity or increased fruit or vegetable intake. This finding has important implications for understanding how stigmatizing weight may do more harm than good in programs for preventing eating disorders and obesity (discussed further in Chapter 10).

Liechty and Lee (2013) examined prospective predictors of extreme weight control behaviors (use of diet pills, laxatives, or self-induced vomiting) in 14,322 adolescents first studied in middle school through high school and reassessed seven years later, when they were between the ages of 18 and 26 years. For both genders, higher BMI increased risk of later use of extreme weight control behaviors, and in girls, greater depression and dieting increased risk for disordered eating at follow-up.

Across prospective, follow-up studies, consistent predictors of risk for disordered eating include higher negative affect (Leon et al., 1999; Liechty & Lee, 2013) and body dissatisfaction (Leon et al., 1999; Neumark-Sztainer et al., 2006). In addition, higher BMI emerged as a risk factor from both retrospective, follow-back designs and prospective, follow-up designs, suggesting that individuals who diverge most from the thin ideal may be at greatest risk for developing disorders characterized by binge eating or purging.

An advantage of prospective, follow-up studies is the ability to test specific hypotheses concerning risk factors for eating pathology. In addition, such studies eliminate concerns about retrospective recall bias. A drawback is the need to begin assessments before eating disorders develop or control for their presence at baseline and to continue follow-up assessments throughout the entire period of risk. Many prospective risk factor studies are constrained by the need to retain a sample within a school setting. Thus with key exceptions noted above, studies of adolescents generally end when students graduate from high school, and studies of college students begin well after eating problems have begun. Given that the transition from high school to college reflects a peak age of onset for many eating disorders, the limited number of studies bridging this period is a notable limitation in risk factor research.

A second drawback of prospective studies is that they require very large samples, because only a very small percentage of individuals will develop an eating disorder (see the discussion of prevalence and incidence in Chapter 3). Most prospective studies are not able to predict the onset of AN or BN, because the rates of these disorders are extremely low. For example, Leon et al. (1999) measured disordered eating attitudes and behaviors but not eating disorders per se. Even studies that bridge the transition from adolescence to young adulthood use expanded definitions, focusing on specific disordered eating behaviors rather than eating disorders (Liechty & Lee, 2013; Neumark-Sztainer et al., 2006). The validity of this approach rests on two assumptions: 1) that eating disorders exist on a continuum with disordered eating (see Chapter 1) and 2) that AN, BN, and BED share risk factors. The latter assumption is only partially supported by prior research (Fairburn et al., 1997, 1998, 1999; Tyrka, Waldron, Graber, & Brooks-Gunn, 2002).

An additional drawback of both retrospective and prospective longitudinal studies is that they do not establish causation between variables. For example, dieting may precede and be associated with an increased risk of binge eating without necessarily causing binge eating. A third, underlying variable could cause both dieting and binge eating even though dieting emerges first. Perhaps biologically driven appetite disturbances cause an individual to set external limits on food intake by dieting, and these same appetite disturbances later cause binge-eating episodes. To demonstrate causation, experimental designs are necessary.

Experimental Studies

Experimental studies identify an independent variable that, when manipulated, causes a change in a dependent variable. Logically, experiments are a type of longitudinal study, because manipulations of the independent variable always precede changes in the dependent variable. With appropriate experimental controls, these designs can be very powerful in demonstrating that one factor causes another.

Analog Studies

As noted above, it would be unethical to attempt to cause an eating disorder. Thus most experimental studies start with a premise concerning a factor that causes eating disorders and then generate a hypothesis that would be true if the premise were correct. The study then tests this hypothesis as an indirect way of evaluating the original premise.

In this case, the specific hypothesis usually involves some analog of the factors thought to be important in causing eating disorders. These investigations are therefore called **analog studies.** Most experimental analog studies use college undergraduates as research participants. Thus the value of these studies depends on the extent to which causal patterns for analogous factors demonstrated in college students accurately represent causal patterns for eating disorders in the population at large.

Polivy and Herman (1985) and others (see Ruderman, 1986, for a review of early experimental research) have conducted a number of experimental analog studies evaluating the premise that dieting causes binge eating. The classic design compares responses to experimental manipulations in individuals with high or low scores on a measure of dietary restraint, referred to as *dieters* and *nondieters*, respectively. For example, individuals have been randomly assigned to consume either a milkshake or nothing (the experimental manipulation) and then asked to participate in a "taste test." When asked to consume nothing before the taste test, dieters consumed less during the taste test than nondieters, consistent with their being on a diet. When asked to consume a milkshake before the taste test, dieters consume a lot more during the taste test than nondieters. This effect has been demonstrated and replicated by a number of labs (Heatherton & Baumeister, 1991; Ruderman, 1986). Thus these studies show that a milkshake will cause a dieter to eat an unusually large amount of food—one of the criteria for BN. However, increased food consumption in these studies does not necessarily constitute a binge-eating episode, and dieters do not necessarily suffer from eating disorders.

Several experimental studies have examined and supported a causal relationship between exposure to media images and body dissatisfaction. Heinberg and Thompson (1995) randomly assigned women to view television commercials containing either appearance-related images or non–appearance-related images. Women in the appearance-related condition reported significantly greater body dissatisfaction after watching the commercials than did women in the non–appearance-related condition. Stice and Shaw (1994) randomly assigned women to view magazine pictures of very thin models, models with average builds, or no models. Women assigned to view pictures of very thin models reported significantly greater body dissatisfaction than did women assigned to the other two conditions. Recently, Mabe, Forney, and Keel (2014) examined the influence of Facebook use on preoccupation with weight or shape using an experimental design. This study sought to understand how recent changes in media consumption from traditional print magazines to images available through social media may or may not influence body image concerns. College women with Facebook accounts were randomly assigned to spend either 20 minutes on Facebook or 20 minutes in a control condition in which they read about the ocelot on Wikipedia and viewed a brief video clip about this rain-forest animal. Weight/shape preoccupation was measured before and after the experimental manipulation. Compared with women in the control condition, women using Facebook reported greater levels of weight/shape preoccupation after the 20-minute period. This finding suggests that social media merges widespread cultural influences (see Chapter 5) and peer influences (see Chapter 6) to influence eating disorder risk by providing women with constant access to viewing friends' favorite celebrities, most flattering photos of themselves, and intentions to diet and exercise. Notably, none of these three studies demonstrated that media images caused eating disorders. However, the studies do provide evidence that media exposure contributes to weight concerns among women.

Mills, Polivy, Herman, and Tiggemann (2002) compared the attitudinal and behavioral responses of dieters and nondieters to exposure to images showing bodies representing the thin ideal. Dieters who were exposed to thin-ideal images described their current and ideal body size as being smaller and increased their food intake; no such changes were seen either in dieters who were not exposed to these images or in nondieters regardless of whether or not they had seen the images. In addition to demonstrating a causal relationship between exposure to media images of idealized bodies and actual eating behaviors, this study suggested that dieting may increase vulnerability to the effects of media exposure.

Clinical Experimental Studies

Clinical experimental studies seek to understand the mechanisms that underlie the presence of eating disorders by comparing individuals diagnosed with an eating disorder with individuals without eating disorders. As such, they do not experimentally study the new onset of eating disorders but can reveal factors that may contribute to illness maintenance.

One example of this type of study involves experimentally manipulating a biological factor and measuring the effects on other variables (including biological and behavioral indices). K. A. Smith, Fairburn, and Cowen (1999) examined the effect of consuming a beverage that would manipulate blood tryptophan levels in 10 women clinically in remission from BN and in 12 healthy female controls. (Tryptophan is the biological precursor to serotonin,

whose hypothesized role in the etiology of eating disorders will be discussed in more detail in Chapter 8.) The women participated in two sessions: In one session, they consumed a nutritionally balanced beverage; in the other, they consumed a tryptophan-depleted beverage. Half the women in each group were randomly assigned to complete the trytophan session first. Participants provided self-ratings of mood and eating disorder cognitions (e.g., "feeling fat," having "urges to eat," and experiencing "weight concern") before and after consuming the beverage, and they recorded in a food diary any loss of control over eating in the 24 hours following each session. Consumption of a tryptophan-depleted beverage caused similar decreases in blood tryptophan levels in women remitted from BN and controls. However, in women remitted from BN, the drop in tryptophan concentration was associated with increased sadness and anxiety, increased eating disorder cognitions, and increased self-rated loss of control over food intake (even though no objectively large binge episodes occurred according to the 24-hour food diaries). Similar differences have been reported for women currently ill with BN compared with controls (Weltzin, Fernstrom, Fernstrom, Neuberger, & Kaye, 1995).

These and similar findings support a model for the development of bulimic symptoms in which dieting causes decreased tryptophan levels, which contribute to decreased serotonin function, which contributes to the emergence of binge-eating episodes (Wurtman & Wurtman, 1986). This model and relevant research findings and covered in more detail in Chapter 8.

Case Study: Emily

During Emily's intake evaluation, she was told about a research study that was being conducted at the university hospital. The study consisted of three assessments. The first assessment involved questionnaires and interviews that would require two to four hours to complete. The second assessment involved having blood drawn once before and twice after consuming a liquid meal. The third assessment involved a scan for bone mineral density. For participating, she would be paid $150 and she would receive the results of the bone scan. Because her doctor planned to have Emily get a bone scan, Emily decided that she might as well get paid to have the procedure done; in addition, she figured she would be contributing to science. (Results of Emily's bone scan are described in Chapter 8.) In reading the consent form for the study, she learned that the research was designed to answer three basic questions: First, is AN associated with increased levels of depression, a disturbed hormonal response to eating, and decreased bone density? Second, are levels of depression in AN related to hormonal disturbances? Third, are the hormonal disturbances related to bone mineral density? Participants in the study consisted of patients with AN and individuals with no history of physical or mental disorders (including any eating disorder).

Clinical treatment trials represent a second experimental study design—one with implications for understanding factors that may contribute to the maintenance of eating disorders. Treatment condition is the independent variable, and eating disorder remission is the dependent variable. If the study finds that certain factors contribute to remission of eating disorder symptoms, then inferences may be made about the contributions these factors make to eating disorders. For example, Fairburn, Jones, Peveler, Hope, and O'Connor (1993)

randomly assigned patients with BN to one of three treatment conditions: behavioral therapy, cognitive–behavioral therapy, and interpersonal therapy. (Therapy for eating disorders is discussed further in Chapter 9.) At the end of treatment, patients assigned to cognitive–behavioral therapy had significantly greater decreases in binge eating and purging that were sustained at 12-month follow-up than did patients in the other two treatment conditions. These results have been interpreted to support the role of cognitions and behaviors in the development of eating disorders (see Chapter 7).

A caveat to using clinical treatment studies to understand the causes of eating disorders is that curative factors are not necessarily related to causative factors. The classic example is that aspirin may be effective in treating a headache even though the headache was not caused by an "aspirin deficit."

Case Study: George

Because George was interested in the possibility of being treated with medication, he volunteered for a medication treatment study. A drug that had been successful in treating patients with type 2 diabetes was going to be examined in patients with night eating syndrome. George understood that he would be randomly assigned to receive either the active medication or a placebo and that neither he nor the physician managing his medicine would know what he was actually taking. George did not mind these limitations, because he knew that he had a 50/50 chance of getting the active medication and that at the conclusion of the study he would be offered the medication if it increased remission compared to the placebo. He also knew that the medication would be offered to all participants if dramatic positive results were seen before the study was meant to be concluded. The medication was associated with some minor side effects that were known to diminish over time. More serious reactions had developed in some instances; however, these severe reactions were very rare, and George would be monitored very closely throughout the study. He felt he had nothing to lose. Unlike most other weight loss programs George was considering, the treatment study was free—and besides, it might work. (Results of George's participation in the study are discussed in Chapter 9.)

Finally, a third type of clinical experimental study can demonstrate that manipulation of a factor can prevent the onset of an eating disorder. Becker, Bull, Schaumberg, Cauble, and Franco (2008) recruited 188 college women in sororities for a study examining whether reducing internalization of the thin ideal reduces risk for the development of eating disorders. Participants were randomly assigned to one of two conditions: either a dissonance-based intervention designed to help them reject the thin ideal by having them personally challenge the ideal or a control media literacy condition designed to improve awareness of the media's role in promoting the thin ideal without actually having women personally challenge this ideal. Results indicated that the dissonance-based intervention reduced internalization of the thin ideal, body dissatisfaction, dieting, and bulimic symptoms compared with baseline and compared with the media literacy control condition. These results suggest that using an intervention to experimentally manipulate internalization of the thin ideal caused changes in bulimic symptom levels. However, no significant effect in preventing diagnosable eating disorders was demonstrated, possibly because of the small size of this study. Chapter 10 reviews this and other prevention studies in more detail.

A concern in experimental studies is that the very controls used to ensure that causal conclusions are sound reduce the **ecological validity** of the results, that is, their bearing for real life. For example, in studies of the association between dieting and binge eating, some dieting participants may be randomized into a condition in which they are forced to break their diets. In real life, however, individuals who diet may respond differently to the numerous challenges they face to break their diets, and it is difficult to account for such individual differences within experimental designs. Similarly, exposure to the thin ideal may demonstrably cause body dissatisfaction in an experiment, but in real life only a very small proportion of individuals with body dissatisfaction will go on to develop eating disorders. Thus the phenomena that can be demonstrated to be caused in experimental studies support links within a causal chain but rarely connect these causal factors to the actual emergence of eating disorders.

Naturalistic Investigations

For certain causal factors, study designs that take advantage of naturally occurring or historical events are possible. Such **naturalistic investigations** tend to be very powerful, because they allow observation of causal relationships in the onset of eating disorders. For example, Polivy, Zeitlin, Herman, and Beal (1994) compared prevalence of binge eating in World War II combat veterans and prisoners of war in German concentration camps. If binge eating was simply a result of increased stress, then elevated prevalence would be expected in both groups. If it was a specific consequence of starvation, then elevated binging would be expected only among former prisoners of war. Results supported an association between starvation and subsequent binge eating.

Adoption and Twin Studies

Both twin and adoption studies present the opportunity to evaluate the effects of genes and of environmental factors. Adoption studies compare rates of a disorder in adopted children with those in their biological and adoptive relatives. If greater similarity is found between adoptive parents and children than between the children and their biological relatives, then an effect of the rearing environment on disorder risk is inferred. If greater similarity is found between the biological parents and children, then an effect of genes is inferred. If rates of a disorder are elevated only among children whose adopted and biological parents are both affected by the disorder, then an interaction between genes and environment is inferred.

Twin studies use the natural occurrence of **monozygotic** (MZ) and **dizygotic** (DZ) **twins** to compare the contributions of genes and environment to the risk of developing a disorder. Monozygotic twins share approximately 100% of their genes, while DZ twins share, on average, 50% of their genes. When a pair of twins is reared together, they fully share environmental factors such as parenting styles, socioeconomic status, and neighborhoods. Twin studies measure the degree of similarity within twin pairs for occurrence of the disorder **(twin concordance).** If MZ twins reared together and DZ twins reared together show similar twin concordance, then the environment plays a larger role in disorder risk compared to genes. If the MZ twins show greater concordance than the DZ twins do, then

genes contribute to disorder risk. Results from adoption and twin studies support significant genetic contributions to eating disorders and are reviewed in Chapter 8.

Cross-Cultural and Epidemiological Studies

Finally, examinations of the epidemiology of eating disorders both cross-historically and cross-culturally (reviewed in Chapter 3) provide interesting insights into etiology. Rates of eating disorders increased during the 20th century and tend to be higher in Western industrialized nations. These observations point to societal features of these nations as contributing to eating disorder risk. However, based on the information reviewed in Chapter 3, we have good reason to believe that unique etiological factors for self-starvation syndromes and syndromes marked by binge eating help to explain their distinct patterns of cross-cultural and cross-historical representation. These findings suggest that risk factors for BN, BED, and purging disorder may be more limited to specific cultural and historical contexts than are risk factors for AN. Moreover, hypotheses concerning associations between media ideals of thinness and dissatisfaction with weight or shape (reviewed in Chapter 5) may be more relevant for understanding the causes of BN than AN.

Although naturalistic observations often provide strong evidence of causal factors that are directly related to the emergence of eating disorders, they often do not point to specific risk factors. For example, concluding that genes contribute to the etiology of AN (Klump, Miller, Keel, McGue, & Iacono, 2001) does not specify which genes increase the risk or what these genes do. Similarly, recognizing that something in modern Western culture increases the risk for BN does not narrow the list of potential culprits.

Conclusion

Several methodologies exist for examining risk factors for eating disorders. Because of ethical considerations and logistical constraints on time and financial resources, the majority of risk factor research examines correlations between posited risk factors and disordered eating attitudes and behavior. Such studies operate on the logic that if a causal relationship exists between two variables, they will be correlated. However, the reverse is not true: Correlation does not prove causation. Thus these studies do not directly examine causal associations. Those studies that do examine causal relationships do not typically evaluate actual eating disorders.

Despite these limitations, certain risk factors emerge across studies. Stice (2002) conducted a meta-analysis of risk as well as maintenance factors in "eating pathology" (not eating disorders). He included only prospective and experimental studies and differentiated between risk factors (results from prospective studies) and causal risk factors (results from experimental studies). Stice concluded that body dissatisfaction, negative affect, perfectionism, impulsivity, and substance use were risk factors for eating pathology. Causal risk factors included perceived pressure to be thin and internalization of the thin ideal. The different results for risk and causal risk factors were due largely to the types of studies that have been conducted rather than to demonstrated differences in these variables' impact on disordered eating attitudes and behaviors. Based on prevalence differences among AN, BN, and BED

(see Chapter 3), studies utilizing composite measures of disordered eating attitudes and behaviors mostly provide predictors of bulimic symptoms (Stice, 2002).

Chapters 5 and 6 will review evidence for the role of social factors in the etiology of eating disorders. Chapter 7 will review psychological factors such as personality and cognitive—affective processes. Rounding out the examination of etiology, Chapter 8 will review biological factors in detail. Although social, psychological, and biological factors are reviewed in separate chapters, they should not be viewed as operating independently or as representing competing models, because no single factor causes all eating disorders. Sociocultural factors cannot be fully responsible for eating disorders, since most women in societies that idealize thinness do not suffer from eating disorders. Similarly, genes cannot fully cause eating disorders because concordance is not 1.0 in MZ twins (that is, genetically identical twins can differ on presence versus absence of an eating disorder). Rather, several factors, operating at different levels, influence vulnerability to developing an eating disorder.

Key Terms

Analog studies
Correlation
Diathesis
Dizygotic twins
Ecological validity
Experimental studies
Interoceptive awareness
Longitudinal studies
Monozygotic twins
Naturalistic investigations
Nuisance variable
Retrospective recall bias
Socioeconomic status
Twin concordance

5

Body Image, Dieting, and Eating Disorders

Can We Blame the Media?

Body dissatisfaction is a robust risk factor for eating disorders (see Chapter 4). But what leads to body dissatisfaction? This chapter explores one major influence: the role of culture in promoting a thin ideal and denigrating fatness that leaves many individuals, particularly girls, feeling dissatisfied with their weight and places them on the path to attempt to control their weight through dieting and disordered eating.

Cultural context shapes beliefs and values, and beliefs and values influence behaviors. Cultural values can be communicated by families, religions, schools, peers, and, in modern culture, mass media. A study examined the influence of Western television on the beliefs, values, and behaviors of adolescent girls in Fiji (A. E. Becker, Burwell, Herzog, Hamburg, & Gilman, 2002). Baseline assessments were conducted within one month of the introduction of televisions into the homes of a cohort of adolescent girls, and assessments were conducted three years later with a new cohort. At baseline, girls reported low levels of body dissatisfaction, dieting, and purging and placed little importance on being thin. These findings were consistent with a cultural context that celebrated feeding as an expression of familial care. Within traditional Fijian culture, thinness has been associated with lacking love and the care of family (A. E. Becker, 1995). Three years later, however, the new cohort of Fijian girls expressed beliefs that success, wealth, and independence were related to being thin and desired to be thin. At baseline 0% of girls reported engaging in self-induced vomiting to control weight, compared with 11% of girls three years later. The percentage of girls with elevated scores on a measure of disordered eating more than doubled in the same period. Thus this study provides strong evidence that the exportation of Western cultural values through television may be accompanied by the exportation of eating disorders. That association in turn provides strong evidence for the role of cultural factors in the emergence of eating pathology.

The Cultural Ideal of Thinness

In describing BN as a "new disorder," Russell (1997) attributed its emergence to the "modern 'cult of thinness' " (p. 23). This explanation represents a model for the development of eating disorders in which societal overvaluation of thinness for women, actively promoted throughout print, television, and film media, causes eating disorders. According to this model, the idealization of thinness leads girls and women to become dissatisfied with their own body weights and shapes and to desire thinner bodies. This desire leads to dieting. If dieting leads to significant and medically dangerous weight loss, then the individual has developed AN. If dieting leads to binge eating, purging may emerge in an effort to avoid weight gain, and then the individual has BN. Alternatively, purging may develop in the absence of binge eating and without leading to significant weight loss, in which case the individual has purging disorder. This model proposes that women want thinner bodies because they see very thin models of beauty celebrated. Thus according to this model the media play a causal role in the development of body image disturbance—a core feature of AN and BN as defined by the *DSM*.

Epidemiological data seem to support this view. As reviewed in Chapter 3, the incidence of AN and BN has increased over recent decades. Coinciding with increasing rates has been a change in beauty ideals for women. Garner, Garfinkel, Schwartz, and Thompson (1980) evaluated trends from 1959 to 1978 in three representations of feminine beauty: articles in magazines marketed specifically to women, *Playboy* centerfold models, and Miss America Pageant contestants and winners. In six popular women's magazines (*Harper's Bazaar, Vogue, McCall's, Good Housekeeping, Ladies' Home Journal,* and *Woman's Day*), the average number of articles on weight loss diets increased from 1.4 to 2.5 per month from the first to the second decade of observation. The figures of *Playboy* centerfold models shifted from a voluptuous hourglass to a trimmer "tubular" look with a significantly lower waist-to-hip ratio. The weights of *Playboy* centerfold models as a percentage of expected weight also decreased significantly over the 20-year period.

Finally, Garner et al. (1980) found that Miss America contestants became increasingly thin relative to their expected weight. Across the period of observation, Miss America contestants and winners weighed less than what would be expected for their height and age (as did *Playboy* centerfold models). However, until 1968, contestants weighed on average at least 86% of their expected weight (see Figure 5.1). After 1968, their average weights fell below 86% of expected (except in 1971). The relationship between the contestants' weights and the winner's weight was inconsistent up to 1970. After 1970, all winners had a lower weight for height than the average for contestants the same year (except in 1977 when the values were nearly identical). From 1970 on, all Miss America winners, without exception, weighed less than 85% of what would be expected for their height. This pattern indicates an alarming ideal for feminine beauty, considering that weighing less than 85% of what is expected for one's height was the threshold for diagnosing AN in the *DSM-IV* (American Psychiatric Association, 1994). Miss America contestants and winners became increasingly thinner relative to population norms for decades following the 1980 article (Owen & Laurel-Seller, 2000; Rubinstein & Caballero, 2000; Wiseman, Gray, Mosimann, &

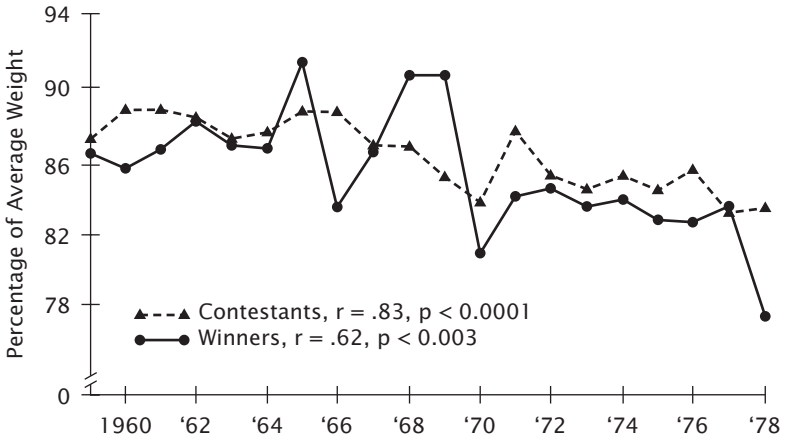


FIGURE 5.1 Percentage of average weight for Miss America Pageant contestants and winners over 20 years. Source: D. M., Garfinkel, P. E., Schwartz,D., & Thompson, M. Cultural expectations of thinness in women. Psychological Reports, 1980, 47, 483–491. © Psychological Reports 1980. Reprinted by permission of Ammons Scientific Ltd.; permission conveyed through Copyright Clearance Center, Inc.

Ahrens, 1992), and then trends could not be tracked because data on contestants' heights and weights were no longer made publicly available.

Patients with eating disorders, like Jean and Valerie, often point to such ideals in explaining why they are so dissatisfied with their own bodies.

Case Study: Jean

When Jean was asked why it was important that she still fit into the same-size jeans she had worn in high school, she replied that it was the only way she would know that she wasn't getting fat. Although at 5 feet 4 inches she was taller than the average Korean woman, this only contributed to her feeling "big." When she saw women in magazines, she never really saw herself. Models were tall, thin, and blonde or they were very exotic looking. Jean felt that she was still short enough that a very small amount of extra weight would make her look very fat, particularly because she tended to carry weight in her face. In fact, the feature she hated most about herself was her "big, round face." She knew that she would never have high cheekbones like the women in movies or TV shows, but she felt that the least she could do was to have a nice, heart-shaped face. She felt she was too Asian looking to be an American beauty and too big and "dumpy" to be an Asian beauty.

Echoing observations from Chapter 3, being Asian does not protect Jean from the thin ideal. Instead, media images of beauty only increase her sense that beauty is everything she is not. However, of all the attributes she does not possess, a thin body seems attainable to her.

Case Study: Valerie

In response to the email Valerie sent, she received information about treatment resources in her area. She sought help from a therapist who specialized in eating disorders in Sydney, Australia, approximately 45 minutes from her home. During their sessions, the therapist would encourage Valerie to stop purging; Valerie worried, however, that if she stopped she

would gain weight and become fat. The therapist reassured her that she was unlikely to become fat, because she did not have a family history of obesity and had not been overweight before she started purging. The therapist agreed that her weight likely would increase if she stopped purging, because purging causes dehydration. The therapist asked Valerie whether nine pounds of mostly water was really worth the damage she was doing to her body and the lies she had to tell family and friends to maintain her eating disorder. Valerie responded: "I don't know. I know I sound nutso, but I just feel better about myself when I'm thinner."

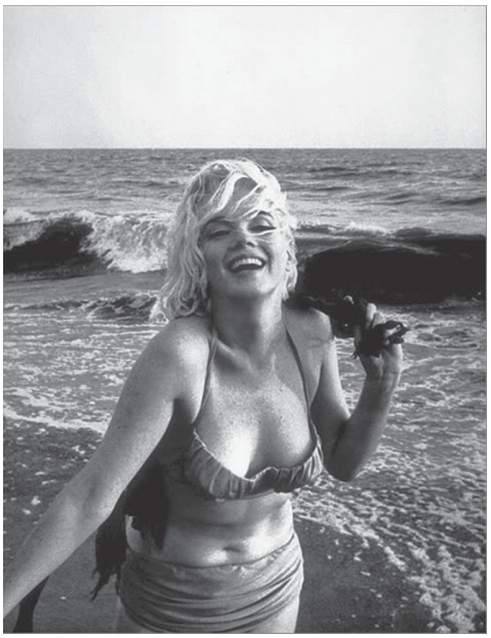
For Valerie, a fear of gaining nine pounds maintains her purging behavior. Although she is not striving to be emaciated, she has a very precise definition of an acceptable weight for herself and a strong connection between being thin and her self-worth.

Why Does Thin = Good?

Several explanations exist for the current emphasis on thinness in culture. Historian Joan Jacobs Brumberg (1989) noted an association between thinness as an aesthetic ideal and periods in which women have been encouraged to adopt responsibilities outside the home and beyond their roles as wives and mothers. For example, the Roaring Twenties represented a time of unprecedented freedom for women following ratification of the 19th Amendment, which gave American women the right to vote. In this period, hemlines rose and flat-chested flappers were in vogue. The flappers' preadolescent figures represented a direct contrast to the hourglass shape produced by turn-of-the-century Victorian corsets. During the Great Depression of the 1930s, many women worked outside the home to help feed their families, and during World War II many women were required to take jobs traditionally held by men. The ideal of feminine beauty during this period was thin (as exemplified by actresses Katharine Hepburn and Claudette Colbert). After the end of World War II and the return of soldiers to home and jobs, women were expected to return to their roles as mothers and wives. Ideal figures returned to an hourglass shape that emphasized the presence of breasts and hips (like that of actress Jane Russell). Figure 5.2a shows the iconic sex symbol of this era, Marilyn Monroe, in a bathing suit; her figure contrasts sharply with that of Cameron Diaz four decades later in Figure 5.2b. The next phase of the revolution for women's independence came during the 1960s with the development of birth control pills, the sexual revolution, and the emergence of the "second-wave" feminist movement. Coinciding with demands for women's rights and freedom came an ideal of thinness (exemplified by the model Twiggy) that seemed to demand that women be free of their traditionally feminine shapes.

Hsu (1990) argued that financial prosperity after the Industrial Revolution led to an abundance of food that disrupted the traditional positive correlation between wealth and weight (Nasser, 1988). In societies where wealth is associated with fatness, Hsu noted, beauty is associated with fatness as well. Conversely, in societies where wealth is associated with thinness, so is beauty. In industrialized nations, there is a significant negative association between socioeconomic status and weight (Morgenstern, Sargent, & Hanewinkel, 2009; Rahkonen, Lundberg, Lahelma, & Huuhka, 1998; Stunkard, 1996; Wardle, Waller, & Jarvis, 2002): Obesity is overrepresented among the poor and uneducated, and thinness

(a)



(b)

FIGURE 5.2 (a): Marilyn Monroe. Source: George Barris, 1962. Licensed under Public Domain via Wikimedia Commons. (b): Cameron Diaz in Charlie's Angels: Full Throttle, 2003.

is more common among the wealthy and well educated. As obesity has become increasingly prevalent over recent decades, however, the association between socioeconomic status and obesity has weakened (Zhang & Wang, 2004). According to the Centers for Disease Control (Ogden, Lamb, Carroll, & Flegal, 2010), this association depends on sex, race, and ethnicity: Obesity is less common among men and women with a college education. In addition, for women, higher income is associated with lower risk for obesity. In terms of Hsu's (1990) theory, the particularly strong association between thinness and wealth for women may explain the overall greater prevalence of eating disorders (particularly BN) in women in industrialized than in nonindustrialized nations and why rates of eating pathology increase as cultures become more Westernized (see Chapter 3). In contrast to patterns for women, both Black and Hispanic men show a positive association between obesity and income.

Kilbourne (1987) has argued that a consumer-driven economy requires the constant invigoration of buying. The sole purpose of the multibillion-dollar advertising business is to encourage people to spend money. One highly successful way to do this is to convince people that there is something they *need* to have. However, basic human needs remain limited—food, shelter, and affiliation. Thus, new "needs" are built upon these basic needs, and the need for affiliation is a particularly good base for generating new needs. Although being beautiful is not a prerequisite for living or procreating, advertisements and media can generate a need to subscribe to certain beauty ideals by creating the impression that one is deficient in some way that will limit one's prospects for affiliation. Establishing an unattainable physical ideal creates a constant market for products. This commercialization of beauty treats women's bodies as objects—a process referred to as **objectification**, in which women's value is reduced to what they look like rather than what they can do. According to

Kilbourne (1987), the presence of body dissatisfaction in a majority of adolescent girls and young adult women is necessary to turn those same women into good consumers. Notably, the quests for thicker eyelashes, luxurious hair, or whiter teeth do not appear to have significant psychiatric consequences. However, the quest for a perfect body may. Alternatively, businesses may use young, thin models because using older or heavier models results in a drop in sales. When a magazine presents a less-than-"perfect" woman on its cover, publishers may worry that the very women whose self-image should be helped by more diverse representations of beauty will not purchase the magazine. Thus such efforts to promote a healthier ideal may feel both pointless and self-punishing.

Recent decades have seen an increasingly lean and muscular ideal for men portrayed through popular media. As reviewed in Chapter 3, Pope et al. (1999) examined action figures marketed to prepubescent boys (much as the unrealistically tall and thin Barbie doll is marketed to prepubescent girls) and found significant increases in the chest-to-waist ratio over time. Similarly, Figure 5.3 provides a "then and now" comparison of actors portraying Superman. From 1948 to 2013, Superman's physique has moved toward greater muscular definition throughout the shoulders, arms, chest, and abdomen. Pope et al. argued that unrealistically muscular ideals contribute to excessive exercise and steroid abuse in men, just as unrealistically thin ideals contribute to eating disorders in women. Although the two ideals lead one gender to seek to reduce overall body size and the other to seek to increase overall body size, both ideals promote attempts to reduce body fat that contribute to the multibillion-dollar weight control industry. The quests for "six-pack abs" and a hairless chest to better display muscle definition have increased male consumption of weight loss and hair removal products, and internalization of the modern male ideal has been linked with eating pathology in men (Tylka, 2011).

This line of investigation suggests that the media play a proactive role in the conception of aesthetic ideals rather than simply following public preferences. It is difficult to believe that preadolescent boys would refuse to purchase action toys on the basis of

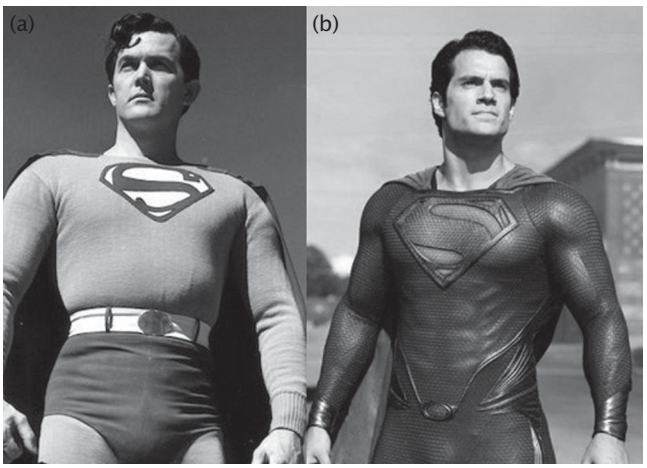


FIGURE 5.3 In 1948's *Superman*, the lead character had notably less muscle definition (a) than in the 2013 release of *Man of Steel* (b).

normal chest-to-waist ratios. Similarly, the fact that men provided a strong market for purchasing magazines before the emergence of the lean, muscular ideal reduces the plausibility of magazine editors simply responding to consumer markets rather than actively shaping them.

A final possible explanation for why "thin is good" emerges from the health risks associated with obesity. According to this view, thin is beautiful because it represents health and youth—qualities associated with increased reproductive fitness. However, this explanation does not seem particularly compelling, because the thin ideal for women promotes a figure that is associated with loss of reproductive function (i.e., amenorrhea). Indeed, there is more room for the body to go above the expected weight for height and remain healthy than there is room for the body to go below that weight while staying healthy. For example, at 5 feet 4 inches, a woman with an average frame might be expected to weigh 120 pounds (Bray, 1986). The BMI for a woman who is 20% above this expected weight would be 24.8 kg/m^2—a value considered to be within the normal range. However, a woman of that same height whose weight falls 20% below the expected level would have a BMI of 16.5 kg/m^2—a value that places her in the moderate range of severity for AN and might necessitate inpatient treatment. The extent to which overweight is denigrated appears to go far beyond concerns about the associated health consequences and may reflect an elaboration of the idealization of thinness, as discussed next.

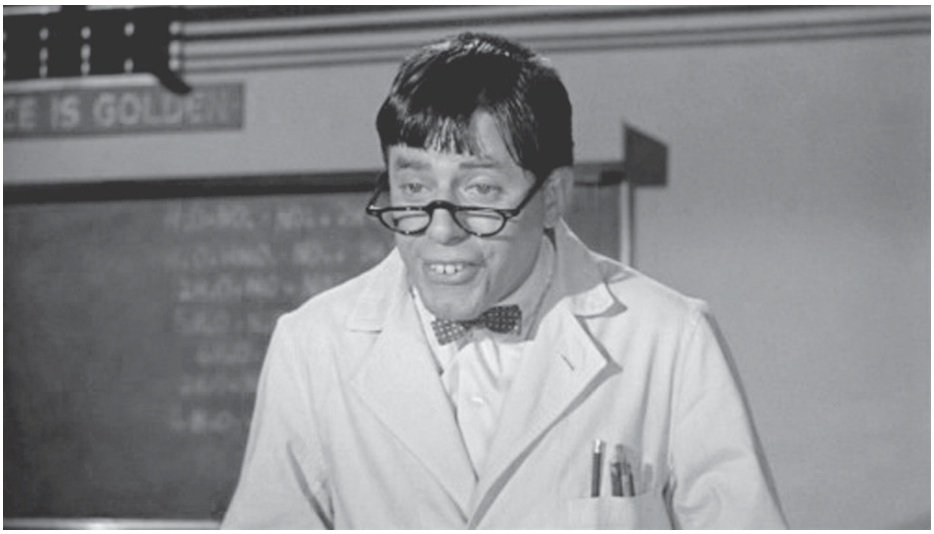
Societal Denigration of Overweight and Obesity

In addition to valuing thinness, Western culture denigrates fatness. Not only is it good to be thin; it is bad to be fat. Jokes about fat people are tolerated in a society that eschews most other forms of physical prejudice. Indeed, in Eddie Murphy's remake of the Jerry Lewis movie *The Nutty Professor*, obesity rather than being a nerd became the central joke of the film (see Figure 5.4). Fat is viewed as bad in part because obesity is associated with significant health risks; however, when we look at attitudes toward individuals who are overweight compared with those toward individuals with other health risks, it becomes clear that society imbues weight with moral qualities. For our case study Emily, fat means a loss of control and weakness; for George, difficulty losing weight represents a character flaw.

Case Study: Emily

Emily denied having any interest in depictions of beauty in magazines, on TV, or in movies. For entertainment, she read novels, and she particularly liked Russian novels. She adamantly refused to accept the idea that her desire to be thin was related to any prepackaged media ideal of beauty. Instead, she pointed to the health risks associated with obesity and to the contrast between the culturally universal preference for symmetry of physical features and her own "asymmetrical" body. Moreover, Emily saw herself as striving for an ideal beyond any pedestrian Hollywood image. She was seeking balance in the food she consumed, in her bodily dimensions, and in her value system. Emily repeatedly stated that she would happily gain weight if she could just do so on her upper body. For these reasons, she did not

(a)



(b)

FIGURE 5.4 (a): Jerry Lewis in The Nutty Professor, 1963. (b): Eddie Murphy in The Nutty Professor, 1996.

believe that she had an eating disorder: "Girls with eating disorders," she said, "just want to be skinny to look like fashion models," and this was not how Emily saw herself. Despite her apparent indifference to being thin for the sake of thinness, Emily reported an intense fear of becoming fat. She stated that she would rather die than lose control over her eating and her body. When asked what she thought of obese people, Emily said that she felt sorry for those who were obese for medical reasons, because there was no way to tell why a person was obese just by looking. However, people who were obese because they could not stop eating repulsed her; they represented everything that she feared she might become.

Case Study: George

Before entering the treatment study for night eating syndrome, George had tried many, many weight loss diets. He and his wife had gone on Weight Watchers and followed the organization's point system. He had tried Nutrisystem, from which he ordered prepackaged meals. He used the Optifast program, in which he was supposed to consume a liquid shake for two meals and then eat a normal dinner. He tried the Atkins and then the South Beach diets, in which he could eat as much as he wanted as long as he avoided carbohydrates. Nothing ever worked, and each time that he tried and failed he wondered what was wrong with him that he couldn't get the results promised in the testimonials. Every diet failure left him feeling lazy and weak—and still hungry.

Risk factor studies reviewed in Chapter 4 indicated that individuals with BN and BED were more likely than controls to have been overweight as children and to have been subject to negative comments about their weight (Fairburn et al., 1997, 1998; Striegel-Moore et al., 2005). At a cultural level, fat persons are viewed as lazy, stupid, lonely, inept, weak, and dependent (Dejong & Kleck, 1986; Puhl & Heuer, 2010; Tiggemann & Rothblum, 1997). The belief that people can control their weight contributes to a tendency to blame overweight individuals for being overweight (Crandall & Martinez, 1996; Crandall et al., 2001; Paxton & Sculthorpe, 1999). Although Emily acknowledges that there is no way to determine why people are overweight by looking at them, she feels disgust toward obese people who cannot control their eating. Just as refraining from the temptation to eat fattening food is perceived as virtuous, overweight seems to mark moral weakness by indicating an inability to resist temptation. Women tend to hold more negative stereotypes of the overweight and obese than do men (Tiggemann & Rothblum, 1988), and research participants view obese women more critically than they view obese men (M. B. Harris, Walters, & Waschull, 1991).

These attitudes appear to be somewhat automatic (Grover, Keel, & Mitchell, 2003; Teachman & Brownell, 2001). Alarmingly, negative attitudes toward overweight are held by people regardless of their own weight (Grover et al., 2003; Latner, Stunkard, & Wilson, 2005), as seen in George's case and by the medical professionals responsible for the care of overweight individuals (Teachman & Brownell, 2001; Puhl & Heuer, 2010). Compared with normal or underweight patients, patients with eating disorders who were overweight (defined by a BMI > 25 m/kg^2) elicited more fear, more emotional distance, more confrontation, less caring, and less sense of responsibility for therapy from their therapists (Toman, 2002). Unlike the reaction to stereotypes of other aspects of physical appearance (e.g., race), there has been little attempt to challenge, alter, or censor these beliefs (Puhl & Heuer, 2010). Further, research supports that attempts to reduce antifat prejudice are far less powerful in altering attitudes than are typical messages that blame fat people for being overweight (Teachman, Gapinski, Brownell, Rawlins, & Jeyaram, 2003).

Longitudinal research suggests that antifat bias and active discrimination against individuals who are overweight may contribute to the association between obesity and

socioeconomic status described earlier in the chapter (Crocker & Major, 1989; Puhl & Heuer, 2010; Roehling, 1999; Rothblum, Brand, Miller, & Oetjen, 1990). The obese are less likely to be hired (Roe & Eickwort, 1976) and less likely to be promoted (Larkin & Pines, 1979) than are normal-weight individuals with equal qualifications. An additional explanation for the association between obesity and socioeconomic status comes from a study that found more fast-food restaurants in poor than in wealthy neighborhoods (Reidpath, Burns, Garrard, Mahoney, & Townsend, 2002). Because fast food that is high in fat and calories is inexpensive, it may form a higher proportion of the dietary intake of poor persons. Notably, these two explanations are not mutually exclusive. It is possible both that being overweight contributes to lower socioeconomic status and that lower socioeconomic status contributes to the risk of obesity.

Antifat prejudice can be found in preschool children (Spiel, Paxton, & Yager, 2012; Worobey & Worobey, 2014). Girls aged 3.5–5 years who were given dolls representing thin, average-weight, and heavy bodies rated the thin dolls as having the most positive characteristics and the "fat" dolls as having the most negative characteristics (Worobey & Worobey, 2014). Similarly, preadolescent children rate their overweight peers as less likable and as less desirable playmates (Strauss, Smith, Frame, & Forehand, 1985). Weight stigma does not appear to be a simple rejection of peers who look different (Puhl & Latner, 2007). Children 10–11 years old show greater prejudice against overweight children than against children with other physical differences, such as being in a wheelchair or having a disfigured face (Latner & Stunkard, 2003; Richardson, Goodman, Hastorf, & Dornbusch, 1961). More alarmingly, as the prevalence of obesity among children has increased, weight stigma among children has increased as well (Latner & Stunkard, 2003). Among elementary school children, the nature of weight biases appears to depend on the gender of the child being judged. Both boys and girls were found to view pictures of overweight children less positively than they did pictures of normal-weight or thin children (Kraig & Keel, 2001). Boys and girls had significantly more positive opinions of pictures of thin girls than of pictures of either normal-weight or overweight girls. Boys viewed pictures of thin and normal-weight boys similarly but held overweight boys in low regard. These findings suggest that thinness may be the salient property for judging the "goodness" of girls, whereas for judging boys, it may be important to simply be not fat. This difference in standards would explain gender differences in the drive for thinness and in body dissatisfaction.

Ironically, in direct contrast to the increasing value accorded to thinness and the increasing prejudice against fatness, the U.S. population has grown increasingly overweight (Flegal, Carroll, Ogden, & Johnson, 2002; Flegal & Troiano, 2000; Kuczmarski, Flegal, Campbell, & Johnson, 1994). As described in Chapter 3, recent data suggest that this trend may be leveling off over recent years (Flegal et al., 2010). Even so, the prevalence of obesity reached 34% of the U.S. population in 2007–2008 (Flegal et al., 2010) and remained at this level in 2011–2012 (Ogden et al., 2014). Given the negative social consequences of being overweight, it is not surprising that starting a diet or an exercise regimen has become a normative New Year's resolution in Western societies. The large and growing discrepancy between ideal and actual weights for women would be expected to produce significant body image disturbance.

Body Image

Before considering what studies show about the impact of the societal idealization of thinness on body image, it is important to discuss what is meant by body image. Cash and Deagle (1997) have characterized body image as comprising facets of perception, cognition, and affect. The perception facet involves both seeing and feeling bodily dimensions such as weight and shape (e.g., "I look fat" or "I feel fat"), independently of actual weight or shape. Disturbance in the perception of weight and shape is included as a possible symptom of AN (see Chapter 1) but not BN, even though studies have not found significant differences in perceptual disturbances between the two disorders (Cash & Deagle, 1997). Notably, perception does not require any particular judgment. A person could perceive herself as fat without making an evaluative statement about what this means.

The cognition facet of body image involves evaluations and thoughts related to body size (e.g., "I weigh more than I should" or "I should lose weight"). As with perception, cognitions need not be related to actual weight. Women with eating disorders report greater body dissatisfaction than do women without eating disorders, even though weight is lower in AN and does not differ between weight-matched controls and women with BN (Keel, Haedt, & Edler, 2005), purging disorder (Keel, Haedt, & Edler, 2005), or BED (Lewer, Nasrawi, Schroeder, & Vocks, 2016). Indeed, the factors that best differentiate between women with and without eating disorders are levels of concern about weight and shape and the importance given to weight and shape in self-evaluation The undue influence of weight and shape on self-evaluation is included as a possible symptom for AN and as a required symptom for BN (see Chapter 1).

The affect facet of body image involves emotions related to weight or shape (e.g., "I must be thin to feel happy" or "When I am unhappy I feel fat"). Longitudinal research suggests that the link between affect and cognitive features of body image may be bidirectional, in which body dissatisfaction contributes to distress and general distress is funneled into body dissatisfaction (Keel, Mitchell, Davis, & Crow, 2001). This association contributes to why body image was linked to negative affectivity in the Leon et al. (1999) study described in Chapter 4.

An association between societal aesthetic ideals and body image among women has been demonstrated in a number of studies (Grabe, Ward, & Hyde, 2008; Groesz, Levine, & Murnen, 2002; Hausenblas et al., 2013). In a review of 141 studies examining associations between media use (including watching television, seeing movies, and reading magazines) and body image in females, Grabe et al. (2008) found a consistent association between media exposure to the thin ideal and body dissatisfaction, internalization of the thin ideal, and disordered eating attitudes and beliefs. For example, Tiggemann and Pickering (1996) found a significant association among adolescent girls between body dissatisfaction and time spent watching music videos, soap operas, and movies. As noted in Chapter 4, a drawback of this and other correlational studies is their inability to determine the direction of association between media images and body dissatisfaction. Potentially, girls who are more concerned with their weight are more likely to spend time seeking out information that reinforces this concern (Joiner, 1999).

In addition, the presence of body image concerns may affect how girls perceive media images. For example, King, Touyz, and Charles (2000) investigated the impact of body shape concerns on perceptions of celebrities. Photographs of thin and heavy female celebrities were altered to produce six distorted photographs (three in which the celebrity appeared thinner than she actually was and three in which she appeared heavier). Participants were asked to choose the photograph that they believed reflected the celebrity's true size. For thin celebrities, college women with higher levels of body concern selected photographs showing thinner-than-actual bodies, whereas women with lower levels of body concern selected more-accurate photographs. This finding suggests that body image concerns contribute to a biased perception of media images as pushing an even thinner ideal than they actually present. Of interest, college women, regardless of their level of body shape concern, selected photographs of heavy celebrities showing heavier-than-actual bodies as the true photographs. This finding suggests that when media images do not portray the thin ideal, women may exaggerate the deviation from that ideal in their perceptions.

Experimental studies, in which participants can be randomly assigned to media exposure conditions, provide the best evidence of a causal link between media images and body image. Among the 141 studies reviewed by Grabe et al. (2008), 80 employed experimental designs and supported significant associations between media exposure and increased body dissatisfaction, thin-ideal internalization, and disordered eating attitudes and behaviors. In a subsequent review of experimental studies examining the effect of media exposure on body dissatisfaction, Hausenblas and colleagues (2013) reported that the effects were significantly more pronounced among individuals who already showed greater internalization of the thin ideal and had elevated weight (which would produce a greater discrepancy between media images of beauty and personal appearance). Thus cultural ideals of beauty promoted by the media can and do influence the level of body dissatisfaction experienced by girls and women, and internalization of those ideals affects girls' vulnerability to those cultural messages.

Gender differences in body image concerns represent one of the most consistent findings in body image research. Females in elementary school, middle school, high school, college, and beyond report greater dissatisfaction with their weight and shape and a greater desire to lose weight than do males (Barnett, Keel, & Conoscenti, 2001; Keel, Fulkerson, et al., 1997; Keel, Baxter, et al., 2007; Leon, Fulkerson, Perry, & Cudeck, 1993; Striegel-Moore et al., 2009). Associations between actual weight and body dissatisfaction also differ between girls and boys. Among girls aged 9–18 years, those above the 50th percentile for BMI reported greater body dissatisfaction than did girls below the 50th percentile (Calzo et al., 2012). In contrast, among boys the same age, those above the 75th percentile and those below the 10th percentile expressed the greatest body dissatisfaction (Calzo et al., 2012). As girls aged, they became increasingly dissatisfied with their weight and shape such that girls with healthy weights began to express body dissatisfaction. Increased body dissatisfaction in women helps to explain gender discrepancies in rates of eating disorders if body image disturbance translates into disordered eating behaviors. The next section discusses findings concerning one behavioral consequence of a desire to lose weight—dieting.

Dieting

Given that the idealized female body is much thinner than most women's bodies, it is not surprising that a majority of girls and women have attempted to lose weight by dieting (Neumark-Sztainer, Wall, Larson, Eisenberg, & Loth, 2011). Reflecting gender differences in body image dissatisfaction, males are less likely to report dieting to lose weight than are females (Keel, Baxter, et al., 2007; Neumark-Sztainer et al., 2011). High school girls attempting to lose weight were, on average, normal weight, and high school boys attempting to lose weight were, on average, overweight (J. C. Rosen, Gross, & Vara, 1987). Moreover, the percentage of college students who want to lose weight exceeds the percentage who describe themselves as overweight (Heatherton et al., 1995). Further, the percentage who describe themselves as overweight far exceeds the percentage who are actually overweight (Heatherton et al., 1995). Thus many people who are normal weight—even many who view their weight as normal—attempt to lose weight by dieting.

The physiology of weight control is a balance between energy intake (eating) and energy expenditure (**resting metabolic rate** plus activity level). So weight loss should be eminently attainable by reducing food intake and increasing exercise. From organized programs to commercially available liquid diets to specific food regimens (the Zone diet, high-protein diets, etc.), the multibillion-dollar weight loss industry makes promises that are accepted each year by individuals attempting to attain the "right" body. The total amount of money spent on weight loss has increased steadily over recent years, without evidence that rates of obesity are declining (Engle, 2014). Although initial weight loss appears to be attainable with numerous weight loss programs, maintaining the weight loss is much more difficult.

As noted in Chapter 4, one possible explanation for the failure of most diets to produce lasting weight loss is that dieting may contribute to binge eating, which then contributes to weight gain. In the first half of the 20th century, Ancel Keys and colleagues (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950) conducted a study of the consequences of starvation and examined methods for safe refeeding in preparation for the return of prisoners of war after World War II. Physically and psychologically healthy young men who had been conscientious objectors to the war were recruited for the study. Participants were placed on a diet and exercise regimen designed to reduce their weight to 75% of their previous weight. The results of this study (known as both the Conscientious Objectors Study and the Minnesota Starvation Study) have provided important insights concerning the consequences of food restriction and significant weight loss.

Initially, participants lost weight quickly. However, as the study continued, weight loss slowed and analyses of the men's physiological functioning revealed a significant drop in their basal metabolic rate, which accounts for approximately 70–75% of energy expenditure (depending on age, gender, body weight, muscle mass, and activity level). Decreased basal metabolic rate was associated with slowed pulse, decreased respiration, lowered blood pressure, and decreased temperature, among other physical changes (all of which are also noted physiological consequences of AN—Chapter 8 provides a detailed description of medical consequences of AN). By diminishing the amount of energy expended in maintaining the body, the men became more efficient in their use of calories. In addition to these physiological

changes, the men experienced increases in depression, introversion, apathy, and food-related obsessions and rituals. When the diet ended, 29% of the men reported beginning binge-eating episodes; that is, they had episodes in which they consumed large amounts of food and experienced a loss of control over their eating. For some men, this overconsumption led to vomiting, and one man required hospitalization after excessive food intake.

These observations showed evidence of physiological mechanisms that prevent weight loss and contributed to the hypothesis that binge eating might represent a behavioral mechanism to prevent weight loss. After a series of animal studies, **set-point theory** emerged to explain why organisms resist weight change (Keesey, 1986). According to this theory, our bodies have evolved weight-defending mechanisms to withstand periods of famine. Such mechanisms could be triggered by dieting and make weight loss more difficult than is portrayed by weight loss programs.

Appealing as this model is, it has received only partial support in research examining weight loss among overweight individuals. The onset of binge eating may be specific to individuals who go from a normal weight to underweight and may not extend to individuals who go from overweight to normal weight. Many adolescent girls begin to diet at essentially a normal weight and during a developmental period when the body is still growing. Thus weight loss from normal weight during a period when weight gain is supposed to occur may be adequate to trigger the same weight-defending mechanisms observed in the Conscientious Objectors Study. Supporting a link between weight loss and binge eating, greater weight suppression (the difference between one's highest lifetime weight and current weight) was found to predict onset of a bulimic syndrome at 10-year follow-up of men and women originally assessed in college (Keel & Heatherton, 2010).

An association between dieting and disordered eating attitudes and behaviors has been demonstrated in prospective longitudinal studies (Stice, 2002). Patton, Johnson-Sabine, Wood, Mann, and Wakeling (1990) found that girls who were dieting at initial assessment were eight times more likely to have developed an eating disorder at 12-month follow-up than were girls who were not dieting. Most modern cases of AN in Western culture begin when the person begins a weight loss diet. Studies have supported a specific link between dieting and binge eating. In longitudinal studies, dieting most often precedes and predicts binge eating among adolescent girls (Patton et al., 1990; Stice, 2002). Most patients with BN recall going on a diet before experiencing their first binge episode (Bulik, Sullivan, Carter, & Joyce, 1997; Mussell et al., 1997). Experimental studies demonstrating an association between dieting status and the propensity to eat larger amounts of food were described in Chapter 4.

In contrast to studies suggesting that dieting contributes to eating pathology, Presnell and Stice (2003) found that individuals with bulimic symptoms who were placed on diets not only lost weight but also showed a reduction in bulimic symptoms. Further, Lowe, Gleaves, and Murphy-Eberenz (1998) found that, among patients with BN, greater dietary restraint was associated with lower binge frequency. The latter finding makes some sense, because recurrent binge-eating episodes reduce overall dietary restriction. Indeed, women with ANR by definition show high levels of dietary restriction without experiencing binge-eating episodes (American Psychiatric Association, 2013). (This does not mean, however, that dieting

is not contributing to their eating pathology.) Among women and men with BED, 55–81% reported that the onset of binge eating occurred before their first diet (Manwaring et al., 2006; Spurrell et al., 1997) compared with only 9–17% of women with BN (Bulik et al., 1997; Mussell et al., 1997). Thus, some individuals develop binge eating in the absence of dieting. However, this does not mean that dieting does not increase the risk of binge eating. It simply points to the existence of additional risk factors for binge-eating episodes (Grilo & Masheb, 2000), particularly for individuals with BED.

For boys, concerns about being too small would not lead to weight loss dieting and may protect them from developing AN and BN relative to girls. However, boys' exposure to magazines portraying a lean, muscular male ideal has been associated with increased desires to gain weight and to increase muscle definition as well as with engaging in muscle-enhancing behaviors, including steroid use (Field et al., 2005). Media exposure to muscular ideals also has been associated with more frequent self-weighing and greater body dissatisfaction among boys (Quick, Larson, Eisenberg, Hannan, & Neumark-Sztainer, 2012). Furthermore, a five-year longitudinal study found that a desire to gain weight was a prospective risk factor for steroid use in boys (van den Berg et al., 2007).

Conclusion

The increasing idealization of thinness for women provides one explanation for the increasing rates of eating disorders such as AN and BN in Western cultures in the second half of the 20th century. The thin ideal contrasts sharply with the appearance of most women's bodies, leaving most women wanting to lose weight. The marketing of the promise that body weight can be controlled makes being overweight appear to be a sign of personal weakness. The final step in the explanatory chain is linking weight loss diets to the core feature of AN—significant weight loss—to the core feature of BN—binge eating, and to the core feature of purging disorder—purging. An increasingly lean muscular ideal for men may protect them from developing illnesses such as AN but may increase their body dissatisfaction and their risk for unhealthy weight control behaviors. Finally, among individuals who find themselves with a genetic predisposition for obesity in an **obesogenic** society, loss-of-control eating and binge eating may arise in reaction to distress caused by the denigration of fatness and the associated societal stigma (Puhl & Brownell, 2010).

Despite its coherence and supporting evidence, reaction to media ideals and social pressures is far from a complete explanation of the etiology of eating disorders. First, evidence of AN outside the context of the modern idealization of thinness (see Chapter 3) suggests that something other than the thin ideal can contribute to this illness. Second, binge eating precedes the first diet in a small, but reliable percentage of women with BN and in a majority of individuals with BED (such as our case study Jamie). Finally, while the thin ideal has led to body dissatisfaction and attempts at weight loss in a majority of American women, eating disorders affect only a very small minority.

Thus an explanation rooted in cultural ideals seems neither necessary nor sufficient to understand the etiology of eating disorders. Such ideals provide an important part of the explanation for the emergence of these disorders, but they are only part of the picture.

Chapter 6 examines how more immediate spheres of social influence—namely, family and peers—may contribute to the risk of eating disorders. As reviewed in Chapter 4, high parental expectations may represent a transdiagnostic risk factor for AN, BN, and BED. In addition, Chapter 6 will discuss how individuals at increased risk for eating disorders may actively shape their immediate social environments in ways that further increase this risk. Thus Chapter 6 provides a transition from examining environmental influences to examining individual differences that influence eating disorder risk.

Key Terms

Obesogenic
Resting metabolic rate
Set-point theory
Objectification

6

Family and Peer Factors in the Development of Eating Pathology

Eating disorders often begin during adolescence, when families and peers form one's immediate social environment. Early hypotheses on the etiology of eating disorders focused almost exclusively on patients' families. Chipley (1860) attributed self-starvation to a desire to gain attention from family, and a common therapeutic recommendation of the late 19th century was to remove afflicted individuals from their family environments. Gull (1874) recommended that patients be surrounded by "persons who would have moral control over them; relations and friends being generally the worst attendants" (p. 26). A focus on the family environment also reflected the dominant theoretical models for mental disorders when AN emerged—namely, psychoanalytic and, later, psychodynamic models.

Because different theoretical orientations have contributed to different explanations for the etiology of eating disorders, this chapter reviews historical models of family influence along with data that have refined our understanding of how the immediate social context, including families and peers, may influence the development of eating disorders.

Psychoanalytic Model of Family Influence

Historically, **psychoanalytic** models provided the first description of how parents contributed to the development of eating disorders in children. Although data have largely failed to support these models as originally presented, the impact of these early explanations can be seen in scales developed to measure factors that have been associated with eating disorders and that, in some instances, have been identified as risk factors for these disorders.

Building on the emergence of AN symptoms near the onset of puberty and a view of neurotic illnesses as stemming from unconscious sexual conflicts, psychoanalytic theory interpreted AN as reflecting a fear of sexual maturity. According to the psychoanalytic model (see Figure 6.1), this fear arose in families in which the father was kind but passive and the

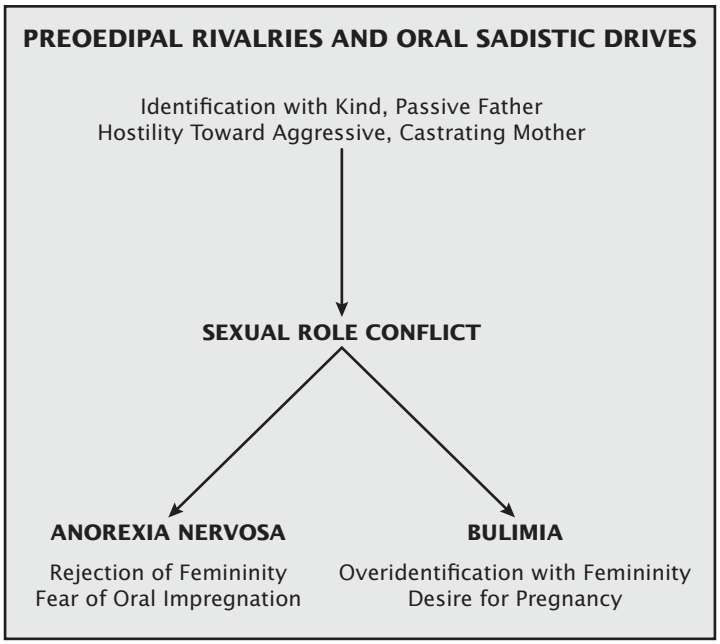


FIGURE 6.1 Psychoanalytic model of anorexia nervosa and bulimia. Source: Boskind-Lodahlm M. (1976). Cinderella's Stepsisters: A Feminist Perspective on Anorexia Nervosa and Bulimia Signs. *Journal of Women in Culture and Society*. 2, pp. 342–356 Copyright © The University of Chicago. Reprinted with permission from the University of Chicago Press.

mother was aggressive and "castrating," a psychoanalytic term meaning overpowering and controlling. Because mothers in these families provided unfit models of femininity, girls feared becoming women like their mothers. This fear became symbolized as a fear of oral impregnation (becoming pregnant via the mouth). Szyrynski (1973; quoted in Boskind-Lodahl, 1976) provided the following summary of the fears thought to underlie AN:

> Fear of pregnancy often dominates the picture; pregnancy being symbolized by food, getting fat means becoming pregnant. Such fantasies are also quite often formulated as oral impregnation. The girl, after kissing a boy for the first time, gets panicky lest pregnancy should follow. She pays particular attention to her gaining weight and not infrequently a casual remark of a visitor, a relative, or a friend, that she is looking well and probably has gained some weight will unleash the disastrous ritual of self-starvation.

Thus girls suffering from AN were thought to have an unconscious hatred of femininity stemming from their unconscious pre-Oedipal mother conflict. Anorexia nervosa was interpreted an attempt to regress to the safety of childhood. The physical signs of AN seemed to confirm this interpretation, as starvation led to a loss of both secondary sex characteristics and menstrual function. For girls, treatment involved (as it did in the psychoanalytic care of most women suffering from mental disorders) "putting an end to that hatred of femininity by helping the woman learn to accept and to act out the traditional female role" (Boskind-Lodahl, 1976, p. 345).

Consistent with this model, Maturity Fears is a subscale on the Eating Disorders Inventory (EDI; Garner, Olmstead, & Polivy, 1983), a frequently used measure of attitudes

and behaviors characteristic of patients with AN and BN (Clausen, Rosenvinge, Friborg, & Rokkedal, 2011). Lower Maturity Fears predicted recovery among patients with AN (Fassino et al., 2001; van der Ham, van Strien, & van Engeland, 1998), and higher Maturity Fears predicted the onset of an eating disorder 10 years following college (Holland, Bodell, & Keel, 2013). These longitudinal studies suggest that a fear of adulthood may contribute to the presence of eating disorders. However, it is unclear whether this fear represents a specific risk factor for eating disorders or a general risk factor for a range of mental disorders (Hurley, Palmer, & Stretch, 1990; K. S. Mitchell, Wells, Mendes, & Resick, 2012).

Psychodynamic Model of Family Influence

Bruch (1978) posited that mothers of girls who later developed AN had difficulty accurately identifying and appropriately responding to their needs when they were infants. For example, a mother might have responded to the baby's hunger with holding, her sleepiness with food, and her distress with naps. This mismatch between the infants' needs (or drive states) and their mothers' responses, according to Bruch, impaired the development of the girls' ability to identify and interpret their own internal states, resulting in what Sifneos (1972) has termed ***alexithymia*** (from Greek roots meaning "inability to read emotional states") and what others (e.g., Garner et al., 1983) have termed poor *interoceptive awareness*. In an attempt to adjust to an environment that did not reflect their internal states, these girls learned to adapt their needs to what they perceived as being provided by their environments—thus becoming "perfect" daughters. However, because their environments either failed to perceive or responded inappropriately to child-initiated cues, these girls suffered from feelings of profound ineffectiveness (Bruch, 1978).

Parents of girls with AN often reported to Bruch that their daughters had been especially well behaved throughout childhood and had never defied parental limitations (Bruch, 1978). Thus the sudden refusal to eat represented a marked departure from the docile, accommodating daughters the parents had congratulated themselves on raising. Bruch believed that adolescence triggered this rebellion because it was a central developmental stage for the emergence of autonomy. A girl who was unable to identify her own needs and desires was ill equipped for the demands of this stage. The resulting emotional crisis contributed to a rebellion centering on the symbolically meaningful rejection of the primary source of maternal nurturance—food. The refusal of food also offered the sole arena in which the daughter could assert complete control and combat her feelings of inadequacy.

Findings from prospective studies have confirmed the importance of poor interoceptive awareness in predicting eating disorder symptoms in adolescents (Gustafsson, Edlund, Kjellin, & Norring, 2010; Killen et al., 1996; Leon et al., 1993, 1999; Leon, Fulkerson, Perry, & Early-Zald, 1995). A second component of Bruch's (1978) theory, that girls attempted to cope with confusion over their internal states by responding perfectly to their environments—that is, high perfectionism, has also received considerable support as a risk factor for eating disorders (Boone, Soenens, & Braet, 2011; Mackinnon et al., 2011). Longitudinal studies have found that higher perfectionism predicts onset of anorexic syndromes (Tyrka et al., 2002), bulimic symptoms (Boone et al., 2011; Killen et al., 1996; Vohs, Bardone, Joiner, Abramson, & Heatherton, 1999), and binge eating (Bardone-Cone,

Abramson, Vohs, Heatherton, & Joiner, 2006; Mackinnon et al., 2011). Although studies have supported poor interoceptive awareness and perfectionism as eating disorder risk factors, other components of Bruch's theory (e.g., feelings of ineffectiveness) have received far less support (Leon et al., 1993, 1995, 1999; Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006) or seem to affect risk only when combined with other risk factors such as perfectionism and weight concerns (Bardone-Cone et al., 2006). Thus empirical research supports some, but not all, of Bruch's (1978) theory.

Moreover, evidence that these risk factors are a result of infant experiences has been limited. Bruch's evidence came from the lack of conflict over infant feeding that mothers recalled: "The early feeding histories of many anorexic patients, when reconstructed in detail, are often conspicuously bland" (Bruch, 1978, p. 43). Still, it is unclear that early rearing experiences of girls who go on to develop AN differ from those of girls who do not. This aspect of Bruch's theory is difficult to test with retrospective, follow-back studies, because patients with eating disorders would be unable to recall such early experiences, and parents would have been unaware that they were not responding to their infant's needs appropriately.

Several studies have examined parental emotional and behavioral patterns surrounding feeding in high-risk and low-risk populations—namely, children of mothers who have suffered from eating disorders and children of mothers who have never suffered from eating disorders, respectively (Agras, Hammer, & McNicolas, 1999; Russell, Treasure, & Eisler, 1998; A. Stein, Woolley, & McPherson, 1999; Waugh & Bulik, 1999). Much of this work followed from the observation that children of women who had suffered from AN were at an increased risk for **nonorganic failure to thrive**—a condition in which young children do not gain adequate weight, and no medical reason can be found for their low weight (Brinch, Isager, & Tolstrup, 1988; van Wezel-Meijler & Wit, 1989). Women with histories of eating disorders were found to have more anxiety, rigidity, and conflict about feeding their children than did women who had never suffered from an eating disorder. This pattern was related to lower weight in the children of the women in the former group (A. Stein, Murray, Cooper, & Fairburn, 1996; A. Stein, Woolley, Cooper, & Fairburn, 1994; A. Stein et al., 1999).

A. Stein et al. (1994) videotaped mother–infant interactions during mealtimes and playtimes. "Index mothers" who had experienced a partial or full eating disorder in the year following the birth of their children (index infants) were compared with healthy control mothers from the same community who were matched to index mothers on income and education. Raters who were blind to the mothers' eating disorder status coded videotapes for several factors, including conflict between the mother and child and how the conflict was resolved. During playtimes, no differences were observed between index and control mothers' interactions with their infants. During mealtimes, however, index mothers were more intrusive, made more critical and derogatory comments, and were more concerned with avoiding a mess than were mothers in the control group. Significantly greater conflict during mealtimes between index mothers and infants resulted in significantly less food intake: Index infants weighed significantly less than control infants, and weight was significantly inversely associated with conflict—that is, greater conflict during feeding was associated with lower weight of the child.

In a related study, A. Stein et al. (1999) examined specific potential antecedents to conflicts and maternal and infant responses to these antecedents. The researchers identified three types of antecedents, which were coded by blind raters:

1. The infant indicated a desire to feed him- or herself while the mother indicated a desire to feed the infant.
2. The mother expressed concern over the manner of the infant's eating (usually about the mess the infant was making).
3. The infant refused offered food.

Three potential responses that might prevent conflict were also coded:

1. The mother acknowledges the infant's cues.
2. The mother puts aside her own desires.
3. The infant disengages.

Finally, three types of episodes were coded:

1. Antecedents end in conflict in the index group.
2. Antecedents do *not* end in conflict in the index group.
3. Antecedents do *not* end in conflict in the control group.

Episodes of antecedents leading to conflict were not coded in the control group because they occurred too infrequently.

The following is an excerpt from a description of one meal in the study:

> The mother is feeding her infant, holding the food dish out of his reach. The infant cries and tries to get hold of the food in the dish, indicating wish to feed himself. The mother then pulls the dish further away and says, "Ooh, we don't want you in here." The infant whimpers and tries to reach the food again. The mother responds in a critical tone, "No, you'll only get it all over the place" (avoiding mess).
>
> The mother now offers the infant another spoonful of food and the infant refuses and tries to put his hand in the dish again.
>
> The mother, ignoring the infant's signals, keeps the dish out of reach and then puts a spoon of food to the infant's lips which he refuses; he also cries.
>
> The mother says, "Oh, you've had enough then?" (describing refusal as satiety).
>
> After a brief pause, the mother then continues to offer food which the infant refuses. The mother then tries to divert the infant with a game, using the spoon as an aeroplane coming in to land in the infant's mouth. The infant repeatedly refuses the mother's attempts, crying increasingly in protest, with the mother eventually looking on in puzzlement at his distress. Very little food was consumed at this mealtime.
>
> A. Stein et al., *1999, p. 458; reprinted with kind permission from the Royal College of Psychiatrists*

Within the index group, antecedents were less likely to lead to conflict when mothers acknowledged infants' cues, mothers set aside their own concerns, or infants disengaged (A. Stein et al., 1999). Index mothers were less likely than control mothers to acknowledge infants' cues or put aside their own concerns. Index infants were significantly more likely than control infants to disengage. Indeed, no control infant disengaged in response to an antecedent.

A. Stein et al. (2006) conducted 5- and 10-year follow-up evaluations of the children and mothers included in their original study (A. Stein et al., 1994). Meal-related conflict was coded on a scale of 1 to 5 based on videotaped meals both when the children were infants and at 5-year follow-up, and eating pathology was measured at 10-year follow-up in both children and mothers. Compared with controls, children of index mothers had significantly higher disordered eating levels at 10-year follow-up, including elevated weight and shape concerns and dietary restraint, even though control and index children showed no differences in BMI. Level of meal-related conflict at 5-year follow-up and duration of the mother's eating disorder both predicted disordered eating levels at 10-year follow-up. Although none of the children met criteria for AN or BN, several of the index children resembled children suffering from ARFID, a new feeding disorder in the *DSM-5*.

Taken together, these data provide evidence that infants may avoid conflict by becoming passive when mothers fail to recognize or respond to their cues and that this reaction may contribute to lower weight during infancy. Continued meal-related conflict increases risk for disordered eating as children approach puberty. A pattern of passive avoidance could contribute to the illusion of a "perfect baby" and serve as a sign of increased risk for the emergence of disordered eating during adolescence observed by Bruch (1978).

Family Systems Model of Family Influence

Around the same time that Bruch (1978) was publishing her theories on the development of AN, Minuchin, Rosman, and Baker (1978) developed a **family systems model** that explained eating disorders as a manifestation of disturbed family relationships. According to this model, all families are made up of subsystems (e.g., spousal, parental, and sibling) involving different roles and responsibilities. Boundaries between subsystems can range from enmeshed to disengaged. **Enmeshment** refers to poor differentiation of boundaries. For example, children might be invited to take part in transactions that would typically remain between husband and wife (spousal subsystem), as when one parent seeks advice from a child about how to deal with frustration over the other parent's behavior. **Disengagement** refers to the existence of boundaries that create isolation among family members. For example, children might feel like an afterthought in the context of their parents' busy lives. Clarity of boundaries may differ across family members and may change within a family over time. Indeed, family systems need to change in response to the normal processes of development (i.e., as children grow to adulthood) and in response to external events (e.g., the family moves).

Minuchin and his colleagues (1978) posited that families in which AN arose were marked by the following characteristics: enmeshment within the family; overprotection of

children, resulting in rigid boundaries separating family members from extrafamilial relationships; conflict avoidance; and concern over bodily functions, including physical symptoms, eating, and diets. According to Minuchin et al.'s model, the lack of boundaries among members of these families triggered conflict as girls approached adolescence. However, conflict in these families had no means for outlet, because distress was suppressed behind a facade of closeness. Girls in enmeshed families were stifled in their attempts to achieve independence and establish relationships outside the family. Because there was no viable option for expressing interpersonal conflict, conflicts were **somaticized;** that is, they were expressed as physical conditions. Within Minuchin et al.'s (1978) framework, conflict could be revealed in a variety of physical maladies in any member of the family. Applied to AN, this model viewed the daughter's symptoms as evidence not of **intrapsychic** conflict, as proposed by Bruch (1978), but of intrafamilial conflict. Thus treatment needed to focus on healing family interactions.

A number of studies have examined the quality of family relationships of individuals with eating disorders. These studies have generally indicated that the families of such individuals show greater dysfunction than do families of healthy controls (Holtom-Viesel & Allan, 2013). In a systematic review of the literature, Holtom-Viesel and Allan (2013) concluded, however, that there was no typical pattern of family dysfunction that characterized or differentiated among eating disorders. For example, studies have variously supported greater conflict, lower conflict, and no significant differences in conflict in families of individuals affected by eating disorders when compared with control families. Notably, the studies reviewed by Holtom-Viesel and Allan (2013) examined concurrent associations between eating pathology and family relationships. Thus the casual implications of any significant associations are unclear. Dysfunctional family interactions might be responses to eating disorders rather than contributors to their development.

Shoebridge and Gowers (2000) used a retrospective, follow-back design to examine whether overprotective parenting precedes the onset of AN. Compared with mothers of matched controls, index mothers were more likely to report providing near-exclusive child care (defined as 95% or more of child care to the exclusion of care by fathers), infant sleep difficulties, maternal distress at first separation, higher maternal trait anxiety, and a later age for the daughter's first sleepover. In seeking to explain the maternal anxieties, Shoebridge and Gowers found that obstetric records revealed that 25% of the index mothers had experienced a severe obstetric loss (e.g., miscarriage) before the birth of the child in the study, compared with only 7.5% of the control mothers. In 90% of the cases of obstetric loss in index mothers, the daughter with AN was the next-born female child. Unsurprisingly, more index mothers than control mothers reported worrying significantly about miscarriage during the pregnancy that led to the birth of the child in the study. Of interest, no significant differences were found between index and control mothers' reports of child eating difficulties in the first five years of children's lives.

Taborelli et al. (2013) extended Shoebridge and Gowers's (2000) approach by comparing maternal experiences of anxiety in relation to daughters with AN and their healthy sisters. Mothers reported having experienced greater anxiety while pregnant with the daughters who later developed AN and also reported waiting until an older age to leave these

daughters in the care of another adult. Taborelli et al. (2013) found no differences in maternal anxiety or overprotectiveness between daughters with BN and their healthy sisters.

Byely, Archibald, Graber, and Brooks-Gunn (2000) used a prospective design to evaluate the relationship between girls' perceptions of family relationships and changes in dieting and body image disturbance. Over a one-year follow-up period, 10- to 14-year-old girls reported increased problems in family relationships. Negative family relationships predicted dieting at follow-up even after controlling for dieting at baseline. Family relationships, however, did not predict changes in body image. Byely et al. (2000) also evaluated maternal modeling of dieting behaviors as a potential predictor of dieting and body image disturbance in girls and found no association.

Laliberte, Boland, and Leichner (1999) examined the effects of family relationships in terms of two categories of variables: "traditional family process variables (conflict, cohesion, expressiveness)" and " 'family climate for eating disorders' . . . Family Body Satisfaction, Family Social Appearance Orientation, and Family Achievement Emphasis" (p. 1021). Both types of family relationship variables significantly predicted disordered eating attitudes and behaviors in college women, with the family climate for eating disorders demonstrating a stronger relationship. In comparisons of eating-disordered patients, depressed controls, and healthy controls, the family process variables distinguished both patient groups from controls but not from each other. The family climate for eating disorders, however, differentiated the eating-disordered group from both the depressed and healthy controls.

Examination of the role of family factors in the development of eating disorders thus far demonstrates some similarities across theories. First, most theories suggest a role for high parental expectations in the form reduced responsiveness to infant cues, which may contribute to a more critical family environment. Second, most theories acknowledge that familial concerns with food, eating, and weight may shape the content of difficult interactions. The next section examines the extent to which children may learn disordered eating attitudes and behaviors from their families' disordered eating attitudes and behaviors. This social learning model is consistent with A. Stein et al.'s (2006) finding that duration of exposure to maternal eating disorders predicted severity of disordered eating in children.

Social Learning Model of Family Influence

From a social learning perspective, disordered eating attitudes and behaviors might be learned from family members just as many other attitudes and behaviors are learned. Certainly food preferences that may have consequences for disordered eating are highly influenced by culture and rearing environment, as can be seen in Jamie's case.

Case Study: Jamie

One problem that Jamie had with all weight loss diets was their emphasis on eating more fresh fruits and vegetables. He felt that if the number of calories he could eat was going to be restricted, he wanted to use as many calories as possible for the foods he liked and not waste any calories on foods he didn't like. Jamie's favorite foods were those that reminded him of his mother's cooking. His mother had grown up in the South and made wonderful

chicken-fried steak with gravy, biscuits, and fried okra. Thinking that okra was a vegetable he liked, Jamie once tried to prepare stewed okra on a diet. However, the consistency of the stewed okra was that of "snot with boogers." It was slimy and slightly sticky, and the okra seeds were tasteless little blobs suspended in the goop. Jamie's father also played a part in his attitudes toward food: They both felt that a meal without red meat was not a "real" meal.

Supporting a social learning model, eating disorders have been found to run in families (Ferreira, de Souza, da Costa, Sichieri, & da Veiga, 2013; Lilenfeld et al., 1998; Strober, Lampert, Morrell, Burroughs, & Jacobs, 1990). Woodside et al. (2002) found significantly elevated weight and shape concerns among parents of eating-disordered subjects compared with controls. Gershon et al. (1983) found that mothers of eating-disordered adolescents had histories of dieting more frequently than mothers of controls. Several cross-sectional studies support the idea that families may provide a context for social learning of disordered eating attitudes and behaviors (Quiles, Quiles, Pamies, Botella, & Treasure, 2013).

Agras et al. (1999) conducted a prospective longitudinal study with 41 mothers with current or past eating disorders (ED mothers) and 153 control non-ED mothers, along with their children, starting when the children were infants. Several differences emerged that pertained specifically to daughters of ED mothers. During infancy, daughters of ED mothers vomited more frequently than did daughters of non-ED mothers. There also was a significant association between ED mothers' eating disorder symptoms and their attitudes toward the weight or shape of their daughters but not of their sons. Regardless of the child's gender, ED mothers reported using food to reward or calm children more often, fed their children on less regular schedules, and reported that their children dawdled more while eating than did non-ED mothers. Stice, Agras, and Hammer (1999) examined rates and predictors of childhood eating disturbances based on the data collected by Agras et al. (1999). Childhood eating disturbances emerged over the course of five-year follow-up in 34% of participants. The relevant behaviors included inhibited eating, secretive eating, overeating, and overeating-induced vomiting. Interestingly, no significant gender differences were found for rates of these disturbed eating patterns, with the exception that overeating-induced vomiting was more common in boys than girls. Children's eating disturbances were predicted by various maternal variables, including the mother's BMI, body dissatisfaction, bulimic symptoms, and dietary restraint.

In a three-year longitudinal study of adolescent girls, the extent to which mothers had internalized the thin ideal predicted bulimic symptoms in their daughters (Linville, Stice, Gau, & O'Neil, 2011). In a 10-year follow-up study of college students, having a mother who dieted frequently predicted increased drive for thinness and bulimic symptoms in women but not men (Keel, Forney, Brown, & Heatherton, 2013). These results, unlike those of Stice et al. (1999), support a prospective relationship between disordered parental eating attitudes and behaviors and the emergence of eating disturbances in daughters but not sons.

An alternative to a social learning explanation for associations between parental concerns about eating and weight and disordered eating in daughters is that biological relatives share not only their environments but also their genetic makeup. Klump and colleagues examined scores on a measure based on the EDI in 680 eleven-year-old and 602 seventeen-year-old female twin pairs (Klump, McGue, & Iacono, 2000c) and reassessed disordered

eating in the 11-year-old twins when they were 14 and 18 years old (Klump, Burt, McGue, & Iacono, 2007). In the 11-year-old cohort, correlations of EDI-based scores within twin pairs were similar for MZ and DZ twins. However, EDI correlations were significantly higher for MZ than for DZ twins in the 17-year-old cohort. Moreover, follow-up of the 11-year-old twins showed significantly higher EDI correlations for MZ than for DZ twins when they were 14 years old and again when they were 18. Reflecting these patterns, shared environmental factors (e.g., family rearing environment) significantly explained the EDI scores in the 11-year-old twins, while genetic factors significantly explained the EDI scores in twins at ages 14, 17, and 18 years. These results suggest that the rearing environment can significantly affect disordered eating attitudes and behaviors in prepubertal girls but that in adolescence genetic makeup becomes more important. The role of genetic factors in eating disorders will be covered in greater depth in Chapter 8.

Influence of Parental Modeling Versus Parental Direct Comments

In contrast to the studies discussed above, several studies have failed to find significant associations between parents' self-reported disordered eating levels and daughters' disordered eating (Byerly et al., 2000; Eisenberg & Neumark-Sztainer, 2010; Keel, Heatherton, Harnden, & Hornig, 1997; Ogden & Steward, 2000; Sanftner, Crowther, Crawford, & Watts, 1996; Steiger, Stotland, Ghadirian, & Whitehead, 1995). A hidden variable in studies finding such an association may be the extent to which family members make direct comments to their daughters concerning weight and eating. Such comments may come in the form of advice ("You should cut back on dessert if you want to lose weight"), criticism ("You could stand to lose a few pounds"), encouragement ("You have such a pretty face; if you could just lose some weight, then everyone would see how pretty you really are"), and teasing ("Yeah, you're big-boned . . . and big-butted and big-bellied—just kidding!").

Striegel-Moore and Kearney-Cooke (1994) found a strong relationship between mothers' dieting and the extent to which they encouraged their children to diet. Keel, Heatherton, et al. (1997) reported that adolescent girls were more likely to diet when their mothers commented on their weight, particularly when the mothers described the daughters as overweight, and that girls' body dissatisfaction was related to their fathers' comments on their weight. Thus simply observing a parent who is concerned about his or her own weight may not be critical to increasing risk for disordered eating, whereas a parent's encouraging a child to diet may increase the child's risk for body image disturbance and disordered eating.

Smolak, Levine, and Schermer (1999) examined the influence of parents' direct comments about their children's weight and parents' modeling of concerns about their own weight on their children's body dissatisfaction and weight loss behaviors. For girls, body dissatisfaction and weight loss behaviors were correlated with mothers' comments on the daughters' weight, mothers' complaints about their own weight, mothers' weight loss attempts, and fathers' complaints about their own weight. For boys, the only consistent predictor of both body dissatisfaction and weight loss behaviors was mothers' comments on the sons' weight. Overall, direct parental comments had a greater effect than did parental

modeling, and mothers had a greater effect than fathers did on children's body dissatisfaction and weight loss behaviors.

The results of these studies suggest that modeling alone may not explain the familial aggregation of eating disorders. Rather, criticism of children's weight or shape may be a particularly pernicious environmental risk factor for the development of eating pathology. This conclusion is consistent with the findings of Fairburn and colleagues (1997, 1998) reviewed in Chapter 4. Jean's case demonstrates how such criticism can coincide with the stress of pubertal development in creating body shame.

Case Study: Jean

Jean knew that a lot of her weight concerns came from her mother. When Jean reached puberty, her mother expressed shock and dismay. Jean's menstrual cycle began when she was 11 years old. Her mother had not expected her daughter to start menstruating until she was 13, the age when her own menses had begun. Jean's mother felt that Jean had begun menstruating at 11 because Jean was "too fat." As with all girls, Jean's weight increased during puberty. However, her mother refused to buy her new clothes until the beginning of the next academic year. So Jean was forced to wear shirts and pants that were too tight. Boys at school started commenting on her development, and one boy kept trying to touch her breasts and buttocks. Jean started wearing a coat throughout the school day to hide her body.

Jean's case illustrates the role of her mother and her peer environment in contributing to body shame. In a longitudinal study of adolescent girls and boys, weight-based teasing—whether by family members or peers—predicted increases in binge eating and extreme weight control behaviors in boys and increases in dieting in girls at five-year follow-up (Haines, Neumark-Sztainer, Eisenberg, & Hannan, 2006).

Importantly, family environment appears not to continue to explain disordered eating risk after puberty in girls, and genetic makeup does not explain 100% of eating disorder risk. Thus it is important to examine the impact of nonshared environmental factors—that is, environmental factors outside of the family that may be unique to each family member—such as specific friendship groups that are formed during adolescence. The next section focuses on the social influence of peers on the development of eating pathology.

Peer Influences on Eating Pathology

Although families represent a central part of the immediate social environment when eating disorders emerge, adolescence marks an important social transition away from immediate family and toward peers. Thus peer values and behaviors come to form an increasingly salient part of the social environment during the period of peak risk for eating disorders. Several studies support significant peer influence on disordered eating behaviors and attitudes during adolescence and young adulthood. The reported frequency of friends' dieting has been positively associated with unhealthy weight control behaviors in adolescents (Clemens, Thombs, Olds, & Gordon, 2008; Eisenberg, Neumark-Sztainer, Story, & Perry, 2005) and with drive for thinness in adults (Gravener, Haedt, Heatherton, & Keel, 2008).

In longitudinal studies, exposure to peer dieting predicts greater disordered eating several years later. Over a five-year follow-up, friends' dieting significantly predicted use of unhealthy and extreme weight control behaviors (e.g., fasting and self-induced vomiting) and binge or loss-of-control eating in girls (Eisenberg & Neumark-Sztainer, 2010; Neumark-Sztainer et al., 2007). Among women (but not men), the frequency of dieting by college roommates predicted drive for thinness, bulimic symptoms, and use of self-induced vomiting to control weight at 10-year follow-up (Keel et al., 2013).

The social influence of peers may contribute to gender differences in risk for eating disorders. To the extent that adolescent girls are more likely than boys to express concerns about being fat, engage in "fat talk" (disparaging remarks about one's own body made to peers, discussed further below), diet to lose weight, and spend more time socializing with other girls than with boys, girls have greater environmental exposure to the thin ideal within their peer group than do boys. Further, longitudinal data suggest that females may be more sensitive than males to social influences on drive for thinness and bulimic symptoms (Keel et al., 2013). This differential sensitivity is seen even when females and males have comparable exposure to such influences: During college, men and women reported that similar proportions of their mothers were frequent dieters; at 10-year follow-up, however, mothers' dieting status predicted drive for thinness only in women (Keel et al., 2013).

Peer Socialization Versus Peer Selection

Prospective longitudinal designs control for levels of disordered eating at baseline when examining predictors of disordered eating (see Chapter 4). This aspect of these designs is important because similarity among peers with respect to weight concerns and disordered eating could reflect socialization (the spreading of symptoms from one individual to another within a peer group) or it could reflect selection of peers with similar weight concerns and eating behaviors ("Birds of a feather flock together"). Without controlling for levels of disordered eating at baseline, peers might look similar to one another at follow-up because they selected each other for this at baseline. Another way to capture the processes by which peers influence one another's disordered eating is to examine changes within and between peer groups over time.

In the process of socialization, attitudes and behaviors spread from one group member to another. Social norms arise for features that are important to the group. Individuals get social rewards, such as an increase in popularity, for conforming to peer expectations and get social punishments, such as a decrease in popularity or even rejection, for deviating from peer expectations. These consequences create peer pressure toward uniformity in behavior. Thus as group members spend more time together, their attitudes and behaviors become more similar.

The first evidence for the socialization of bulimic symptoms came from a study by Crandall (1988) of friendship groups in college sororities. Girls living in one of two sorority houses completed questionnaires regarding their own binge eating and their friendship groups in the fall and the late spring of one school year. Crandall hypothesized that socialization during the year would lead to similarity in binge-eating patterns within peer groups in the spring. Crandall found that friends' binge-eating practices grew more similar from the fall to the spring semester. In both sororities, an individual girl's popularity was related to the extent to which her binge eating was similar to the average level of binge eating in

her sorority. However, binge-eating patterns differed between the two sororities, suggesting that the "right" level of binge eating depended on social norms within a sorority rather than reflecting college-wide norms.

In an extension of this work, Zalta and Keel (2006) conducted a longitudinal study of college participants' similarities to selected peers (friends who chose to be randomly assigned to live in the same upper-class house as a blocking group), unselected peers (students from other blocking groups who had been randomized into the same house as participants and with whom participants dined as frequently as with their selected peers), and nonpeers (members of other upper-class houses with whom participants did not dine regularly). Zalta and Keel evaluated how similarity in self-esteem, perfectionism, and bulimic symptoms varied with exposure to selected and unselected peers. With respect to self-esteem and perfectionism, participants were more similar to their selected peers than unselected peers and nonpeers. Neither length of contact nor a period of separation (summer break) influenced the extent to which participants were more similar to their selected peers. These findings thus supported an effect of personality variables on peer selection rather than an effect of socialization on personality variables. For bulimic symptoms, by contrast, participants' greater similarity to selected peers varied with their length of contact with those peers and disappeared after summer break. Participants were no more similar to unselected peers or nonpeers on bulimic symptoms than would be expected by chance, regardless of degree of exposure. These results suggest that selected peers exert a social influence on bulimic symptoms. Consistent with Zalta and Keel's (2006) findings, peer selection was found to be an important predictor of peer similarity in body dissatisfaction and bulimic symptoms in a longitudinal study of adolescent girls (Rayner, Schniering, Rapee, Taylor, & Hutchinson, 2013).

Case Study: Valerie

One factor that contributed to Valerie's confusion about whether she had a "real" eating disorder was that a lot of girls she knew used purging to get ready for beach trips. In fact, she had first purged, when she was 13, before just such a trip. Her friends would take turns running to the pharmacy to pick up "supplies," which generally included tanning lotion, diet pills, diuretics, and laxatives. No one hid the behavior, and everyone considered it a lark—nothing serious, shameful or concerning. Valerie clearly recalled her friend Diana's stating matter-of-factly: "Well, I better get rid of this potbelly before I try to put on a bikini. Can I pick up some pills for you while I'm at the chemist's?" Given that Diana was 5 feet 7 inches tall and weighed no more than 130 pounds, Valerie found herself thinking that if Diana viewed herself as having a potbelly, she must think that Valerie was a disgusting pig.

Valerie's attitudes about her own eating disorder are influenced not only by her friends' behaviors but also by how they talk about themselves. Knowing that her friends use purging to control their weight makes this behavior seem potentially normal to her, and hearing Diana's derogatory comments about her own weight left Valerie feeling more insecure and worried about looking fat to others. While neither believing that purging was normative among her friends nor hearing fat talk may have contributed directly to the onset of Valerie's eating disorder, both factors could have contributed to its maintenance over time.

"Fat Talk" and Eating Disorder Risk

The term *fat talk* was created in the mid-1990s to describe a pattern of derogatory statements that girls and women make about their own bodies when they are with peers to create solidarity around a shared concern (Nichter & Vuckovic, 1994). Unlike weight-based teasing, fat talk is generally directed not toward another's weight but toward one's own ("Do these jeans make me look fat?"). If fat talk is directed toward another's weight or eating, it is presented as a combination of a compliment and self-deprecating humor ("That's such a healthy lunch—I wish I had your self-control instead of being such a fat pig"). Fat talk is common among adolescent girls and women (Sharpe, Naumann, Treasure, & Schmidt, 2013) and can function to elicit support or reassurance from peers. For example, in response to "Do these jeans make me look fat?" a friend might respond: "No! I'm so jealous of how good you look. I could never pull that off with my thunder thighs."

However, fat talk has several potentially pernicious effects. First, when fat talk elicits reassurance, the positive reinforcement increases the likelihood of future fat talk and reassurance seeking, leaving the speaker increasingly dependent on the opinions of others to feel good. Second, when the speaker is clearly within a healthy weight range, fat talk communicates that a healthy weight is fat and thereby strongly endorses an unhealthy, thin ideal ("If *she* thinks she's fat, she can't possibly believe that I look good in these jeans"). Thus the act of fat talk contradicts any reassuring statements made during fat talk.

Longitudinal studies support that exposure to fat talk is a risk factor for disordered eating (Sharpe et al., 2013). Experimental studies have examined the causal effects of fat-talk exposure on body dissatisfaction and weight concerns. In these studies, participants believe that they are simply interacting with another research participant when in fact they are interacting with a research assistant who is following a script to ensure that participants' exposure to fat talk is experimentally manipulated within the study. Stice, Maxfield, and Wells (2003) used a thin confederate who talked about how fat she was and about her plans to lose weight in the experimental "fat talk" condition and spoke about a neutral topic in the control condition. Thus all participants were exposed to a thin peer; however, half were exposed to a thin peer engaging in fat talk. Following these interactions, female college students randomized to the fat-talk condition expressed greater body dissatisfaction than did women in the control condition. Tucker, Martz, Curtin, and Bazzini (2007) extended these findings by randomizing women to one of three conditions: fat talk, self-acceptance, or self-aggrandizement (the opposite of fat talk, in which the research assistant would make especially positive statements about her appearance). Thus in all three conditions, the confederate was talking about her body rather than a neutral topic, but the valence of her comments ranged from highly negative to highly positive. While there were no differences among the conditions in how likable participants perceived the confederate to be, participants in the fat-talk condition were more likely than those in the other two conditions to make negative statements about their own weight and shape afterward: Fat talk begot fat talk.

The Emerging Role of Social Media

Chapter 5 reviewed the role of the media in reinforcing the thin ideal by portraying unrealistically thin women as ideals of beauty. Since publication of the first edition of this book in 2005, social media have gained increased importance in young women's daily lives. For example, a recent study by Mabe et al. (2014) found that college women reported using Facebook approximately four times a day for approximately 20 minutes each time, and most (87%) accessed the site through an application on their smartphones. More than two-thirds of college women preferred to look at photos over other Facebook activities.

The ability to post carefully curated photos that may have been digitally altered with online tools such as "Plump&Skinny Booth" allows Facebook users to present and view images that display unrealistic beauty ideals. Social media thus create an opportunity to reinforce the thin ideal via posts and "likes" of, and comments on, idealized images. Indeed, participants with more disordered eating viewed receiving comments and "likes" on their status updates and receiving comments on their photos as more important than did participants with less disordered eating. Social media also provide a primarily visual medium that enhances women's ability to objectify themselves and their peers and to engage in social comparison of themselves with their peers. Mabe et al. found that participants with more disordered eating "untagged" photos of themselves and compared their photos with those of their female friends more often than participants with less disordered eating did. Finally, higher disordered eating levels were associated with spending more time overall on Facebook.

Because a correlation between higher disordered eating levels and greater Facebook use does not prove causation, Mabe et al. (2014) used an experimental design to determine whether Facebook use caused changes in risk for eating pathology. Participants were randomly assigned to one of two conditions. In the experimental condition, participants completed measures of eating disorder risk before and after spending 20 minutes using Facebook as they normally would. In the control condition, participants completed measures of eating disorder risk before and after spending 20 minutes reading a Wikipedia article on the ocelot and watching a brief YouTube video clip about this animal. This control condition was selected to match the experimental condition for exposure to images while avoiding any references to food, eating, or weight. Compared with women in the control condition, women who used Facebook experienced greater reinforcement of weight and shape concerns and increases in anxiety—both of which are risk factors linked to the development of eating disorders (see Chapter 4).

To summarize, both longitudinal and experimental studies indicate that peer environment is a potent source of social influence on risk factors for eating disorders. Individuals are more likely to compare themselves with peers than with random individuals in their environment because they see peers as being more similar to themselves and as providing role models for attainable goals. Further, peer selection is influenced by personality features that may themselves be risk factors for eating disorders, such as perfectionism (discussed more in Chapter 7). Thus when considering the influence of social environment, it is important to consider how individuals choose their environments as they get older based on their personality traits. These personality factors may contribute individuals' taking up pursuits, such as being in ballet or athletics, that expose them to social environments that further reinforce the importance of

weight and shape and contribute to risk for eating pathology. The manipulation of the images people present of themselves on social media may also lay the groundwork for increased risk for eating disorders. Friends who view posted images may not be as cognizant of the extent to which these pictures do not reflect reality as they are when looking at, for example, pictures of celebrities on magazine covers. Moreover, self-objectification during the act of posting an image can hurt an individual as well: Even as she alters her own pictures before posting, she is communicating to herself that she is somehow not good enough.

Conclusion

Although it is easy to focus on the differences among various theories about the role of family factors in the development of eating disorders, it may be more useful to focus on their similarities. Families of individuals with eating disorders have more problems than do families of individuals without eating disorders, These involve general problems in interaction style and specific problems related to disordered eating attitudes and behaviors.

Rearing environments in which child-initiated cues are not recognized or appropriately responded to may increase the pressure on children to conform to external demands. As a consequence, these individuals may be particularly vulnerable to messages concerning how they should behave and how they should look, which they may encounter from society-wide influences such as the media or from family members or peers. The rearing environment also may contribute to difficulties identifying and differentiating among one's internal drives and may reinforce the futility of attempting to do so. At the beginning of adolescence, the thin ideal may provide a road map for achieving social acceptance, particularly if family members and peers subscribe to attitudes that contribute to eating pathology.

Review of family factors in the development of eating disorders shows that families of individuals with these disorders do not starve their children or force them to binge eat. Instead, families influence children's thoughts, behaviors, and personality styles in ways that may increase the risk of eating disorders. These thoughts, behaviors, and personality features may influence children's selection of friends, and the resulting peer groups may either further increase risk for eating disorders or provide protection against developing them. Thus environmental vulnerability influences personal vulnerability, and personal vulnerability influences environmental vulnerability. The next chapter discusses psychological factors that may influence individuals' selection of high- versus low-risk social environments as well as how individuals respond to their environments.

Key Terms

Alexithymia
Disengagement
Enmeshment
Family systems model
Intrapsychic
Nonorganic failure to thrive
Psychoanalytic
Somaticize

7

Psychological Factors in the Development of Eating Disorders

The Contributions of Personality and Cognitive–Affective Processes

Chapters 5 and 6 covered two spheres of social influence on the development of eating disorders: society and family/peers. Although other spheres of social influence (e.g., participation in specific sports) may also have an effect, this chapter shifts the focus to psychological factors that contribute to eating disorder etiology. Psychological factors occur within the individual and explain individual differences in susceptibility to developing an eating disorder in response to social influences. Chapter 4 introduced some psychological factors in the context of risk factor research, and Chapter 6 considered psychological factors in the context of psychoanalytic, psychodynamic, and social learning models. This chapter focuses on the roles of personality and cognitive–affective processes in risk for developing and maintaining eating disorders.

Personality

Personality has been defined as a stable way in which individuals perceive, react to, and interact with their environments that is influenced by both biology and experience (Phares, 1988). **Temperament** has been defined as a biologically based predisposition to experience certain emotional and behavioral responses (Cloninger, Svrakic, & Przybeck, 1993). Temperament is the building block upon which personality develops. While acknowledging that considerable debate surrounds the definitions of temperament and personality (Craik, Hogan, & Wolfe, 1993), this section will review models that have been applied to understanding the development of eating disorders.

Cloninger described four dimensions of temperament: **novelty seeking, harm avoidance, reward dependence,** and **persistence** (Cloninger, 1987; Cloninger et al., 1993) that have been examined in relation to eating disorders. Novelty seeking is a tendency to pursue rewards. Harm avoidance is a tendency to avoid punishment by inhibiting behavior. Reward dependence is a tendency to continue rewarded behavior. Persistence is a tendency to continue behavior that is not immediately rewarded and has been linked to ambition, obstinacy, and obsessive–compulsive features.

On self-report measures, AN has been associated with low novelty seeking, high harm avoidance, and high persistence (Amianto, Abbate-Daga, Morando, Sobrero, & Fassino, 2011; Bulik, Sullivan, Weltzin, & Kaye, 1995; Fassino, Abbate-Daga, et al., 2002; Fassino, Amianto, & Abbate-Daga, 2009; Klump, Bulik, et al., 2000), even when individuals with AN are compared with their healthy siblings (Amianto et al., 2011). Bulimia nervosa has been associated with high harm avoidance but high novelty seeking (Bulik et al., 1995; Fassino, Abbate-Daga, et al., 2002; Fassino et al., 2009). In a review of studies examining self-reported differences between patients with eating disorders and healthy controls, Harrison, O'Brien, Lopez, and Treasure (2010) found that eating disorder patients, regardless of diagnosis, were more sensitive than controls to punishment. However, the restricting subtype of AN was characterized by less sensitivity to reward compared to controls, whereas both the binge-eating/purging subtype of AN and BN were characterized by greater sensitivity to reward compared to controls.

These findings map onto the behaviors typical of these disorders. In ANR, low sensitivity to reward could contribute to decreased eating, and high harm avoidance could contribute to behaviors intended to prevent weight gain, such as excess activity. High persistence would enable women with AN to reach and maintain extremely low weights. For ANBP and BN, the combination of high sensitivity to both reward and punishment captures the conflict between engaging in rewarding behavior (increased eating during binges) and trying to avoid punishing weight gain (engaging in inappropriate compensatory behaviors).

Data regarding temperamental differences that may contribute to BED have been less clear-cut. Compared with healthy controls, individuals with BED have higher novelty seeking and higher harm avoidance (Fassino, Leombruni, et al., 2002), consistent with patterns observed in BN. No differences emerged, however, when BED patients were compared with obese controls who did not have BED (Dalle Grave, Calugi, Marchesini, et al., 2013; Fassino, Leombruni, et al., 2002).

Tellegen (1982) described three personality dimensions: **positive emotionality, negative emotionality,** and **constraint** that have also been examined in relation to eating disorders. Positive emotionality is the tendency to enjoy and actively engage in work and social interactions. Low positive emotionality is characterized by high levels of introversion or a tendency to keep to oneself. Negative emotionality is the tendency to experience negative mood states (e.g., sadness, anxiety, and anger). Low negative emotionality is characterized by a tendency to be calm and unflappable. Constraint is the tendency to inhibit impulses and show caution, restraint, and conventionalism. Low constraint is characterized by a tendency to act impulsively.

Consistent with the results regarding temperament, AN has been associated with high levels of constraint, low levels of positive emotionality, and high levels of negative

emotionality compared with controls (Casper, Hedeker, & McClough, 1992; Pryor & Wiederman, 1996). Women with BN also have low levels of positive emotionality and high levels of negative emotionality (Casper et al., 1992; Peterson et al., 2010; Pryor & Wiederman, 1996), but BN is associated with lower constraint than is seen in AN (Casper et al., 1992; Pryor & Wiederman, 1996). In contrast, no differences have emerged on these basic personality dimensions between women with BED and either normal-weight or obese control participants without BED (Peterson et al., 2010).

Research findings on temperament and personality in patients with eating disorders map onto early clinical descriptions of patients suffering from eating disorders. Bruch (1978) characterized patients with AN as having a high level of perfectionism. Her patients tended to be straight-A students with many accomplishments. In addition, they were less likely than their peers to drink alcohol, use illicit substances, or be sexually active—suggesting higher levels of constraint. Prior to the onset of their illness, they tended to hide negative feelings. In contrast to their happy external appearance, Bruch's patients experienced significant anxiety, sadness, and concern about disappointing others (high negative emotionality). Finally, they tended to isolate themselves from others and avoid social engagement (low positive emotionality). Many of these features can be seen in Emily's case study.

Case Study: Emily

Emily's eating disorder came as a shock to her family, because she always seemed so in control of her life and destiny. That she might have a problem beyond her control did not make sense to them or to Emily. Even as a small child, Emily had always managed to keep things in perfect order. She had alphabetized her books by author and then title as soon as she learned the alphabet. She organized her dresser drawers so that her white ribbed socks were kept separate from her white terry socks, and both were kept separate from her colored socks. Emily recalled always wanting things to be perfect. She said she could even remember that on her first day of school she had decided that she would always do everything exactly as it should be done. In elementary school, she cried the first time she received a grade of less than 100%. She was a straight-A student throughout school. However, this consistent performance came at a cost. Because she feared ruining her perfect grade point average, Emily started avoiding classes in which she thought she might not do well. She took advanced placement courses in English and history, but she decided against taking advanced placement biology and math. Although she had been interested in becoming a doctor, she feared that those classes might be too hard for her and that she might receive less than an A. In contrast to her outward appearance of confidence and success, Emily constantly feared that people would realize that she actually wasn't very smart. Her need for perfect grades was as much a defense against being "found out" as it was a means to achieve entry into a good college.

Emily's story provides a classic picture of ANR. Long before her eating disorder emerged, Emily cared deeply about achieving perfection and avoiding failure, as if perfection and failure were the only two options in life. Her eating disorder seems to reflect another manifestation of an unrelenting drive to achieve and surpass all external standards. If the ability to control eating and weight is viewed as evidence of virtue (see Chapter 3), then Emily's behavior asserted her moral superiority over all others. This drive is mirrored in the

case of St. Veronica (see Chapter 3), who "carried more water and chopped more wood than anyone else" because she believed she "was in a race against all the other novices to show who loved God the most" (Bell, 1985, p. 71).

In contrast to early clinical descriptions of AN, Russell (1979) characterized his patients with BN as engaging in antisocial behaviors—abusing drugs, stealing, and being sexually promiscuous (suggesting low constraint)—as well as having social anxiety, depression (suggesting high negative emotionality), and poor social adjustment (suggesting low positive emotionality). The early clinical observations of Bruch (1978) and Russell (1979) indicate that both AN and BN patients have high negative emotionality but that they differ in constraint.

Rather than seeking to identify personality differences between AN and BN patients, others have tied personality function more closely to restricting versus binge-eating/purging behavior than to weight, reporting that a constricted/overcontrolled personality is characteristic of ANR while an impulsive/emotionally dysregulated personality is characteristic of ANBP and BN (Steiger, Puentes-Neuman, & Leung, 1991; Westen & Harnden-Fischer, 2001). Heterogeneity among AN patients on personality features supports this view (Lavender et al., 2013). Wildes, Forbush, and Markon (2013) recently identified weight phobia as an additional feature associated with personality differences within AN, with its absence being associated with less negative emotionality. Westen and Harnden-Fischer (2001) reported that a high-functioning/perfectionistic personality style was present in both AN and BN. Thus perfectionism may increase the risk of developing both eating disorders. Perfectionism combined with high constraint may contribute to the development and maintenance of ANR (Wildes et al., 2013). Perfectionism combined with low constraint may contribute to the development of bulimic symptoms (Westen & Harnden-Fischer, 2001).

One concern in interpreting results from correlational studies (such as comparisons of personality between women with and without eating disorders) is that the state of illness may affect personality measures (Vitousek & Manke, 1994). As was discussed in Chapter 5, starvation impacts personality functioning. In Keys et al.'s (1950) study of starvation, participants' scores on a measure of personality pathology changed as a consequence of weight loss. Several participants showed significant deviations from their normal personality and behavioral functioning. Some, for example, exhibited impulsive behaviors that they had never shown before starvation (e.g., stealing food). Other participants became preoccupied with food and developed eating rituals they had never performed before (see Figure 7.1). In the most extreme example of starvation-induced disturbance, participant No. 20 engaged in self-mutilation and chopped off three fingers of his left hand with an axe. In most participants, personality normalized after weight gain, and even No. 20 showed a "slow return toward normality" toward the end of nutritional rehabilitation (Keys et al., 1950, p. 897). These observations highlight the importance of using caution in interpreting personality function among individuals with current eating disorders as contributing to the illness, given the limited inferences that can be drawn from a cross-sectional design (see Chapter 4).

Because of concerns about the potential influence of eating disorders on personality function, results from longitudinal studies are particularly valuable. As reviewed in Chapter 4, perfectionism has emerged as a risk factor for eating disorders in both

FIGURE 7.1 Eating rituals such as licking a plate may be a consequence of starvation, as demonstrated by a participant in Keys et al.'s (1950) study. Source: Photograph by Wallace Kirkland, licensed by Getty Images.

retrospective, follow-back designs and prospective, follow-up designs. Studies have found that higher perfectionism also reduces likelihood of recovery from AN (Nilsson, Sundbom, & Hägglöf, 2008; Rigaud, Pennacchio, Bizeul, Reveillard, & Vergès, 2011). In contrast to perfectionism, high negative urgency—the tendency to act impulsively when distressed—is a prospective risk factor for binge eating (C. M. Pearson, Combs, Zapolski, & Smith, 2012).

In summary, research examining the temperament and personality of patients with AN supports early clinical descriptions. Women with ANR appear to be characterized by high levels of perfectionism and constraint. These qualities seem consistent with the overt behaviors of AN patients—an unrelenting pursuit of thinness through self-denial of food in the modern era and an unrelenting pursuit of moral purity through self-denial of food in past historical periods (see Chapter 3). Moreover, these characteristics predict both illness onset and maintenance. In contrast to findings for ANR, research has found ANBP and BN to be associated with high levels of impulsiveness and poor emotional regulation. Difficulties in controlling impulses have been implicated in the chaotic symptoms of women with bulimic symptoms—the oscillation between dietary restraint and binge episodes followed by purging.

Patients with eating disorders experience high levels of depression and anxiety. As described in Chapter 4, higher levels of negative emotionality predicted the development of new-onset eating pathology in a prospective longitudinal study (Leon et al., 1999). Although negative emotions tend to be more muted in patients with AN than in patients with BN,

the intensity of affect often increases as patients with AN gain weight, leading some experts to speculate that weight loss serves as an effective numbing mechanism (Kaye, Wierenga, Bailer, Simmons, & Bischoff-Grethe, 2013). Similarly, among patients with BN, binge-eating episodes appear to produce temporary reprieves from intense negative emotions by allowing women to "zone out" (Heatherton & Baumeister, 1991). A tendency to experience increased negative emotions could increase the likelihood of using extreme methods to regulate emotions. Further, a tendency to experience changes as particularly stressful could explain why eating disorders most often start during adolescence (Leon et al., 1999; Leon, Keel, Klump, & Fulkerson, 1997).

From this psychological perspective, personality and temperament explain the tendencies of individuals with eating disorders to experience certain emotional states, while eating disorder symptoms represent learned responses to those experiences.

Cognitive and Affective Processes

Learning Processes and Eating Disorders

Chapter 6 discussed how disordered eating might be learned through modeling or direct instruction within families. This explanatory approach is predicated on the idea that whether or not an individual develops an eating disorder depends on what they are exposed to within their environment. But given the widespread presence of messages reinforcing the thin ideal, why do only some individuals develop disorders of eating? This section addresses both basic processes of learning and how individual differences in those processes could contribute to vulnerability to developing eating disorders.

One type of learning, **operant conditioning,** may play an important role in the acquisition and maintenance of disordered eating behaviors. In operant conditioning, learned associations between behaviors and their emotional consequences influence the likelihood that those behaviors will recur. Evidence reviewed above indicates that individuals with eating disorders have altered sensitivity to rewards and punishment (Harrison et al., 2010). We would expect these differences to influence acquisition and maintenance of behaviors, including behaviors such as dietary restriction, binge eating, and purging.

In operant conditioning, **positive reinforcement** occurs when desirable consequences of a behavior increase the likelihood that this behavior will recur. For most individuals, dieting is accompanied by initial success in weight loss. This change may result in considerable positive social reinforcement, in the form of compliments and attention. Branch and Eurman (1980) conducted a survey of friends and relatives of patients with AN to understand social attitudes toward the patients' appearance and behaviors. Although all respondents expressed concern about the patients, 50% reported envy of patients' self-control and discipline concerning food. Further, friends and relatives were more likely to endorse positive than negative statements about the patients' appearance, agreeing more often with descriptions of them as "slender" (58%), "neat" (83%), "well groomed" (100%), and "fashionable" (100%) than with descriptions such as "skinny" (8%), "haggard" (8%), and "emaciated" (8%). Branch and Eurman concluded that even in their emaciated state, patients with AN

experienced more approval than disapproval. Thus both attention and admiration may serve as positive reinforcers of continued food restriction.

Like weight loss, binge eating may be highly positively reinforcing. Indeed, the foods typically consumed during binge episodes (e.g., ice cream, cookies, and candy) often are used by parents as rewards for good behavior. Although patients find binge-eating episodes very upsetting, in treatment they also express a sense of loss when they realize that they may never have binge episodes again after treatment. Jamie experiences binge episodes as highly pleasurable.

Case Study: Jamie

Jamie wished he could take a pill instead of eating food. He complained that diet pills were fine for curbing appetite, but the perfect diet pill would replace food altogether so that in addition to not feeling hungry, he would not need to eat. This would be his perfect diet pill because he could not imagine ever being able to stop eating once he started. Food always tasted too good to stop. Moderation was missing in other areas of Jamie's life as well. As an athlete in school, he had always worked out harder and longer than everyone else on the team. He loved the "high" he got from exercising. Because of this work ethic, he became a star member of the team and eventually its captain. Jamie's tendency to persevere was also an asset in his job. More than any other employee, Jamie would persist in pursuing clients until he landed the deal. The bonuses that Jamie received for signing new clients allowed him to purchase new "toys"—Jamie was a self-described technophile who loved computers, computer games, big-screen TVs, and DVDs. He described his apartment as a shrine to an electronics department.

Clearly, one reason Jamie has binge episodes is that he enjoys them. However, eating disorders do not consist solely of pleasurable, rewarding behaviors. To understand the full array of symptoms, one must examine both the consequences of a given behavior and the consequences that would result from *not* engaging in the behavior.

Like positive reinforcement, **negative reinforcement** increases the likelihood of a behavior recurring. However, negative reinforcement increases a behavior's likelihood because *not* engaging in the behavior has an undesirable consequence. The behavior prevents such an undesirable consequence or terminates an undesirable experience. For example, self-starvation may be negatively reinforced if it contributes to emotional numbing and reduces distress (Kaye et al., 2013). Similarly, binge eating may be negatively reinforced if it offers even a temporary respite from negative feelings (Heatherton & Baumeister, 1991). Although feeling nothing is not necessarily pleasurable, it is preferable to feeling distress. In a meta-analysis of studies examining emotional changes immediately before binge episodes, Haedt-Matt and Keel (2011) found consistent evidence that an increase in negative affect predicted binge eating. However, when a binge-eating episode ends, anxiety typically arises about how the binge will affect the individual's weight, and distress after the binge episode is even higher than it was before the episode (Haedt-Matt & Keel, 2011). Purging offers a means of avoiding this negative consequence. Although purging is not an effective form of weight control, it does decrease anxiety (Haedt-Matt & Keel, 2011). This reduction in anxiety provides powerful negative reinforcement for purging—even in the absence of a binge-eating episode.

Case Study: Valerie

Valerie's therapist asked her to keep a food diary so that they could work together to identify triggers of Valerie's purging behavior. In reviewing the diary, Valerie was able to see that some purging episodes were triggered by what she had eaten. For example, if she went out to dinner with friends and tried to eat "like a normal person" without restricting, she would usually excuse herself after the entrée and go the bathroom to purge before returning to order dessert, which she would also get rid of once she got home. However, her purging was not restricted to meals out with friends. She also purged at home, and there wasn't always a consistent difference between which foods she kept down and which she threw up. Instead, the consistent pattern that emerged was how she felt before and after the eating episode. Any time that she felt highly anxious before eating, she was very likely to purge after eating, and after purging her anxiety would go down. In these instances, it seemed as though she was eating solely to trigger a purging episode to alleviate her anxiety. In addition, sometimes her eating triggered feelings of extreme fullness, which made her feel anxious about becoming fat. In these cases, even if the type and amount of food fell within her accepted rules for being "safe," she would purge to relieve the feelings of fullness and anxiety. In contrast, on some days she was able to keep down everything she ate. These days were marked by lower levels of negative affect and higher levels of positive affect and a general absence of physical discomfort after eating.

Valerie's purging behavior appears to be maintained through the learned experience that it will alleviate physical discomfort and anxiety. Even though purging is not inherently rewarding (it does not make Valerie feel happy) it eliminates a range of negative experiences. Haedt-Matt and Keel (2015) recently documented that increases in negative affect preceded episodes on purging in purging disorder, and negative affect decreased after purging, supporting the role of negative reinforcement in maintaining this behavior.

In many cases, negative reinforcement is more powerful in maintaining a behavior than positive reinforcement is. Each time someone engages in the behavior, that person experiences both the actual and the perceived consequences. In positive reinforcement, the pleasurable consequence is experienced exactly as it is. In negative reinforcement, the consequence is experienced both as the actual elimination of something undesirable and as the prevention of something that is undesirable. For example, Valerie experiences her purging as reinforcing because it decreases her anxiety and because she believes it prevents her from gaining weight. One consequence is actually experienced; the other consequence is only perceived. Thus no matter what the real consequences of a negatively reinforced behavior are, each time people engage in the behavior they can conclude that they would have been worse off had they not engaged in it. This assumption may well be wrong; Valerie, for example, was much happier and healthier before she developed purging disorder. The only way to challenge this assumption is to stop the purging so that the person can learn that it does not make life better. However, in the presence of intense fear of gaining weight, giving up purging may seem to be too great a risk.

Punishment is a negative consequence that inhibits behavior. Eating, for example, can be very punishing for patients with AN and purging disorder. It can trigger anxiety, gastrointestinal discomfort, and water retention. Similarly, eating in front of others or wearing clothes that reveal weight and shape (e.g., shorts or a bathing suit) may trigger feelings of shame. These feelings may then inhibit patients with eating disorders from eating in the

presence of others and from joining activities in which their bodies will be exposed. In eating disorders it is often easy to perceive the behaviors in which patients engage. However, the behaviors in which they do not engage frequently remain hidden, and many of these inhibited behaviors contribute to significant psychosocial maladjustment.

Case Study: Jamie

Jamie's excessive food intake contributed to his excess weight. In addition, Jamie was far less physically active than he had been when he was younger. He attributed this change mostly to his demanding work schedule. However, his therapist observed that he seemed to have considerable time to devote to online gaming. Jamie acknowledged that one of the things he enjoyed about computer games is that his avatar could be muscular and lean and exhibit the physical strength and stamina he felt had slipped away from him over time. Although he wanted to join his friends in pickup games of basketball and soccer, he was too self-conscious about his weight to feel comfortable running in shorts on the court or field. Similarly, numerous gym memberships had gone unused because he felt too ashamed of his body to work out in front of others.

As with negative reinforcement, behavioral responses to punishment are driven by a desire to avoid negative consequences. The primary difference is that a negative reinforcer increases a behavior (e.g., purging), while a punishment decreases a behavior (e.g., exercising in Jamie's case). In both instances, a temperamental or personality style that predisposes an individual to experience negative emotions would increase the power of these consequences, and increased sensitivity to punishment would inhibit a wide range of behaviors (Harrison et al., 2010).

Farmer, Nash, and Field (2001) examined the association between reward sensitivity and specific bulimic symptoms (binge eating, self-induced vomiting, and purging) in 13 individuals with BN and 21 individuals with an "eating disorder not otherwise specified" according to the then-current *DSM-IV*. To measure reward sensitivity, participants engaged in a verbal learning task. An experimenter presented 80 index cards. A verb (e.g., *run*) appeared in the middle of each card, and six pronouns (*I, we, she, he, they,* and *you*) appeared in the lower left corner (see Figure 7.2). The order of the pronouns on

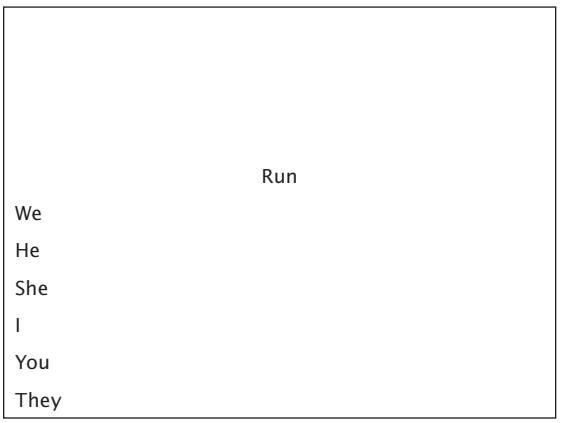


FIGURE 7.2 Depiction of card used in Farmer et al. (2001) study.

the card was randomized across cards. For each card, participants made up a sentence using the verb and one of the six pronouns. For the first 20 cards, experimenters gave no response to sentences. After the 20th card, the experimenter rewarded the participant for sentences that used either of the first two listed pronouns from the previous card. Rewards consisted of giving the participant 5 cents and saying "good." Reward sensitivity was measured as the increase in the use of rewarded words over baseline use of these words (measured during the first 20 cards). Results showed a significant positive association between reward sensitivity and frequency of vomiting and purging but not frequency of binge-eating episodes. These results were somewhat surprising as they suggest that vomiting and purging are linked to positive reinforcement while binge-eating is not, but these results await replication.

This kind of clean distinction between rewards and punishment is an artificial product of a laboratory environment. In real life, good things have some negative consequences, and bad things have some good consequences. Moreover, a patient with AN may receive positive reinforcement for weight loss but also may endure many negative consequences of her low weight. Similarly, patients who purge may experience relief from a range of negative experiences, but the purging itself contributes to shame and a host of medical problems (discussed in Chapter 8). Finally, avoiding social situations out of fear of public eating or of wearing revealing clothing leaves individuals with eating disorders feeling alone and isolated—the very opposite of what they hope to achieve by controlling their eating and weight. Thus an important question is how the combination of rewards and punishments influences the decision to engage in or inhibit a given behavior in eating disorders.

The Iowa Gambling Task (IGT; Bechara, Damasio, Damasio & Anderson, 1994) was developed to evaluate decision-making in response to rewards and punishments. In the IGT, participants are presented with four decks of cards, labeled A, B, C, and D, on a computer screen. Participants freely draw one card at a time from any of these decks over a series of trials, with the aim of earning as much money as possible. Most cards result in winning some money, but some cards can result in losses. Decks A and B tend to give large rewards, but even larger losses. Decks C and D tend to have small rewards but have even smaller punishments. As a result, in the long run it pays off to draw more cards from decks C and D than from decks A and B. Participants demonstrate learning by the extent to which they draw more cards from decks C and D than from A and B over the course of the task.

Across numerous studies, patient with AN have shown worse learning on the IGT than healthy control participants by making decisions that do not maximize reward over punishment (Abbate-Daga et al., 2011; Bodell et al., 2014; Brogan, Hevey, & Pignatti, 2010; Cavedini et al., 2004, 2006; Danner et al., 2012; Tchanturia et al., 2007). One study suggested that this problem does not improve with weight restoration (Bodell et al., 2014). Patients with BN also perform worse on the IGT compared to healthy controls (Boeka & Lokken, 2006; Brogan et al., 2010; Garrido & Subirá, 2013; Liao et al., 2009). Finally, patients with BED have worse IGT performance compared to healthy, normal weight controls (Davis, Patte, Curtis, & Reid, 2010), but do not differ from obese controls without an eating disorder (Brogan et al., 2010; Davis et al., 2010).

Given that eating disorders lead to distress, impairment, and an increased risk of medical complications, it can be difficult to understand why sufferers continue their self-destructive behaviors. Results using the IGT suggest that individuals with eating disorders may be more sensitive than healthy individuals to the immediate consequences of behaviors and less sensitive to long-term consequences. Thus many of the symptoms of eating disorders may be explained by their immediate or perceived consequences. Eating disorders become a trap for many individuals because the harmful long-term consequences are overshadowed by the immediate consequences. As the disorders lead to problems and distress, patients may be pushed further into disordered eating behaviors in an attempt to cope. Differences in vulnerability to developing eating disorders might therefore be related to how salient immediate consequences are, which in turn is related to the psychological dimensions of attention, perception, and evaluation.

Attention

Cognitive research on eating disorders has demonstrated that women with AN or BN show attentional biases associated with food- and body-related cues. That is, women with eating disorders pay more attention to information about food and body weight or shape than do women without eating disorders. Although some of this difference might be deliberate (e.g., women who are worried about becoming fat may choose to weigh themselves more frequently than do women who are unconcerned about their weight), food- and body-related information might also be more salient to women with eating disorders even at an unconscious level. Such an unconscious attentional bias might help explain their preoccupations with food, weight, and shape and why negative consequences pertaining to weight and shape seem so much more important to them than do negative consequences in other aspects of life.

The Stroop test has been used to evaluate attentional processes in women with eating disorders. In the classic Stroop test, a participant is asked to name the color of the ink that words are printed in when the words are names of colors. For example, if the word *green* is printed in red ink, the correct response is "red." This task is more difficult when the ink color is not that same as the word than when they agree, because in the former case information about the meaning of the word competes for attention with information about the color of the ink. The Stroop test has been modified for purposes of evaluating the salience of food- and body-related words. Words such as *thighs, hips, stomach, candy*, and *chocolate* are printed in different colors, and the speed with which participants can name the colors is assessed. Although results of these studies have been mixed (Van den Eynde et al., 2011), they do provide evidence of attentional biases (M. Cooper & Todd, 1997; Flynn & McNally, 1999; Formea & Burns, 1996; Johansson, Ghaderi, Hällgren, & Andersson, 2008; Jones-Chesters, Monsell, & Cooper, 1998; Lokken, Marx, & Ferraro, 2006; Lovell, Williams, & Hill, 1997). Compared with controls, women with eating disorders are slower to name the ink color for names of food or body parts than for names of neutral objects.

In a recent study, Blechert, Ansorge, and Tuschen-Caffier (2010) examined attentional biases not just toward general weight/shape cues but specifically toward images of one's own body. They took pictures of female participants wearing a neutral-colored

full-body leotard and then cropped the photos so that they showed the body only from the neck down. Participants with AN or BN showed no differences from healthy, weight-matched controls in the ability to recognize the pictures of their own bodies and to discriminate those pictures from pictures of other participants of similar weight.

Next, participants completed a dot-probe task. Dot-probe tasks involve attending to a dot in the middle of the screen and then shifting one's gaze as quickly as possible to one of two images that appear simultaneously on either side of the dot. In Blechert et al.'s (2010) study, rectangular boxes, one green and the other blue, appeared on either side of the dot. Participants randomly assigned to the blue-target condition were instructed to shift their gaze toward the blue rectangle. Participants randomly assigned to the green-target condition were instructed to shift their gaze toward the green target. The investigators used eye tracking to measure the speed with which participants were able to shift their gaze to the targeted box. No differences were observed across groups or between the blue and green target conditions on this task.

Finally, Blechert et al. (2010) presented pictures of bodies on either side of the dot immediately before the rectangles appeared. One picture was of the participant's own body, and the other was of another woman's body. As before, all participants were instructed to maintain their attention on the dot in the center of the screen until the rectangles appeared and then to shift their gaze to the target rectangle as quickly as possible. The authors hypothesized that if there is an attentional bias toward one's own body, then participants would be faster to shift their gaze to the target rectangle when it appeared on the side on which their own picture had appeared than when the target rectangle appeared on the side with the picture of another's woman body. The logic was that if the woman had been covertly attending to the side of the screen with her own picture, she would be able to respond more quickly when the target rectangle appeared on that side. Moreover, when the target rectangle appeared on the opposite side from her own picture, she would find it harder to shift her gaze away from the covertly attended location, slowing her response. On the other hand, if there is no attentional bias, then women should shift their gaze toward the target rectangle equally quickly regardless of where their own picture had appeared.

Results showed that women with AN shifted their gaze toward the target rectangle significantly faster when it matched the location of the picture of their own body than when it was on the opposite side from that picture. Further, the more negatively women with AN viewed their own bodies, the greater the attentional bias they showed toward the side showing their body. Women with BN tended to be *slower* to shift their gaze toward the correct rectangle when it appeared on the side where their own body had been, though this tendency did not reach statistical significance. Control women showed no attentional bias. There were also significant differences in attentional bias between women with AN and both control women and women with BN.

Blechert et al. (2010) concluded that the attentional bias toward one's own body may be a consequence of and maintenance factor for severe body image disturbance in women with AN. Women who view their bodies very negatively may increase their attention to their appearance to maintain vigilance over a perceived threat to their self-worth. Focusing

on one's own appearance may limit opportunities for corrections to one's body image based on observing what other women's bodies actually look like. This attentional bias could thus help maintain perceptual and cognitive distortions that characterize body image disturbance in AN.

Perceptual and Cognitive Distortions

Perceptual distortions and **cognitive distortions** are experiences and thoughts that do not correctly reflect reality. Both AN and BN are characterized by numerous such distortions (Peterson & Mitchell, 2001). Indeed, a perceptual disturbance in the experience of weight is included in the diagnostic criteria for AN (see Chapter 1).

Dichotomous thinking (also known as *black-and-white* thinking) is a cognitive distortion expressed in many of the features common to eating disorders. For example, individuals with eating disorders may see thin as "good" and fat as "bad." Similarly, foods become classified as either good or bad, and one's eating patterns are categorized the same way. Many diets encourage dichotomous thinking about foods—pizza, potato chips, cookies, and ice cream may all be bad foods to someone interested in losing weight. Conversely, foods such as celery, carrots, and diet sodas are low in calories and therefore good. In reality, no edible food is inherently good or bad—as evidenced by the fact that different diet plans have different bad foods. For example, on a low-fat diet bacon is a bad food, and grapefruit is a good food. However, on a low-carbohydrate diet bacon is good, and grapefruit is bad. (The roles of different nutrients, such as fat and carbohydrates, in eating disorders will be reviewed in more detail in Chapter 8.)

Such dichotomous, rule-bound thinking can be seductive for individuals in significant distress, because it greatly simplifies the world. When individuals experience increased stress, their ability to handle complex information diminishes (Vedhara, Hyde, Gilchrist, Tytherleigh, & Plummer, 2000). Unfortunately, dichotomous thinking does not adapt to changes in context. For example, in AN, losing weight is good and gaining weight is bad even when weight loss becomes dangerous. Similarly, bad foods are bad even when the social context and the initial portion size make consumption of those foods normal and desirable.

Cognitive rigidity in individuals with eating disorders reveals itself as a perseverative approach to problems. Patients with AN have a rigidly held belief that weight loss will bring happiness and freedom from the fear of becoming fat. As they lose more and more weight, however, they tend to become more depressed and more terrified of weight gain. Thus patients with eating disorders can engage in behaviors motivated to achieve a specific end, only to find that the opposite result occurs. However, these individuals do not re-evaluate the usefulness of their behaviors according to the consequences; they simply persist in believing that they "just haven't done enough" or "just haven't done it right." Cognitive rigidity has been observed in neuropsychological testing of patients with AN (Friederich & Herzog, 2011) and BN (Van den Eynde et al., 2011).

Patients with BN often come to treatment with the hope of eliminating binge-eating episodes so that they can successfully lose weight through dieting. As in Jean's case, they rarely view dieting as a problem.

Case Study: Jean

Jean believed that her binge eating and purging were caused by her decision to move in with her boyfriend and the resulting availability of forbidden foods. She contemplated asking him to stop buying these foods, but she was afraid of having to explain why. At first, she tried to talk him into going on a diet with her "to be more healthy." However, Jean's boyfriend was completely unconcerned about his weight or eating habits. He was able to eat a few cookies at a time, and as far as he knew, a package of cookies would last a month. Jean did not understand how he could show so much self-control. However, when asked, Jean was able to acknowledge that before her weight loss diet, she had been able to have one dessert after dinner at a restaurant and not lose control of her eating later. She had always thought that the reason she showed restraint in this situation was that the restaurant controlled how much food she got. However, she had to admit that the restaurant did not keep her from later going to a store and buying a package of cookies or a gallon of ice cream. So she had, at one time in her life, been able to eat dessert without losing control.

Jean's loss of control appears to be related in part to her beliefs about her ability to eat forbidden foods in moderation. Although she has been able to eat just one dessert, she has convinced herself that she can't do so when she is alone. Therefore her only choices are to eat nothing or eat everything (a case of dichotomous thinking). Unlike a patient with ANR, who rigidly abstains from eating, patients with bulimic symptoms shift back and forth between eating nothing and eating everything.

As a short-term approach to losing weight, abstinence from forbidden foods may yield some success, because it may be easier to avoid some foods altogether than to eat a small amount of those foods. Still, abstinence is not a flexible approach. Consistent with dichotomous thinking, one is either abstinent or not. Thus eating one piece of cake on a special occasion would count as a failure. Beyond the potential for dieting to trigger physiological weight-defending mechanisms that contribute to binge eating (see the discussion of Keys et al., 1950, in Chapter 5), it is likely that the cognitive processes involved in dieting contribute to binge eating. Indeed, would it even be possible to experience a loss of control over eating if one weren't attempting to control eating in the first place?

Polivy and Herman (1985) proposed that cognitive regulation of dietary intake (eating in response to rules about when to eat and what to eat rather than eating in response to hunger) introduces opportunities for loss of control over eating (disinhibition). Disinhibition could occur in response to a cognitive trigger, an affective trigger, or a pharmacological trigger. An example of a **cognitive disinhibitor** is attending a friend's birthday party and being offered a piece of birthday cake that is not allowed on a weight loss diet. After having the cake, dieters might think that they have "blown" their diets and might as well eat whatever they want for the rest of the evening. An example of an **affective disinhibitor** is a fight with a loved one that leads to frustration and sadness. In this case, one may use cognitive resources normally employed to restrict food intake to cope with distress. In addition, one may use eating as a source of comfort and justify it as a special treat. An example of a **pharmacological disinhibitor** is becoming intoxicated and losing track of what or how much one has eaten.

In each case, the cognitive control of food intake is interrupted, resulting in a loss of that control. Polivy and Herman (1985) argued that individuals who become chronic dieters, or *restrained eaters*, lose the ability to determine when they feel hungry or full. Thus sensations of hunger and satiation—physiological controllers of food intake—cannot take the place of cognitive control when it is interrupted, resulting in the consumption of an unusually large amount of food (Ruderman, 1986). According to this explanation, also known as the *restraint hypothesis*, Jean's binge-eating episodes are caused by her weight loss diet. Jean's diet creates the rule that she should eat no junk food. When she is alone in the apartment and eats a cookie, she has failed and there is no reason for her to stop eating. In addition, her dieting has disrupted her ability to eat in response to hunger and stop eating in response to satiation. Thus what was interpreted as alexithymia or poor interoceptive awareness in psychodynamic theory (see Chapter 6) is interpreted as a consequence of weight loss dieting in a cognitive–behavioral model like the restraint hypothesis.

A series of experimental studies have demonstrated the influence of dieting-related cognitions on food intake. In the classic study of this phenomenon, Spencer and Fremouw (1979) divided participants into restrained eaters and unrestrained eaters based on scores on a self-report measure of dietary restraint. Participants were then brought into a laboratory for a taste test (see Chapter 4 for a description of the basic taste-test study design). Prior to the taste test, all participants were asked to consume a milkshake. However, participants were randomized into one of two conditions. In one condition, they were told they were consuming a low-calorie milkshake. In the other condition, they were led to believe they were consuming a high-calorie milkshake. The milkshakes did not differ in actual nutritional content, so there would be no physiological differences in satiation. During the taste test, restrained eaters consumed significantly more in the "high calorie" than in the "low calorie" condition. This pattern was not observed in unrestrained eaters. (See Figure 7.3.) Thus the extent to which restrained eaters *believed* that they have broken their diets predicted subsequent food consumption during the taste test. Further, rather than compensating for the high-calorie shake by eating less (which would help them adhere more closely to their diets), restrained eaters ate more.

Another cognitive phenomenon relevant to eating disorders is **selective abstraction.** Selective abstraction occurs when a part comes to represent the whole (Beck, 1970). For example, Emily was able to acknowledge that several areas of her body were not fat (e.g., her shoulders and arms) and even expressed the desire to build muscle mass in her upper body. However, she perceived fat on her thighs, and this one region caused her to evaluate her whole body as being in danger of becoming fat. Selective abstraction is particularly likely among perfectionists, because if something would be perfect if not for one specific flaw, then that one flaw carries undue importance in evaluating the worth of the whole.

Neuropsychological tests suggest that selective abstraction in patients with AN may be linked to poor **central coherence**—a poor ability to see the forest for the trees. One test of central coherence is the Rey–Osterrieth Complex Figure task. In this task participants are asked to copy an abstract figure with several detailed features onto a blank sheet of paper. Later, participants are asked to draw the figure from memory. Although patients with AN provide excellent initial copies of the complex figure, their ability to reproduce

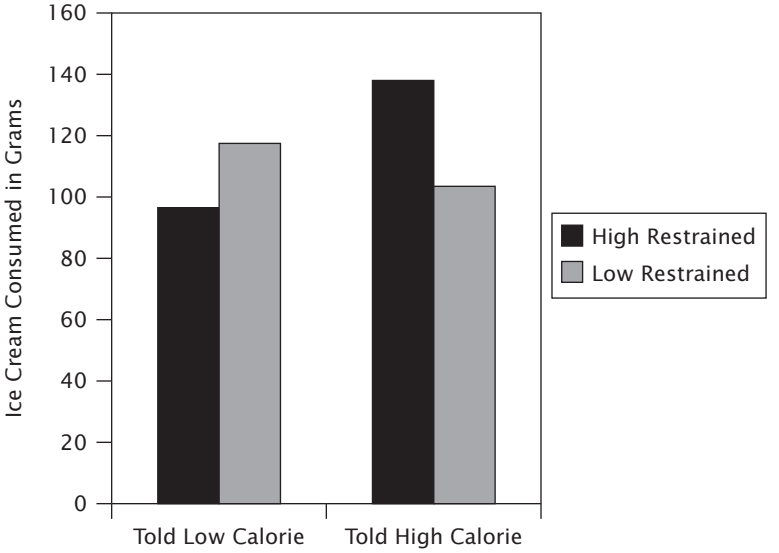


FIGURE 7.3 Average grams of ice cream consumed by high- and low-restrained subjects given high- and low-calorie instructions. Source: Spencer, J. A., & Fremouw, W. J. (1979). Binge eating as a function of restraint and weight classification. *Journal of Abnormal Psychology*, 88, 262–267. Reprinted with permission from the American Psychological Association.

the whole figure from memory is severely impaired (Lopez, Tchanturia, Stahl, & Treasure, 2008). Observations of how they approach this task suggest that when the figure is present they achieve great accuracy in copying by going from detail to detail but never incorporate the overall shape of the image or how the parts fit together in a whole (Lopez et al., 2008; Sherman et al., 2006). Thus patients with AN show a tendency to focus on specific details at the expense of global meaning. Of particular interest, these deficits are observed both in patients who have recovered from AN and in their unaffected sisters (Roberts, Tchanturia, & Treasure, 2013), suggesting that this difference in central coherence may be a risk factor that is transmitted in families rather than a consequence of illness.

Putting this information together with that from previous chapters, societal messages that contribute to disordered eating, such as images that convey the importance of being thin, may be particularly salient to individuals who develop eating disorders. In addition, the ways that individuals use and evaluate that information likely contribute to disordered eating. Rather than feeling good about successes, whether large or small, people with eating disorders have cognitive distortions that leave them vulnerable to suffering innumerable perceived failures with respect to their eating, their bodies, and their lives. Funneling general dysphoria into dissatisfaction with weight or shape (Haedt-Matt, Zalta, Forbush, & Keel, 2012) has been proposed as an etiological factor in the development of BN (Keel, Mitchell, Davis, & Crow, 2001). For adolescents, controlling weight and shape may seem like a manageable way to alleviate distress; however, for many, it becomes a trap. A vicious cycle develops in which general threats to self-evaluation are directed into a need to obtain or maintain a specific body weight or shape, as if doing so will solve all of life's problems. The few successes and numerous failures to control body weight and shape then negatively

influence self-evaluation and lead to increased efforts to alleviate distress through weight control (Heatherton & Baumeister, 1991).

One limitation in this line of reasoning is that the cognitive features characteristic of eating disorders may not have predated the onset of an eating disorder. Instead they may reflect an aspect or even a consequence of having an eating disorder. Research that suggests cognitive differences from healthy controls in women recovered from eating disorders offers some support for the idea that these thought processes predated and contributed to the onset of the eating disorder. However, this reasoning warrants caution as well. A consequence of having an eating disorder could remain after the eating disorder is in remission, much as a scar remains after a wound has healed.

Conclusion

Research has revealed a high degree of overlap across aspects of personality, learning, and perceptual and cognitive distortions associated with the development of eating disorders. This overlap may be explained by the role of personality in shaping how individuals perceive, react to, and interact with their environments. Thus an individual who is highly perfectionistic and constrained and who demonstrates high persistence and harm avoidance may show a cognitive style marked by rigidity, dichotomous thinking, and selective abstraction that makes it difficult to make decisions that balance positive and negative consequences. This person would be more vulnerable to social messages conveying the importance of being thin. He or she also may be more likely to persist in the pursuit of this ideal even when the rewards diminish in intensity and the costs increase. Conversely, an individual who is impulsive may experience cognitive disinhibition more readily than others do. This person might find it more difficult to resist the rewarding aspects of food and eating. In combination with higher reward and punishment sensitivity, such a personality may contribute to a vicious cycle of binge eating and purging. These patterns provide a fairly coherent explanation of symptomatic differences across eating disorders described in Chapter 1.

High levels of negative emotionality are characteristic of all eating disorders. Disordered eating symptoms may serve as a maladaptive solution to problems that elicit intense negative emotional states. The failure to recognize that the symptoms do not actually solve the problem may result from a combination of cognitive distortions and the greater salience of immediate and perceived consequences than of long-term actual consequences. Thus negative emotionality may be a common factor that bridges the different types of eating disorders.

Key Terms

Affective disinhibitor
Central coherence
Cognitive disinhibitor
Cognitive distortions

Constraint
Dichotomous thinking
Harm avoidance
Negative emotionality
Negative reinforcement
Novelty seeking
Operant conditioning
Persistence
Personality
Pharmacological disinhibitor
Positive emotionality
Positive reinforcement
Punishment
Reward dependence
Selective abstraction
Temperament

8

Biological Bases, Correlates, and Consequences of Eating Disorders

Research has revealed a variety of biological differences between individuals with eating disorders and those without eating disorders. Biological differences may represent risk factors that were present before the onset of eating disorders. However, just as eating disorders affect psychological function, they affect biological functions, and biological differences may reflect consequences of eating disorders. As discussed in Chapter 4, it would be unethical to directly test factors believed to cause eating disorders in humans by experimentally manipulating those factors to see if they really did cause a disorder. Much of the biological research on eating disorders therefore cannot disentangle causes from consequences. Thus this chapter reviews biological bases, correlates, and consequences of eating disorders together.

The first section of this chapter reviews the biological bases of appetite and weight regulation and reward systems in the brain, as well as associations between the functions of different neurochemicals that impact these systems and eating disorders. This review reveals how the brain influences hunger and satiation based on experimental studies in animals. No one would argue that eating can be explained fully as a response to hunger and satiety, as people often eat for reasons completely unrelated to biological needs, and this point will be explored in the review of the brain's reward systems. However, it would be equally unwise to completely discount the role of such mechanisms in the etiology or maintenance of eating disorders.

The second section of this chapter discusses the contribution of genes to the etiology of eating disorders. The section begins with an examination of data from behavioral genetic studies of families, twins, and adopted versus biological siblings. (See Chapter 4 for discussion of the methods used in these studies.) It then reviews results of molecular genetic studies designed to identify specific genes that increase the risk of eating disorders.

The third section reviews known consequences of eating disorders. Because eating is a basic biological function required to sustain life, disruption of eating has profound effects

on biological systems throughout the body. This fact explains the ambiguity associated with interpreting results from studies that attempt to reveal the biological bases of eating disorders by examining individuals currently suffering from these disorders.

Brain Function and Eating Disorders

Appetite and Weight Regulation

The **hypothalamus** (Figure 8.1) is a structure in the brain that plays a central role in appetite and weight control. It can be divided into different sections named for their locations in relation to each other (Figures 8.1 and 8.2). These sections are associated with different effects on appetite and weight. Surgically damaging the **ventromedial hypothalamus** in animals produces increased food intake and significant obesity (Hetherington & Ranson, 1942). In contrast, surgically damaging the **lateral hypothalamus** produces dramatic decreases in food intake and weight loss (Anand & Brobeck, 1951; Teitelbaum & Stellar, 1954). Electrical stimulation of these brain regions produces the opposite effects (Kandel, Schwartz, & Jessell, 1991). These results suggest that the ventromedial hypothalamus is responsible for inhibiting appetite and food intake and that the lateral hypothalamus is responsible for increasing appetite and food intake. In healthy individuals these areas appear to work together to maintain a balance in weight and appetite. They are thought to be important in understanding satiety function as it may relate to eating pathology.

In addition to regulating eating, the hypothalamus regulates body functions such as sexual activity, circadian rhythm, thermal regulation, and fluid balance. Thus, for a structure that is approximately the size of an almond and represents only 1/300th of the brain's weight,

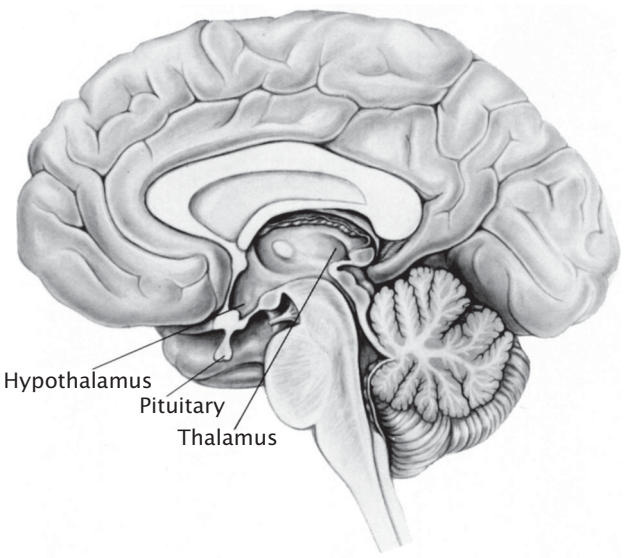


FIGURE 8.1 The location and structure of the hypothalamus. Medial view showing the relationship of the hypothalamus to the pituitary and thalamus. Source: Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (1991). *Principles of Neural Science* (Third Edition). New York, NY: Elsevier Science Publishers, 738. Reprinted with permission from Elsevier.

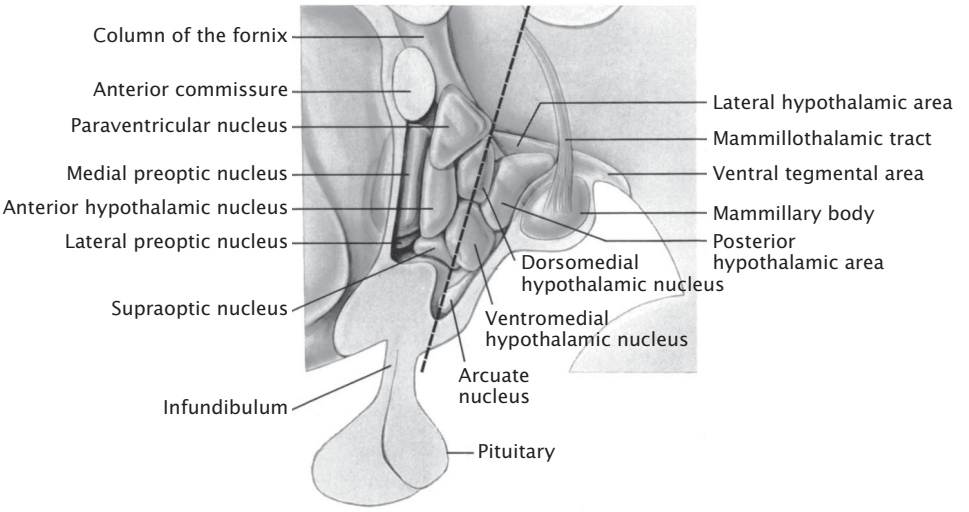


FIGURE 8.2 The location and structure of the hypothalamus. Medial view showing positions of the main hypothalamic nuclei. Some nuclei are visible only in the frontal view in Figure 8.3. Source: Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (1991). *Principles of Neural Science* (Third Edition). New York, NY: Elsevier Science Publishers, 738. Reprinted with permission from Elsevier.

the hypothalamus has a lot of important functions central to survival. The location of the hypothalamus is key to the roles it plays. It is located near the pituitary gland at the base of the brain. The hypothalamus and pituitary gland are involved in two important systems—the **hypothalamic–pituitary–adrenal (HPA) axis** and the **hypothalamic–pituitary–gonadal (HPG) axis.** The HPA axis has been implicated in responses to stress, and the HPG axis is involved in the release of sex hormones and the process of maturation. Both functions are altered in patients with eating disorders. Another key to the hypothalamus's ability to fulfill its various functions is its ability to respond to signals released within the brain as well as signals that arise from the body's long-term energy balance (its weight) and short-term energy balance (recent food intake). The following sections review evidence of altered neurophysiology in eating disorders that may contribute to alterations in brain function—specifically, evidence of dysfunction of **neurotransmitters** and **neuropeptides** related to appetite and weight.

Neurotransmitters and Eating Disorders

Neurotransmitters are chemicals that facilitate communication between brain cells (**neurons**). Examples of neurotransmitters include **serotonin** (also called 5-hydroxytryptamine, **5-HT**), **norepinephrine,** and **dopamine.** Food intake is associated with release of these three neurotransmitters in the hypothalamus (Fetissov, Meguid, Chen, & Miyata, 2000). Activity of 5-HT in the medial hypothalamus decreases food intake (Fetissov et al., 2000), suggesting that 5-HT is important to that structure's role in reducing appetite and weight. Activity of dopamine and norepinephrine in the hypothalamus inhibits function (Fetissov et al., 2000). Specifically, activity of dopamine and norepinephrine in the lateral hypothalamus decreases food intake, whereas dopamine and norepinephrine activity in the medial hypothalamus increases food intake. Among these neurotransmitters, 5-HT has received

the most attention in the field of eating disorders, possibly because the only medication approved by the U.S. Food and Drug Administration for the treatment of BN influences 5-HT function in the brain.

Serotonin (5-HT)

Animal and human studies have demonstrated that 5-HT plays an important role in the regulation of mood, appetite, and impulse control. Diminished serotonin function is associated with dysphoria, increased appetite, and decreased impulse control. Because mood, appetite, and impulsivity are altered in patients with eating disorders, 5-HT became a prime candidate in attempts to understand the biological correlates (and potential causes) of eating disorders. Specifically, research on BN has focused on the hypothesis that inadequate 5-HT function contributes to binge eating, and recent research on AN has focused on overfunction of 5-HT as a potential explanation of self-starvation (Kaye, 2008). Less research has focused on neurotransmitter function in BED, because of its relatively recent introduction as an official eating disorder.

An early hypothesis proposed that inadequate 5-HT function produced "carbohydrate craving" that caused binge-eating episodes (Wurtman & Wurtman, 1986). Dieting was thought to contribute to carbohydrate craving, because many weight loss diets of the 1970s emphasized restricted carbohydrate intake. Serotonin is not found in food, and it cannot cross from the blood into the brain. However, **tryptophan,** an amino acid required to make 5-HT, is found in food and can cross the blood–brain barrier. So diets that diminish tryptophan intake could lead to diminished 5-HT production, whereas diets high in carbohydrates that might increase tryptophan could enhance 5-HT production. Thus binge-eating episodes that typically consisted of high-carbohydrate foods (e.g., cookies, cakes, or chips) were thought to represent attempts at self-medication for low 5-HT function. Low 5-HT function would certainly explain the dysphoric mood and large appetites of women with BN. Later studies failed to support key hypotheses of the carbohydrate-craving model. For example, blood concentrations of tryptophan did not differ between women with BN and healthy controls in a fasting state (Brewerton et al., 1992; Lydiard et al., 1988; Weltzin, Fernstrom, Fernstrom, Neuberger, & Kaye, 1995).

Nonetheless, research on the brains of women with AN and BN suggests that 5-HT function plays a role in eating disorders (Frank & Kaye, 2012). **Positron emission tomography (PET)** is a method of brain imaging that permits researchers to view the distribution and density of neurotransmitter **receptors** in the brains of living participants. For neurotransmitters to influence brain activity, they must be transmitted by one neuron and received by another at the junction between neurons, referred to as a synapse. The number and sensitivity of receptors for a neurotransmitter influence how well it works in the brain. Compared with control subjects, individuals with current AN or BN (Bailer et al., 2007; Tiihonen et al., 2004) as well as individuals recovered from AN or BN (Bailer et al., 2010; Galusca et al., 2008) have increased densities of a type of 5-HT receptor—the postsynaptic 5-HT_{1A} receptor (i.e., a receptor on the signal-receiving side of the synapse) in several brain regions. Additionally, PET studies have found decreased densities of a different receptor, 5-HT_{2A}, in individuals recovered from AN or BN (Bailer et al., 2004; Frank et al., 2002;

Kaye et al., 2001). However, densities of the 5-HT_{2A} receptor have not been found to differ between controls and patients with *current* AN or BN (Bailer et al., 2007).

The meaning of these results is far from straightforward. Receptor function depends on the type and location of that receptor. For example, 5-HT_{1A} receptors located presynaptically (i.e., on the signal-sending side of the synapse) decrease 5-HT communication between neurons, because these receptors absorb 5-HT released by the neuron they are found on—a process known as **reuptake**—before the 5-HT can bind to receptors on the postsynaptic neuron. Elevated density of these 5-HT *autoreceptors* would therefore reduce 5-HT function. Conversely, postsynaptic 5-HT_{1A} and 5-HT_{2A} receptors facilitate effects of 5-HT. Thus reduced density of these receptors in patients recovered from eating disorders may suggest further depletion of 5-HT function in their brains. However, the absence of differences in 5-HT_{2A} receptor density between patients during the ill state and controls is a mystery.

Jimerson, Lesem, Kaye, and Brewerton (1992) introduced a model that may explain findings concerning 5-HT function in BN. In this model, periodic binge-eating episodes cause sudden increases in 5-HT in the brain. These increases cause postsynaptic 5-HT receptors to become less sensitive (a process known as **downregulation**). When a patient with BN resumes dietary restriction, 5-HT levels normalize or decrease. However, the patient's 5-HT receptors are now less responsive to even normal levels of 5-HT. This situation could be worsened if the density of presynaptic 5-HT autoreceptors also increased as a consequence of periodic surges in 5-HT concentrations, as this would reduce the amount of 5-HT that reaches postsynaptic receptors when 5-HT levels subsided. Although sustained low concentrations of 5-HT could eventually lead to increased sensitivity of 5-HT receptors (**upregulation**), downregulation occurs more readily than upregulation in neurotransmitter systems. For example, downregulation of opioid receptors in response to heroin use (which causes tolerance to the drug) can occur over hours or days; by contrast, upregulation of opioid receptors to previous levels of function can take more than three weeks (i.e., until the end of the withdrawal period). If binge-eating episodes are occurring several times per week, then there may be repeated insult to 5-HT receptors that cannot be reversed by the periods of dietary restriction between episodes. Thus Jamie's experience of his binge episodes as representing an addiction may be accurate (see Chapter 1). Other similarities between processes that contribute to addiction and eating disorders are reviewed below in the section on the neural circuitry of reward.

Just as binge eating in BN has been attributed to low 5-HT function, fasting in AN has been attributed to high 5-HT function. However, as reviewed below, most studies suggest diminished 5-HT levels when AN patients are ill. To explain this paradox in AN, self-starvation has been interpreted as a form of self-medication that helps to reduce 5-HT to more tolerable levels (Kaye et al., 2008). According to this model, individuals who go on to develop AN initially have high 5-HT function that contributes to anxiety and rigidity and success in restricting food intake. Self-starvation then diminishes 5-HT levels. During illness, patients with AN demonstrate lower concentrations of the major metabolite of 5-HT, 5-hydroxyindoleacetic acid (5-HIAA), in their cerebrospinal fluid (Frank & Kaye, 2012). 5-HIAA is a leftover product from 5-HT activity that is washed out into cerebrospinal fluid. For example, if you wanted to measure how many candy bars a person ate without

directly observing the person, you could count the number of candy wrappers in the trash. Examining 5-HIAA in the cerebrospinal fluid is a very similar approach. One challenge in testing this model is that it is unknown whether 5-HT function is elevated before the onset of AN. Measures of 5-HT function (e.g., using radioactive isotopes in PET scans or spinal taps for cerebrospinal fluid) are expensive and invasive and cannot be easily incorporated into prospective longitudinal designs to predict who will develop AN in the future. Further, a potential logical problem with this hypothesis is that patients with AN, like the participants in the Keys et al. (1950) study, tend to become more rigid and overcontrolled as they lose weight. Thus, increased rigidity occurs as their extreme dietary restriction and weight loss causes their 5-HT levels to decrease. Unlike the 5-HT hypothesis for BN, in which vacillations between dietary restriction and binge-eating episodes could create a vicious cycle, continued dietary restraint motivated by premorbidly elevated levels of 5-HT should resolve as the 5-HT level normalizes.

Thus while the idea that diminished 5-HT contributes to binge episodes in BN and excessive 5-HT contributes to self-starvation in AN has intuitive appeal, it is difficult to reconcile this explanation with the findings of diminished 5-HT function in both illnesses. It also would be naive to expect the function of a single neurotransmitter to explain any complex mental disorder.

Moreover, neurotransmitter function within the brain relies on signals regarding energy state that the brain receives from the body. As noted above, 5-HT does not cross the blood–brain barrier. However, other signals from the body do. The following section reviews the role of neuropeptides in signaling the brain about the body's need to eat and how the function of these chemicals is altered in individuals with eating disorders.

Neuropeptides and Eating Disorders

Neuropeptides function similarly to neurotransmitters but are larger. Relevant neuropeptides include **leptin, neuropeptide Y, cholecystokinin, peptide YY**, **ghrelin,** and **glucagon-like peptide 1**, among others. Leptin, cholecystokinin, peptide YY, and glucagon-like peptide 1 all work to decrease appetite through activation of the medial hypothalamus. In contrast, neuropeptide Y and ghrelin increase appetite by stimulating the lateral hypothalamus.

Leptin

The neuropeptide leptin provides a negative feedback loop in the brain's control of weight and food intake. Receptors for leptin have been found in the **arcuate nucleus** and **paraventricular nucleus** of the hypothalamus (see Figures 8.2 and 8.3) as well as in the ventromedial hypothalamus. Leptin is produced by the *ob* gene and is released from fat tissue in the body. Thus the more fat there is in the body, the more leptin is circulating in the blood. When an organism loses fat, leptin levels decrease. Higher levels of leptin decrease sensitivity to signals that normally lead to increased food intake, while lower levels of leptin increase sensitivity to these signals.

Mice with a mutated *ob* gene (known as *ob/ob* mice) are obese, weighing three times more than normal mice and having five times the amount of body fat. (Indeed, the gene for

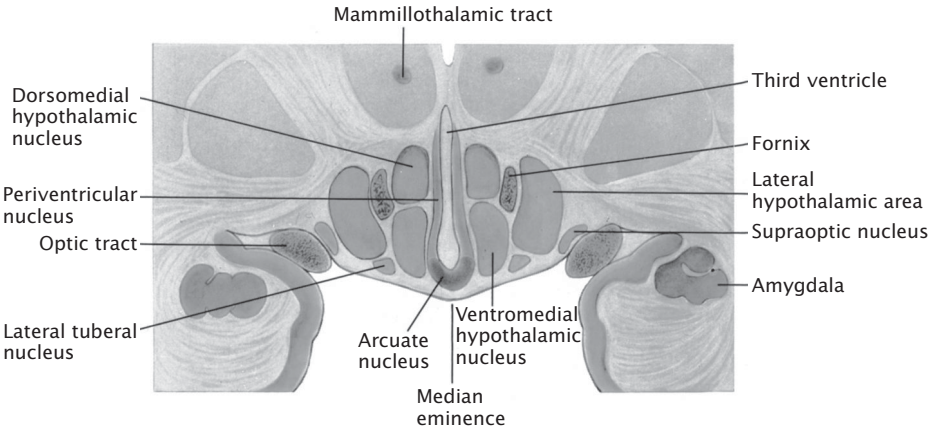


FIGURE 8.3 The location and structure of the hypothalamus. Frontal view of the hypothalamus (section along plane shown in Figure 8.1). Source: Kandel, E. R., Schwartz, J. H., & Jessell, T. M. (1991). *Principles of Neural Science* (Third Edition). New York, NY: Elsevier Science Publishers, 738. Reprinted with permission from Elsevier.

leptin was named after this effect: *ob* stands for *obesity*.) Injections of leptin produce weight loss in these mice (Friedman & Halaas, 1998), and leptin decreases weight by reducing food intake (Friedman & Halaas, 1998; Heymsfield et al., 1999; Schwartz, Baskin, Kaiyala, & Woods, 1999). One subtype of obesity in humans is caused by mutation of the human form of the *ob* gene and has been successfully treated by leptin injections. However, most individuals who are obese have high leptin concentrations, and there is evidence of reduced leptin receptor sensitivity in obese BED (Monteleone, Fabrazzo, Tortorella, Fuschino, & Maj, 2002).

Not surprisingly, several studies have demonstrated decreased leptin concentrations in patients with AN compared with controls (Di Carlo et al., 2002; Föcker et al., 2011; Germain, Galusca, Le Roux, & Bossu, 2007; Hebebrand et al., 1997; Lear, Pauly, & Birmingham, 1999; Mantzoros, Flier, Lesem, Brewerton, & Jimerson, 1997; Monteleone, Di Lieto, Tortorella, Longobardi, & Maj, 2000; Monteleone et al., 2002; Tolle et al., 2003). This decrease is likely a consequence rather than a cause of weight loss because leptin levels improve after recovery from AN (de Alvaro et al., 2007; Manara, Manara, & Todisco, 2005; Oświecimska, Ziora, Geisler, & Broll-Waśka, 2005; van Elburg, Kas, Hillebrand, Eijkemans, & van Engeland, 2007). Some studies of women recovering from AN have found that leptin concentrations normalize before weight reaches a normal level (Hebebrand et al., 1997; Holtkamp et al., 2003; Mantzoros et al., 1997). One study found that when leptin levels at treatment discharge were higher than expected based on BMI, AN patients were more likely to experience future weight loss and relapse (Holtkamp et al., 2004). Thus it is possible that women with AN receive physiological signals indicating normal weight before they actually reach normal weight. If so, this phenomenon could contribute to their ability to maintain below-normal weights.

Given the association between body weight and leptin concentration, one would expect normal-weight women with BN to have normal concentrations of leptin. However, several studies have demonstrated diminished concentrations of leptin in women with BN compared with healthy controls (Brewerton, Lesem, Kennedy, & Garvey, 2000; Jimerson, Mantzoros, Wolfe, & Metzger, 2000; Jimerson, Wolfe, Carroll, & Keel, 2010; Monteleone,

Di Lieto, Tortorella, Longobardi, & Maj, 2000; Monteleone et al., 2002). By contrast, some studies have reported elevated leptin levels in individuals with BED (Monteleone et al., 2000, 2002) and night eating syndrome (Stunkard, 2000) compared with levels in healthy-weight controls. Contradictory results have emerged from comparisons between individuals with BED and obese controls, with BED being associated with lower leptin (Brandao et al., 2010), higher leptin (Adami, Campostano, Cella, & Scopinaro, 2002), and no significant differences (Geliebter, Gluck, & Hashim, 2005). As with BN, leptin levels are lower in patients with purging disorder compared with controls (Jimerson et al., 2010). Diminished leptin in BN and reduced leptin receptor sensitivity in BED (Monteleone et al., 2002) might contribute to binge-eating episodes by increasing the physiological drive to eat. This effect might be mediated by leptin's influences on neuropeptide Y function, discussed next.

Neuropeptide Y

Neuropeptide Y increases food intake and is inhibited by leptin. Thus, lower leptin contributes to higher neuropeptide Y, which helps to increase food intake in response to weight loss. Studies have found that concentrations of neuropeptide Y are elevated in patients with AN (Kaye, Berrettini, Gwirtsman, & George, 1990; Sedláčková et al., 2011) and normalize with recovery (Gendall, Kaye, Altemus, McConaha, & La Via, 1999; Kaye et al., 1990; Oświecimska et al., 2005). Consistent with finding lower leptin concentrations in BN as described above, one study has reported elevated neuropeptide Y concentrations in patients with BN (Sedláčková et al., 2011).

After a high-carbohydrate breakfast, patients with AN and BN failed to show a decrease in neuropeptide Y observed in controls (Sedláčková et al., 2011). This finding shows that patients with eating disorders not only may have alterations in fasting levels of neuropeptides that influence hunger and satiation but also may have altered responses to food intake that could further contribute to disrupted eating behavior. Importantly, body weight does not change dramatically throughout the day, implying that changes in leptin do not directly influence initiation or cessation of food intake in a given meal or binge episode. To understand dynamic influences on food consumption during eating episodes, it is important to consider the role of meal-related signals. The following sections review neuropeptides that are released by the gut depending on food intake and that are directly implicated in the initiation and cessation of eating.

Cholecystokinin

Cholecystokinin is released in the small intestine following food ingestion (Gibbs, Young, & Smith, 1972). Like 5-HT, cholecystokinin does not cross the blood–brain barrier. However, it binds to receptors on the **vagus nerve** in the stomach (Gibbs & Smith, 1977; Robinson, McHugh, Moran, & Stephenson, 1988). The vagus is a cranial nerve that sends signals directly to the brain, and stimulating the vagus nerve stimulates neurons projecting from the brainstem to various parts of the brain, including the ventromedial hypothalamus (Kandel, Schwartz, & Jessell, 1991). Postmeal release of cholecystokinin should therefore trigger brain mechanisms inhibiting appetite and food intake. In addition, cholecystokinin causes contraction of the **pyloric sphincter,** a muscle that controls the rate at which food

passes from the stomach to the small intestine (Moran, Robinson, & McHugh, 1985). As a person eats, the food in the stomach causes the stomach to expand. This triggers gastric stretch receptors that also stimulate the vagus nerve. Cholecystokinin enhances this process by slowing the rate at which food can pass from the stomach into the small intestine. Consistent with these mechanisms, administration of cholecystokinin has been shown to trigger satiation in animals (Antin, Gibbs, Holt, Young, & Smith, 1975; Gibbs, Young, & Smith, 1973) and humans (Kissileff, Pi-Sunyer, Thornton, & Smith, 1981; Greenough, Cole, Lewis, Lockton, & Blundell, 1998) and to produce ratings of increased fullness in humans (Stacher, Bauer, & Steinringer, 1979; Greenough et al., 1998).

Several studies have demonstrated reduced cholecystokinin response to food in women with BN compared with controls (Devlin et al., 1997; Geracioti & Liddle, 1988; Keel, Wolfe, Liddle, De Young, & Jimerson, 2007; Pirke, Kellner, Friess, Krieg, & Fichter, 1994) and with patients with purging disorder (Keel, Wolfe, et al., 2007). Thus another reason that women with BN may engage in large binge-eating episodes is that they need more food to feel satiated. Studies of cholecystokinin function in AN have produced inconsistent results (A. C. Prince, Brooks, Stahl, & Treasure, 2009; Tong & D'Alessio, 2011). Studies of BED (Geliebter, Yahav, Gluck, & Hashim, 2004) and purging disorder (Keel, Wolfe, et al., 2007) indicate normal cholecystokinin response to food intake.

Ghrelin, Glucagon-like Peptide 1, and Peptide YY

Like levels of cholecystokinin, levels of ghrelin, glucagon-like peptide 1, and peptide YY change in relation to acute changes in food intake. Ghrelin, which is released from the stomach, exhibits diurnal variation, with the highest levels observed in the morning when the body is in a fasting state. Ghrelin levels decrease dramatically with food intake and then rise slowly during the periods leading up to subsequent meals during the day. Ghrelin triggers food intake in animals and humans and is linked to subjective reports of hunger in humans. Based on its time course and its effects in experimental studies in animals, ghrelin is considered a hunger hormone that stimulates eating.

Glucagon-like peptide 1 shows a pattern of release and function very similar to that of cholecystokinin and likewise triggers stimulation of the vagus nerve to stimulate satiety centers of the brain. Levels of cholecystokinin and glucagon-like peptide 1 peak within 30 minutes after the end of a meal. Cholecystokinin and glucagon-like peptide 1 are considered short-term satiation peptides that help signal when an eating episode should stop.

Peptide YY is released from lower in the intestinal tract than cholecystokinin and glucagon-like peptide 1, and its levels rise more slowly than levels of those neuropeptides. Peptide YY levels peak approximately 50–90 minutes after the end of a meal and do not subside until approximately two hours after the meal. Peptide YY is seen as a satiety peptide that delays the onset of the next eating episode (e.g., the onset of lunch after breakfast or the onset of dinner after lunch) rather than as a satiation peptide that terminates a current eating episode.

Studies of AN patients suggest that they have higher fasting concentrations of ghrelin than controls (A. C. Prince et al., 2009; Tong & D'Alessio, 2011), which may be a consequence of their dietary restriction. In contrast, findings for fasting ghrelin concentrations

in BN have been mixed (A. C. Prince et al., 2009). Patients with BN may experience a less robust decrease in ghrelin after food intake than control participants (Kojima et al., 2005; Monteleone et al., 2005). For AN, findings on ghrelin response to food intake have been inconsistent (A. C. Prince et al., 2009; Tong & D'Alessio, 2011). Studies of BED have also produced mixed results: One study found no differences in fasting ghrelin concentrations or in ghrelin response to food intake between control and BED participants (Munsch, Biedert, Meyer, Herpertz, & Beglinger, 2009), while another suggested that BED patients have lower fasting concentrations of ghrelin and a less robust decrease in ghrelin after food intake (Geliebter, Hashim, & Gluck, 2008). The disrupted circadian rhythm in night eating syndrome is associated with disruptions in circadian release of ghrelin (K. C. Allison et al., 2005; Rosenhagen, Uhr, Schüssler, & Steiger, 2005; Goel et al., 2009; Birketvedt, Geliebter, Kristiansen, Firgenschau, Goll, & Florholmen, 2012).

Research indicates lower levels of glucagon-like peptide 1 in women with BN than in controls (Naessén, Carlström, Holst, Hellström, & Hirschberg, 2011; Dossat, Bodell, Williams, Eckel, & Keel, 2015). Patients with BN have also been found to have significantly lower glucagon-like peptide 1 levels than do patients with purging disorder (Dossat et al., 2015). Patients with AN had higher levels of glucagon-like peptide 1 than controls in one study (Germain et al., 2007), while another found that they had lower-than-control levels of this neuropeptide (Tomasik, Sztefko, & Starzyk, 2004). No differences have been reported in glucagon-like peptide 1 levels between controls and women with BED (Geliebter et al., 2008) or purging disorder (Dossat et al., 2015).

For peptide YY, two studies have supported blunted responses to food intake in BN compared with controls (Kojima et al., 2005; Monteleone et al., 2005), while another observed no difference in peptide YY response (Devlin et al., 2012). A significantly decreased peptide YY response to food intake in AN patients was found in one study (Stock et al., 2005), a significantly increased response in another (Nakahara et al., 2007), and no significant difference in a third (Otto et al., 2007). For BED, one study found no differences from controls in postmeal peptide YY release (Geliebter et al., 2008), while another suggested an elevated peptide YY response (Munsch et al., 2009).

Taken together, results suggest that AN is consistently associated with lower leptin, higher neuropeptide Y, and higher ghrelin concentrations than seen in controls, all of which should promote increased hunger and food intake. However, patients with AN often deny hunger and they restrict their food intake. While these differences may not explain self-starvation in AN, they might contribute to risk for binge eating in the illness (A. C. Prince et al., 2009). Mixed results have emerged for other neuropeptides in AN.

For BN, the picture is clearer. Despite maintaining a normal weight, individuals with BN have lower leptin concentrations, higher neuropeptide Y concentrations, and less robust changes in the neuropeptides that alleviate hunger (ghrelin) and produce short-term satiation (cholecystokinin and glucagon-like peptide 1) and long-term satiety (peptide YY) compared to controls. Although it is unclear whether these differences were present before the onset of BN, all would contribute to maintenance of binge eating in the disorder.

Few consistent results concerning neuropeptides have emerged to explain the unique patterns of disordered eating behavior in BED, night eating syndrome, and purging disorder.

For BED, one challenge is distinguishing alterations in neuropeptide levels and responses that may be part of the eating disorder versus those that are linked to obesity, making it is important to employ a weight-matched control group. However, doing so creates a particularly high threshold for identifying alterations uniquely associated with binge eating, given that much of the research on neuropeptides has indicated considerable overlap between factors that regulate eating behavior and weight.

Neural Circuitry of Reward

As noted at the beginning of this chapter, people eat for reasons beyond physical need. One major factor that influences food intake is that eating feels good. Indeed, food is a natural reinforcer—that is, a stimulus that is rewarding without any prior training or exposure. Because food is so central to our survival, we are born with the hardwired capacity to orient toward and obtain food and to learn associations between the behaviors that lead to food and external cues. Just as there are brain regions that regulate energy balance and respond to internal signals by signaling when we need to eat more food, there are brain regions centrally involved in helping us learn cues that signal rewards (including food). Thus researchers have posited that individual differences in this neural circuitry may contribute to risk for eating disorders.

The release of dopamine from the **ventral tegmental area** to the **nucleus accumbens** of the brain is centrally involved in the experience of reward as well as in learning cues that signal the availability of a reward. These regions have receptors for both leptin and glucagon-like peptide 1, and both peptides reduce the activity of this dopaminergic reward pathway. In addition to this basic pathway, which is present across many species, circuits in or connected to regions involved with emotional salience (the amygdala), detecting conflict (the anterior cingulate cortex), integrating bodily senses with emotional experience (the insular cortex), and decision-making (prefrontal cortex) have been posited to be involved in risk for developing eating disorders.

Functional magnetic resonance imaging (fMRI) has been used to examine the activation of brain regions and how regions act together during exposure to stimuli that are potentially relevant to eating disorders (e.g., pictures of delicious food vs. neutral objects; images of bodies of different weights and shapes, and receipt of a sip of a chocolate milkshake versus a tasteless solution). FMRI also has been used to measure activation during tasks that measure learned associations between cues and relevant stimuli (Frank & Kaye, 2012).

Fladung and colleagues (2010) asked women with AN and healthy controls to evaluate images of bodies that were underweight, of normal weight, or overweight in relation to their own bodies while undergoing fMRI. Women with AN reported a preference for thin bodies and showed the greatest activation of the ventral striatum (which includes the nucleus accumbens) while viewing thin bodies. In contrast, controls reported a preference for normal-weight bodies and showed the greatest ventral striatum activation while viewing normal-weigh bodies. Brooks and colleagues (2011) compared the activity of several brain regions when food or nonfood images were presented to women with AN, women with BN, and controls. Women with BN showed less activation of the insula in response to food images than controls did; no differences were found between controls and AN participants.

In contrast, Schienle, Schäfer, Hermann, and Vaitl (2009) found greater insula activation in response to food images in women with BN than in controls, as well as greater activation of the anterior cingulate cortex. In the same study, patients with BED showed greater activity in the orbitofrontal cortex while viewing food images than did normal-weight controls, overweight controls, and patients with BN.

Using an associative learning task, Bohon and Stice (2011, 2012) had participants with BN and controls perform a series of tasks in which cues were paired with the delivery of a sip of chocolate milkshake or a sip of a tasteless solution. Periodically, the cue for the milkshake was followed by a sip of the tasteless solution and the cue for the tasteless solution was followed by a sip of milkshake. These conditions were designed to distinguish between anticipation of receipt of the milkshake versus actual receipt of the milkshake. Compared with controls, women with BN showed a trend for less activation in the insula both when anticipating the milkshake and in response to receiving the milkshake (Bohon & Stice, 2011). This result suggests that individuals with BN have reduced sensitivity to the emotionally reinforcing properties of food, which could contribute to needing to eat more food to produce a desired level of reinforcement. However, the effect was not statistically significant, and there may be no difference between women with BN and controls.

The above experiments represent a small selection of studies examining reward processing in eating disorders, which is an emerging focus of research in this field.

Summary of Brain Function and Eating Disorders

The hypothalamus is responsible for the regulation of food intake and weight. The lateral hypothalamus is associated with increasing eating and weight, and the paraventricular and ventromedial hypothalamus are associated with decreased eating and weight. However, these basic functions can be activated or inhibited depending on what neurochemical is active in a given area. Given these complex associations, it is not surprising that numerous inconsistent results have been found concerning neurochemical correlates of eating disorders. Further, given that many of the observed neurochemical differences between individuals with eating disorders and healthy individuals disappear after recovery, results from studies of neurophysiological function in eating disorder patients may represent consequences of the illnesses more than causes. Studies of reward circuitry in the field of eating disorders remain in their infancy but suggest that the overt attitudes and behaviors expressed by individuals with eating disorders are associated with patterns of activation in their brains that differ from those of healthy controls. However, as with physiological studies of eating disorders, it can be difficult to disentangle cause from effect.

The limitations of cross-sectional comparisons for inferring causation are not a problem in genetic studies of eating disorders. While an eating disorder may cause reductions in leptin, it cannot cause someone to be a certain kind of twin nor can it cause someone to carry a specific variant of a gene. Guided by findings regarding physiological abnormalities in eating disorders, the following section explores whether these abnormalities may have genetic bases that could contribute to the development of eating disorders.

Genetic Contributions to Eating Disorders

Chapter 6 discussed how children may learn disordered eating behaviors and attitudes in their rearing environment. However, families share genes as well as environments, and eating disorders run in families (as described briefly in Chapter 6). In studies of the heredity of eating disorders, the individual affected with the disorder is referred to as the **proband.** Biological relatives of eating disorder probands are 5 to 12 times more likely to have an eating disorder than are individuals in the general population or relatives of individuals without eating disorders (Lilenfeld et al., 1998; A. Stein et al., 1999; Strober, Freeman, Lampert, Diamond, & Kaye, 2000; Strober et al., 1990; Strober, Morrell, Burroughs, Salkin, & Jacobs, 1985; Woodside, Field, Garfinkel, & Heinmaa, 1998). First-degree female relatives (e.g., mothers, sisters, and daughters) of probands are more likely to suffer from eating disorders than second-degree female relatives (e.g., aunts, grandmothers, and granddaughters) or third-degree relatives (e.g., cousins) (Strober, Freeman, Lampert, Diamond, & Kaye, 2001; Woodside et al., 1998).

Twin and adoption studies have been conducted to differentiate the influence of genes from that of environment in the development of eating disorders. See Chapter 4 for a full discussion of the basic design and logic of these studies.

Behavioral Genetic Studies

Results of population-based twin studies in Virginia, Minnesota, Australia, and Denmark indicate that genes play an important role in the development of eating disorders. These investigations found high rates of concordance for the presence of eating disorders in MZ twins compared with DZ twins and also reported high **heritability estimates** (percentages representing how much genes contribute to variation in a trait's presence in a given group of people) for the disorders in question. Heritability estimates have ranged up to 76% for AN (Klump, Miller, et al., 2001) and up to 83% for BN (Bulik, Sullivan, & Kendler, 1998); the lower limit of reported heritabilities is 50% across disorders (Baker et al., 2009; Bulik et al, 1998, 2006; Bulik, Sullivan, Wade, & Kendler, 2000; Javaras et al., 2008; Kendler et al., 1991; Klump, Kaye,& Strober, 2001). In a twin study, Bulik and colleagues (2010) found high genetic correlations between AN and BN. These results suggest that there is overlap in the genetic risk for these disorders and provide one potential explanation for diagnostic crossover (discussed in Chapter 11).

Questions have been raised about whether findings from twin studies can be taken at face value. Fichter and Noegel (1990) posited that being a member of a twin pair, particularly a MZ twin pair, might decrease the development of independence and consequently increase the risk of eating disorders. If this is true, then twins would not be representative of the general population (a violation of the **representativeness assumption**), and findings from twin studies would not accurately reflect how eating disorders develop in nontwins. However, studies using large, population-based twin samples have found that twins and nontwins were at equal risk for several types of psychopathology, including eating disorders, and have reported no differences in clinical presentation between twins and nontwins (Kendler, Martin, Heath, & Eaves, 1995; Klump, Kaye, & Strober, 2001).

As described in Chapter 4, an assumption of twin models is that members of twin pairs reared together, whether MZ or DZ, share 100% of their home environment (**equal-environments assumption**). Nevertheless, members of a MZ twin pair might be treated more similarly than members of a DZ twin pair, particularly in relation to comments about their appearance. A greater similarity in environment, rather than a higher percentage of shared genes, might explain why MZ twins are more similar with respect to eating disorders than are DZ twins. Supporting this concern, MZ twins show greater physical similarity (Hettema, Neale, & Kendler, 1995) and socialize more with their cotwins (Kendler & Gardner, 1998) than do DZ twins, and these factors are significant predictors of concordance for BN in twins (Hettema et al., 1995; Kendler & Gardner, 1998). However, Klump, Holly, Iacono, McGue, and Willson (2000) found that neither general physical similarity nor similarity of body size or shape was significantly associated with similarity in eating attitudes and behaviors between twins. Thus violations of the representativeness and equal-environments assumptions do not fully account for the greater similarity in eating disorders between MZ twins than between DZ twins.

As described in Chapter 4, to the extent that family environment is important in shaping risk for eating pathology, siblings should be more similar to each other than expected by chance, regardless of whether they are related biologically or by adoption. To the extent that genetic makeup is important in shaping risk for eating pathology, similarity will be greater for biological siblings than for adoptive siblings. Results from the only adoption study on eating disorders conducted so far offer further support for the greater influence of genetics than of shared family environment on eating disorders. Klump, Suisman, Burt, McGue, and Iacono (2009) studied similarity of disordered eating levels in 152 pairs of sisters, of whom 51 pairs were biological siblings and 101 were adoptive siblings. One concern for adoption studies is that being adopted might be a stressor that would increase risk for psychopathology. However, Klump and colleagues found that overall levels of disordered eating did not differ between biological and adopted children. Consistent with a significant effect of genes on eating disorder risk, disordered eating levels were significantly and positively correlated between biological sisters. In contrast, adoptive sisters were no more similar to each other with respect to disordered eating than they would be to a random woman. The estimated heritability of disordered eating in this study was 85%, with the remaining influences related to nonshared environmental factors (that is, nongenetic factors that differ between siblings reared together).

In attempting to understand what specific inherited factor or factors increase risk for eating disorders, researchers have examined several candidate traits. Family, twin, and adoption studies have yielded significant heritability estimates for BMI (Allison, Heshka, Neale, Lykken, & Heymsfield, 1994; de Castro, 1999; Koeppen-Schomerus, Wardle, & Plomin, 2001) and daily diet (de Castro, 1999). Using a twin design, Slof-Op't and colleagues (2008) examined the heritability of BMI and disordered eating behaviors. Both BMI and disordered eating behaviors demonstrated significant heritability, and shared genes explained 43–51% of the association between BMI and disordered eating behavior. However, only a minority of the genetic influence on disordered eating behaviors was shared with the genetic influence on BMI. These results suggest that one way families contribute to eating disorder

risk is by their genetic contributions to body weight but that most genetic influences on eating disorders are independent of the genes that influence body weight.

Studies have suggested a substantial genetic influence on personality traits, including those traits hypothesized to contribute to the risk of developing an eating disorder (see Chapter 7). Using a population-based twin sample, Klump, McGue, and Iacono (2002) found that associations between personality and disordered eating were best explained by shared genetic factors (as opposed to shared environment). However, only a limited proportion (2–22%) of the genetic influence on disordered eating was shared with the genetic influence on personality. Thus in addition to BMI, families may contribute to eating disorders by their genetic contributions to personality. Again, however, most genetic influences on disordered eating appear to be independent of those for personality.

In summary, twin and adoption studies have yielded impressive estimates of heritability for eating disorders. While these studies provide compelling evidence of a genetic contribution to eating disorders, they cannot reveal the influence or action of specific genes. With advances in mapping of the human **genome**, researchers have begun to examine specific genetic loci associated with increased risk of eating disorders.

Molecular Genetic Studies

A gene is a sequence of **deoxyribonucleic acid (DNA)** that is transcribed into a specific sequence of **ribonucleic acid (RNA)** that is used to build a chain of amino acids into a protein. For example, a gene could code for a neurotransmitter receptor, such as the 5-HT_{1A} receptor. An **allele** is one of several forms of the same gene. For some genes, there are only two possible alleles; other genes have numerous alleles. A Different forms of a gene's alleles are collectively referred to as polymorphisms. A DNA strand consists of a string of smaller units known as nucleotides, which are identified by the different bases in their structures: adenine (A), cytosine (C), guanine (G), or thymine (T). When alleles differ by a single nucleotide in the DNA sequence (such as the substitution of an A for a G nucleotide), this is referred to as single-nucleotide polymorphism (SNP). When alleles differ in terms of the repetition of nucleotides, this is referred to as copy-number variant polymorphism.

For a given gene, a child receives a single allele from each parent. The resulting combination of alleles is the individual's **genotype** for that gene. When the child receives the same allele from each parent, the genotype is **homozygous**. When the child receives different alleles from each parent, the genotype is **heterozygous**. The effects of different genotypes (i.e., different combinations of alleles for a given gene) can range from differences in eye color (see Figure 8.4) to whether or not one can curl one's tongue into a U shape (see Figure 8.5), to differences in the risk of developing an eating disorder. The observable manifestation of a genotype is referred to as a **phenotype.** Thus in the search for specific genes that increase the risk of an eating disorder, eating disorders are phenotypes.

Unlike results from family and twin studies, well-replicated findings have yet to emerge from studies examining specific genes that may increase the risk of developing an eating disorder. Indeed, the key conclusion that has come from genetic research is that rather than thinking of disorders as representing natural categories, we may be better served by understanding that many genetic variants could give rise to the same cluster of

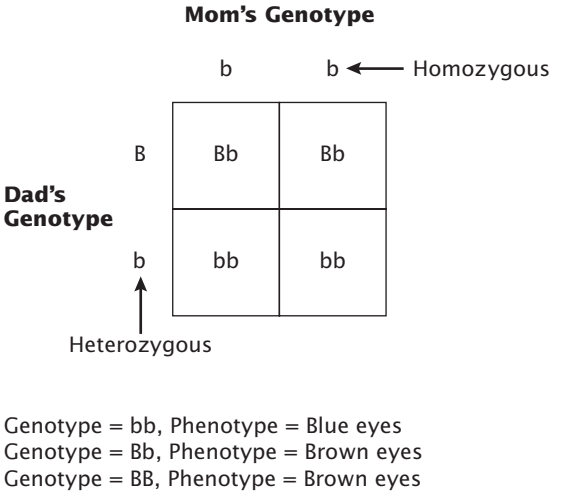


FIGURE 8.4 Punnett square for eye color. Four squares represent possible genotypes for children of a Mom with the bb genotype and Dad with the Bb genotype.

overlapping symptoms, so that our definitions of eating disorders result in groups with heterogeneous genotypes. Nonetheless, it is useful to review some studies that have led to this conclusion.

Most molecular genetic research has involved **association studies,** in which the frequencies of alleles for specific candidate genes (genes hypothesized to be involved in an eating disorder) are compared between individuals affected with a disorder and those unaffected. These studies examine the association between a specific genotype and the presence versus absence of eating disorders as a phenotype. For example, several **candidate gene studies** have examined allele frequencies for the gene that codes for the 5-HT_{2A} receptor. This gene has two possible SNP alleles, called A and G after the different bases present at one position in the gene. These alleles can be combined into three distinct genotypes: homozygous A/A and G/G and heterozygous A/G (which is functionally the same

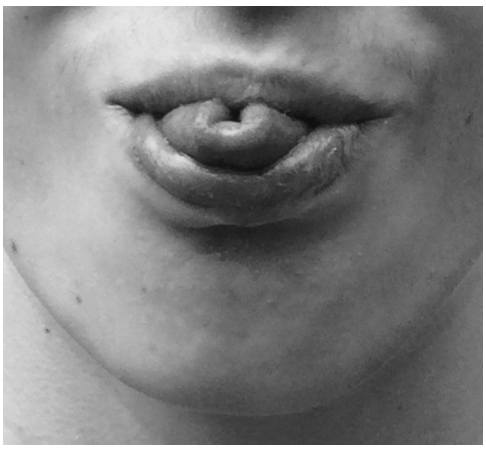


FIGURE 8.5 Ability to curl tongue into a U-shape is determined by a single dominant gene.

genotype as G/A). If individuals with an eating disorder are found to be more likely than controls to have a specific allele (e.g., A) or genotype (e.g., A/A), this supports an association between the structure of the 5-HT_{2A} receptor gene and the risk of developing that eating disorder.

In a **transmission disequilibrium test (TDT) study,** the frequency of allele transmission from parents who are selected to be heterozygous for the corresponding gene to affected offspring is compared with the frequency expected if there is no association between the allele and the disorder. For example, in a sample of parents who all have the heterozygous A/G genotype for the 5-HT_{2A} receptor gene, approximately 25% of children should have the homozygous A/A genotype, 25% should have the homozygous G/G genotype, and 50% should have the heterozygous A/G phenotype by chance (because 25% should receive A/G and 25% should receive G/A). If it is found that children with an eating disorder are more likely to have a specific allele (e.g., A) or genotype (e.g., A/A) than expected, this supports an association between the structure of the 5-HT_{2A} receptor gene and the risk of developing that eating disorder.

Both candidate gene studies and TDT studies rely on identifying a specific gene to examine. Genes that affect 5-HT function represent prime candidates for understanding the genetic etiology of eating disorders. Particular attention has been given to the gene for the 5-HT_{2A} receptor. Studies have shown that the A allele of the 5-HT_{2A} receptor gene reduced the density and sensitivity of that receptor (Parsons, D'Souza, Arranz, Kerwin, & Makoff, 2004; Myers, Airey, Manier, Shelton, & Sanders-Bush, 2007; A. K. Smith et al., 2008), consistent with findings for women who have recovered from AN and BN discussed earlier in this chapter. Thus the following section reviews findings for the 5-HT_{2A} receptor gene in studies of AN.

The 5-HT_{2A} Receptor Gene

Collier and colleagues (1997) reported that patients with AN had higher frequencies of the A allele and the A/A genotype of the 5-HT_{2A} receptor gene than controls. Several independent studies replicated this finding (see the top half of Table 8.1). However, an equal number of studies did not find an association between eating disorders and the 5-HT_{2A} receptor gene (see the bottom half of Table 8.1). Differences in eating disorder phenotypes may account for these inconsistent findings. For example, three studies (Nacmias et al., 1999; Ricca et al., 2002; Sorbi et al., 1998) have suggested that the A allele and A/A genotype for the 5-HT_{2A} gene are more common in ANR than in ANBP.

In a TDT study, Gorwood et al. (2002) examined transmission of the A and G alleles of the 5-HT_{2A} receptor gene from heterozygous (A/G genotype) parents of children with AN. The distribution of the A and G alleles did not differ significantly from that expected by chance. However, a follow-up investigation by Kipman et al. (2002) showed increased transmission of the A allele in a subset of patients who had an older age of onset. Kipman and colleagues suggested that the 5-HT_{2A} receptor gene may contribute to the etiology of AN in patients who develop the eating disorder in mid-adolescence or later. This finding is particularly interesting given evidence presented in Chapter 6 that disordered eating before

TABLE 8.1 Results of Candidate Gene Studies of 5-HT$_{2A}$ Receptor Gene

Study	Relative frequencies in groups with and without eating disorders
Significant association of 5-HT$_{2A}$ gene	
Collier et al. (1997)	AN > controls for A allele and A/A genotype
Enoch et al. (1998)	AN > BN, controls for A allele and A/A genotype
Sorbi et al. (1998)	ANR > AN purging only (ANP), controls for A allele and A/A genotype
Nacmias et al. (1999)	ANR > ANP, BN purging (BNP), controls for A allele and A/A genotype
Nishiguchi et al. (2001)	AN binging only (ANB), BN > ANR, controls for G allele
Kipman et al. (2002)	AN with later onset > controls for A allele
Ricca et al. (2002)[a]	AN, ANR, BN > controls for A allele and A/A genotype ANP, controls no difference Obese BED, obese non-BED, controls no difference
No significant association of 5-HT$_{2A}$ gene	
Hinney, Ziegler, Nothen, Remschmidt, & Hebebrand (1997)	AN, obese controls, underweight controls no difference
Campbell, Sundaramurthy, Markham, & Pieri (1998)	AN, controls no difference
Ziegler et al. (1999)	AN, BN, controls no difference
Ando et al. (2001)	AN, controls no difference
Karwautz et al. (2001)	AN, unaffected sisters no difference
Fuentes et al. (2004)	BN (with or without history of AN), controls no difference
Rybakowski et al. (2006)	AN > controls for A allele at trend level
Martásková, Slachtová, Kemlink, Záhoráková, & Papezová (2009)	AN > controls for A allele at trend level

[a]Includes participants in the study of Nacmias et al. (1999).

puberty is best explained by family environment, whereas disordered eating after puberty is best explained by genetic makeup.

The Brain-Derived Neurotrophic Factor Gene

Brain-derived neurotrophic factor (BDNF) regulates growth and is centrally involved in appetite regulation, with decreased BDNF function associated with increased food intake (Culbert, Slane, & Klump, 2008). Reduced BDNF levels have been found in AN and BN (Favaro, Monteleone, Santonastaso, & Maj, 2008). An allele of the BDNF gene, namely, the Met allele of the Val66Met polymorphism, reduces secretion of BDNF (Egan et al., 2003), and carrying the Met allele has been found to be significantly associated with both AN (Ribases et al., 2004; Rybakowski et al., 2007) and BN (Ribases et al., 2004). In addition, a meta-analysis (Gratacos et al., 2007) supported an association between carrying the Met allele of the BDNF gene and eating disorders broadly defined (including ANBP, BN, and disorders in the *DSM-IV* category "eating disorder not otherwise specified"). However, as with findings for the A allele of the 5-HT$_{2A}$ gene, not all studies

have supported an association between the Met allele and eating disorders (Gratacos et al., 2007).

Challenges in Identifying Genetic Contributions to Eating Disorder Risk

Behavioral genetic studies have clearly supported a genetic diathesis to eating disorders, but molecular genetic studies have yet to clearly identify specific genes that increase the risk. This likely reflects the early stage of molecular genetic research in this area as well as the etiologic complexity of the disorders. As with most psychiatric disorders, the genetic diathesis to eating disorders is likely to involve **complex inheritance** rather than **Mendelian inheritance** (Risch & Merikangas, 1993). Mendelian inheritance refers to the situation in which the phenotype is due to the action of a single gene (as in the case of eye color). Complex inheritance refers to the situation in which the combined action of many genes is responsible for the phenotype. All candidate gene studies examine the association between phenotypes and a single gene, as if the genetic risk for eating disorders were transmitted in Mendelian fashion.

Two epidemiological patterns suggest that it is unlikely that a single gene accounts for the genetic risk for eating disorders. First, eating disorders have higher prevalences than typical Mendelian diseases do. Alleles for diseases that are inherited in Mendelian fashion and decrease the ability to survive and have children (as eating disorders do) tend to be removed from the gene pool, resulting in low prevalences. Second, the reported risk ratios for eating disorders in first-degree relatives of eating-disordered probands (that is the likelihood that a person will develop a disease if they have a first-degree relative with the disease versus having no family history of the disease) generally range from 5:1 to 12:1 and fall far below what is expected for a Mendelian disease produced by a dominant gene (5,000:1) or a recessive gene (2,500:1). Risk ratios for Mendelian diseases are decreased when there is **reduced penetrance** (the genotype does not always lead to the phenotype), **variable expressivity** (the genotype leads to variable phenotypes), or **phenocopies** (the phenotype occurs in the absence of the genotype). For example, while a cleft chin is caused by a single, dominant gene, the phenotype associated with this genotype varies, ranging from a clear cleft (Figure 8.6) to smaller depressions. Even with these considerations, however, risk ratios for Mendelian diseases remain well above those reported for eating disorders.

In complex inheritance, each gene contributes a small amount to developing a disorder, and a large number of research participants is needed to show a reliable effect of a specific gene. Thus some inconsistencies in research findings may be caused by inadequate sample sizes as well as not looking at all relevant genes. Given this, an alternative way of examining molecular genetic contributions to eating disorders is to take a completely agnostic approach to what genes may be important by examining all genes. With advances in technology, mapping DNA has become faster and less expensive, introducing the option of **genome-wide association studies** (GWAS). In a GWAS, researchers evaluate whether or not alleles for

FIGURE 8.6 A cleft chin is caused by a single dominant gene, but phenotypic expression of this genotype varies.

all genes in the genome are more common in individuals with an eating disorder than in controls. Because looking at so many possible differences creates a high risk of false positive results, the threshold for statistical significance in a GWAS is set at a very stringent level, and these studies require huge samples to detect significant effects.

The first GWAS in AN failed to find a statistically significant difference in allele frequencies between 1,033 AN cases and 3,733 control subjects but did find trend-level associations between polymorphisms for an opioid receptor and for the 5-HT_{1D} receptor (Wang et al., 2011). Similarly, Wade et al. (2013) found no significant associations between particular genes and either disordered eating behaviors or syndromes grouped as "AN spectrum," "BN spectrum," and "purging via substances," but they did find trend-level results for six genes (though none of them related to prior findings in the field). Neither study found the A polymorphism of the 5-HT_{2A} receptor gene to be linked to AN.

A final challenge in the search for genetic loci associated with eating disorders is difficulty defining eating disorder phenotypes. Unlike eye color, where it is reasonably easy to conclude that brown and blue are different colors (and different phenotypes), it remains unclear, for example, whether ANR and ANBP represent one or two disorders. Thus more research on the definition of eating disorders is necessary to identify valid phenotypes. Indeed, this point applies to the full range of studies seeking to understand the pathophysiology of mental disorders and represents the key premise behind the formation of the RDoC initiative, discussed in Chapter 1. Research to better define eating disorders according to core behavioral dimensions rather than complex collections of symptoms will allow more efficient progress toward establishing the genetic and neurobiological underpinnings of eating disorders.

Given the challenges facing molecular genetic research, the findings suggesting an association between the 5-HT_{2A} receptor gene and AN are quite promising. It is also important to acknowledge an advantage that molecular genetic research has in the search for the biological bases of eating disorders. Unlike other potential biological contributors, gene sequences do not alter as a function of the presence or absence of an eating disorder. Thus it is possible to examine candidate genes among individuals with eating disorders without

worrying that an association between a specific genotype and the presence of an eating disorder reflects the effect of the disorder on the genome. In the next section we turn to the profound effects that eating disorders can have on the body.

Physical Consequences of Eating Disorders

For many studies of biological differences between individuals with eating disorders and healthy individuals, it is not possible to determine whether the difference predated the presence of the eating disorder or is a consequence of the eating disorder. When the body has inadequate resources to maintain body weight and must revert to using its fat stores, other physical changes necessarily follow. This section discusses known physical consequences of eating disorders. As discussed below, some consequences may contribute to eating disorder maintenance. As a preview of this section, Table 8.2 shows the medical complications frequently associated with eating disorders organized by whether they are attributed consequences of starvation, binge eating, or purging.

Consequences of Starvation

Neuroanatomical Changes

Patients with AN have larger cerebral ventricles than healthy controls do (D. K. Katzman, Zipursky, Lambe, & Mikulis, 1997; Kingston, Szmukler, Andrewes, Tress, & Desmond, 1996; Krieg et al., 1989; Roberto et al., 2011; Swayze et al., 1996). Ventricles contain cerebrospinal fluid, and increases in ventricle size reflect decreases in brain mass (loss of gray and white matter). Thus this finding indicates that in addition to causing breakdown of fat, muscle, and bone (see Table 8.1), starvation triggers the breakdown of brain matter. Some of the lost brain matter is regained with recovery (Kingston et al., 1996; Roberto et al., 2011; Swayze et al., 1996). However, evidence of diminished gray matter persists after recovery from AN (Katzman et al., 1997). McCormick et al. (2008) found that the volume of the anterior cingulate cortex was reduced in patients with AN compared with controls, although it increased significantly with inpatient treatment and weight recovery. Smaller increases in anterior cingulate cortex volume prospectively predicted relapse after hospital discharge (McCormick et al., 2008).

Bone Mineral Density Changes

Several studies have demonstrated decreased bone mineral density and increased rates of osteoporosis in patients with AN compared with controls (Grinspoon et al., 1999; Hotta, Shibasaki, Sato, & Demura, 1998; D. K. Katzman & Misra, 2013). A study of women with BN also found reduced bone mineral density; however, differences were attributable entirely to a history of AN in a subset of patients (Naessén, Carlström, Glant, Jacobsson, & Hirschberg, 2006). Bone mineral loss has been linked to a combination of starvation and hormonal changes that result from starvation—notably decreased concentrations of sex hormones such as estrogen and progesterone and increased concentrations of cortisol (Newman & Halmi, 1989). In addition, the diets of patients with AN are often

TABLE 8.2 Medical Complications of Eating Disorders (by Major Organ System)

System[a]			Medical complication	Definition
CARDIOVASCULAR				
A	B	C	Orthostatis/hypotension	Low blood pressure
A			Bradycardia	Slow heartbeat
A		C	Congestive heart failure	Loss of heart function
A	B	C	EKG abnormalities	Abnormal heartbeat patterns
A			Refeeding cardiomyopathy	Loss of heart tissue
A			Refeeding edema	Swelling/water retention
		C	Arrhythmias	Irregular heartbeat
A	B	C	Sudden cardiac death	Sudden heart attack leading to death
RENAL/FLUID BALANCE				
A	B	C	Dehydration	Decreased body fluid
A	B	C	Decreased glomerular filtration rate	Decrease in kidneys' ability to filter and remove waste products from blood
A	B	C	Abnormal electrolytes	Abnormal concentrations of electrically charged substances in blood
A	B	C	Kaliopenic nephropathy	Insufficient potassium in the body due to kidney function
A			Refeeding edema	Swelling/water retention
		C	Reflex edema (after cessation of laxatives/diuretics)	Swelling/water retention
ENDOCRINE				
A			Growth retardation	Delayed maturation
A			Delayed-onset puberty	Delayed menarche
A	B		Abnormal HPG-axis function	Disrupted sex hormones
A			Abnormal HPA-axis function	Disrupted stress hormones
A			Abnormal thyroid function	Disrupted mood and appetite hormones
GASTROINTESTINAL				
	B		Parotid gland swelling	Swelling of salivary glands
	B	C	Hyperamylasemia	Elevated blood concentrations of salivary gland enzyme that helps digest glycogen and starch
	B		Esophageal tear	Tear in the esophagus
	B		Mallory-Weiss tears	Tears in the esophagus's mucus between esophagus and stomach
A	B	C	Delayed gastric emptying	Slowed digestion of food in stomach
A			Superior mesentery artery syndrome	Partial obstruction of the intestine by the superior mesenteric artery
	B		Gastric dilation/rupture	Enlargement of and tear in stomach
A			Refeeding pancreatitis	Infection/irritation of pancreas
A	B	C	Constipation	Inability to pass solid waste
		C	Melanosis coli	Dark discoloration of colon's mucus after longstanding use of certain laxatives
		C	Steatorrhea/protein-losing gastroenteropathy	Fecal matter that is frothy, is foul smelling, and floats because of high fat content
		C	Cathartic colon	Anatomical and physiological change in colon linked to stimulant laxatives
A	B	C	Hypokalemic ileus	Temporary immobility of intestine, preventing passage of solid waste, caused by low blood potassium levels

TABLE 8.2 Continued

System[a]			Medical complication	Definition
METABOLIC				
A			Osteoporosis/osteopenia	Loss of bone mass
A	B	C	Trace mineral deficiencies	Decreased minerals
A	B	C	Vitamin deficiencies	Decreased vitamins
A			Hypercholesterolemia	Increased blood cholesterol
	B	C	Obesity	Overweight
PULMONARY				
	B		Subcutaneous emphysema	Presence of air beneath skin
	B		Pneumomediastinum	Rupture of alveoli of lungs
	B		Aspiration pneumonitis	Lung infection/irritation caused by inhaling vomit
DERMATOLOGICAL				
A			Hair loss	Balding
A			Lanugo-like hair growth	Fine, downy hair covering body
A			Dry skin, brittle hair, nails	Caused by lack of fat in diet
A			Petechia, purpura	Discolorations in the skin caused by small bleeding vessels near skin surface
	B		Finger calluses/abrasions	Caused by friction against teeth while gagging
HEMATOLOGICAL/IMMUNOLOGICAL				
A			Anemia	Decreased iron in blood
A			Leukopenia with relative lymphocytosis	Decreased white blood cells with unusually high number of normal lymphocytes
A			Thrombocytopenia	Decreased platelets in the blood, resulting in decreased clotting/increased bleeding
A			Abnormal CD4/CD8 counts	Reduced white blood cell counts reducing immune function
A			Abnormal cytokines	Disrupted cell–cell communication; thought to act directly on hypothalamic neurons
NEUROLOGICAL				
A			Enlarged ventricles	Loss of gray matter in the brain
A	B		Sleep disorders	Disrupted sleep patterns
A	B		Abnormal PET scans	Disrupted brain use of glucose
OTHER				
	B		Dental complications	Increased cavities and gum infections
A			Impaired thermoregulation	Low body temperature
	B	C	Vitamin K–deficient coagulopathy	Defect in blood clotting
		C	Skeletal muscle myopathy	Loss of muscle

Note. Adapted with permission from "Medical Evaluation and Medical Management," by C. Pomeroy, in *The Outpatient Treatment of Eating Disorders: A Guide for Therapists, Dietitians, and Physicians* (Table 13.2, pp. 306–318), edited by J. E. Mitchell, 2001, Minneapolis: University of Minnesota Press. Copyright 2001 by University of Minnesota Press.

[a] A indicates that complication arises from starvation of AN; B, that complication arises from binge/purge symptoms of BN and ANBP; C, that complication arises from other symptoms of eating disorders.

nutritionally unbalanced, with overconsumption of caffeine and underconsumption of calcium-rich foods (Newman & Halmi, 1989). Many patients with eating disorders learn about the effects of starvation on bone mineral density through research participation. Emily is an example.

Case Study: Emily

As part of Emily's participation in the research study (see Chapter 4), the results of her bone scan were given to her doctor, who shared them with Emily. The scan indicated that Emily's bone mineral density was 35% lower than expected for her age. When Emily asked what this meant, the doctor explained that because of Emily's diet and weight loss, her body had begun to feed on itself. This was the process that produced weight loss. However, the weight loss was not restricted to a loss of fat. It included a loss of muscle and bone. A consequence of decreased bone mineral density was an increased risk of bone fractures. Essentially, Emily at 20 years of age had the bone structure of a woman past menopause. Emily's doctor prescribed calcium supplements to help her regain bone tissue. However, she was told that the best way to reverse this damage was to gain weight.

Consequences of Binge Eating

Binge eating may contribute to enlarged gastric capacity, which has been linked to delayed gastric emptying (Klein & Walsh, 2004). Because the peptides that regulate satiation are released after food passes from the stomach to the intestine, delays in gastric emptying could contribute to the blunted neuropeptide responses to food intake observed in BN. Binge eating has also been linked to elevated cortisol release (N. A. Lester, Keel, & Lipson, 2003), which may affect the stress response of the HPA axis by impacting the negative feedback loop that regulates stress response. Elevated cortisol also may increase risk for weight gain, particularly around the abdomen, a location where excess weight has been linked to poor cardiac health. Finally, binge eating increases risk for weight gain and obesity (Tanofsky-Kraff et al., 2007).

Consequences of Purging

Purging by self-induced vomiting and severe laxative and diuretic abuse has been associated with electrolyte imbalances in the bloodstream (Mitchell, Pyle, Eckert, Hatsukami, & Lentz, 1983; Mitchell, Seim, Colon, & Pomeroy, 1987). Specifically, the loss of stomach acid in vomit decreases the presence of positively charged ions such as potassium (K^+). In addition, patients with eating disorders can have decreased blood concentrations of magnesium (Mg^+) and calcium (Ca^+). Electrolyte imbalances can contribute to fluid retention **(edema)** when patients attempt to reduce their disordered eating (Mitchell, Pomeroy, Seppala, & Huber, 1988).

Self-induced vomiting can also contribute to significant dental problems, because the stomach acid in vomit erodes tooth enamel. This erosion leaves teeth vulnerable to plaque and bacteria (Milosevic, 1999; Simmons, Grayden, & Mitchell, 1986). Although not life-threatening, this effect can result in tooth loss and the need for dentures at a young age.

Tears in the esophagus are a rare but serious consequence of using fingers or instruments to induce vomiting by producing a gag reflex (Mitchell, 1990). Use of fingers to induce vomiting can also cause calluses to develop on the hand (known as *Russell's sign*). Self-induced vomiting also can lead to swelling of the salivary (parotid) glands. Because these glands are near the face, patients sometimes misinterpret this side effect as a "fat face" and increase the frequency of their self-induced vomiting. This consequence of purging might have increased Jean's dislike of her face, described in Chapter 6.

Case Study: Jean

Jean was startled when a dental hygienist asked if she had ever had an eating disorder. Jean lied and said that she had not. Later, she asked the hygienist the reason for the question. The hygienist explained that Jean had some tooth enamel erosion that was consistent with purging. In addition, she commented that self-induced vomiting was associated with swelling of the salivary glands, and she had noticed that Jean's cheeks appeared a little swollen. Jean said that she had always had round, "chipmunk" cheeks. However, later that day, Jean looked at pictures of herself in her photo album. She realized that her face did look more round than it had when she wasn't purging.

Mortality

Finally, eating disorders can lead to death. Mortality as an outcome will be reviewed in more detail in Chapter 11. Anorexia nervosa can lead to death by starvation. At a certain point, the body cannot function without fuel, and multiple organ failure occurs. The body also can be substantially weakened by prolonged starvation, and death can occur as a consequence of an infection that would not normally be life-threatening. For example, pneumonia is a rare cause of death in healthy young women. However, pneumonia becomes a more significant threat to life for a young woman physically weakened by AN. Consequences of specific symptoms can interfere with organ function on a cellular level. For example, low potassium concentrations (known as *hypokalemia*) can contribute to heart failure and kidney failure, particularly in individuals whose organs have been weakened by a sustained state of starvation.

Conclusion

The contribution of biology to the etiology of eating disorders is indisputable. Studies of 5-HT and gut neuropeptide function indicate several abnormalities in individuals with eating disorders. Many of these abnormalities, however, normalize upon recovery from the disorder. Clear evidence supports the role of genes in increasing the risk of developing an eating disorder. Mirroring results from physiological studies, preliminary results suggest that genes involved in 5-HT function may increase the risk of eating disorders.

Many of the findings concerning biological function in patients with eating disorders likely represent correlates or consequences of the disorder in question. To the extent that biological indices are correlated directly with the severity of weight loss or binge frequency,

they may reflect "state" rather than "trait" qualities—that is, aspects of the individual's current condition rather than permanent parts of his or her biological makeup.

The influence of eating disorders on biological function has long been appreciated. It is difficult to dramatically alter food intake without causing significant alterations in biological function. The biological consequences of eating disorders constitute an important area of inquiry, because understanding them may help us improve the treatment and long-term outcomes of these disorders. Some biological consequences of eating disorders may become maintenance factors, capturing patients in a vicious cycle. For example, binge episodes in BN may cause increased gastric capacity, which reduces gastric emptying, which weakens the response of gut neuropeptides that regulate hunger and satiation so that individuals need larger quantities of food to feel satisfied. Thus even though gut neuropeptide response may be a consequence of disordered eating rather than an initial cause, it contributes to maintaining the illness. Identifying such maintenance factors is important for effective treatment, because treatment must reduce maintenance factors to succeed in producing remission.

Key Terms

Allele
Arcuate nucleus
Association studies
Brain-derived neurotrophic factor
Candidate gene study
Cholecystokinin
Complex inheritance
Deoxyribonucleic acid (DNA)
Dopamine
Downregulation
Edema
Equal-environments assumption
Functional magnetic resonance imaging (fMRI)
Genome
Genome-wide association study
Genotype
Ghrelin
Glucagon-like peptide 1 (GLP-1)
Heritability estimate
Heterozygous
Homozygous
Hypothalamic–pituitary–adrenal (HPA) axis
Hypothalamic–pituitary–gonadal (HPG) axis
Hypothalamus
Leptin

Mendelian inheritance
Neuron
Neuropeptide Y
Neuropeptides
Neurotransmitters
Norepinephrine
Nucleus accumbens
Paraventricular nucleus
Peptide YY (PYY)
Phenocopy
Phenotype
Positron emission tomography (PET)
Proband
Pyloric sphincter
Receptor
Reduced penetrance
Representativeness assumption
Reuptake
Ribonucleic acid (RNA)
Serotonin (5-HT)
Transmission disequilibrium test (TDT) study
Tryptophan
Upregulation
Vagus nerve
Variable expressivity
Ventral tegmental area

9

Treatment

This chapter covers interventions designed to remedy eating disorders. It first provides an overview of the amount (duration and frequency), types, and costs of treatments received by patients with eating disorders. Following this discussion of treatment use, the rationales for and techniques associated with different methods of intervention are presented, along with evidence of their degrees of success.

Treatment Use

As reviewed in Chapter 8, AN is associated with serious medical complications. Thus many individuals with the disorder require medical as well as psychological treatment. This necessity increases both the amount and the cost of treatment associated with AN. In the United States, Striegel-Moore, Leslie, Petrill, Garvin, and Rosenheck (2000) reported that female patients with AN averaged 26 days of inpatient care and 17 days of outpatient care a year. Costs associated with this care were $19,728/year per female patient. In contrast, male patients with AN averaged 16 days of inpatient care and 9 days of outpatient care a year.

Although most women who suffer from BN may never seek treatment (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Fairburn, Welch, Norman, O'Connor, & Doll, 1996), treatment use is high among those who do. In the United States, Striegel-Moore et al. (2000) reported that female patients with BN averaged 15 days of inpatient care and 16 days of outpatient care a year. Costs associated with this care were $10,970/year per female patient. Male patients averaged 22 days of inpatient care and 9 days of outpatient care a year.

The costs associated with treating patients with eating disorders are very high. In a review of healthcare costs across different nations, Simon, Schmidt, and Pilling (2005) found that inpatient care for AN cost 3.5 million pounds/year in the United Kingdom, 4.6 million euros/year in Denmark, and 59.1 million euros/year in Germany. Across all eating disorder patients, Denmark spent 6.4 million euros and Australia spent 14 million Australian dollars on inpatient care a year. In Germany the full range of healthcare costs, including primary care, outpatient treatment, and rehabilitation, totaled 65 million euros/year for AN and 10 million euros/year for BN.

As is evident from the figures above, women with AN spent significantly more days and money in inpatient treatment than women with BN in the United States (Striegel-Moore et al., 2000). This likely reflects the greater need for medical care in AN. However, no significant differences in days or cost of outpatient treatment have been found between women with AN and women with BN (Keel, Dorer, et al., 2002; Keel & Herzog, 2004). Amount of treatment with antidepressant or anxiolytic medication also did not differ between AN and BN (Keel, Dorer, et al., 2002). With regard to comparisons between male and female patients, amount of inpatient care did not differ in Striegel-Moore et al.'s (2000) study, but women received significantly more outpatient care than did men. This difference may reflect reluctance among male sufferers to seek treatment for a disorder strongly associated with women. It may also reflect reduced recognition of eating disorders in individuals who do not match the stereotype for an eating disorder (see Chapter 3).

Other predictors of increased treatment use for eating disorders included greater symptom severity, worse psychosocial function, and the presence of personality disorders or mood disorders (Keel, Dorer, et al., 2002). Thus more severe problems lead to increased treatment use. This relationship may reflect the role of personal suffering in motivating the search for treatment. It also may reflect the extent to which individuals with more severe disorders take longer to recover and therefore need continued treatment.

Fewer data are available for treatment use in BED than in AN or BN. Fichter, Quadflieg, and Gnutzmann (1998) reported that 66% of patients with BED in Germany received additional inpatient treatment following discharge from an inpatient program, reflecting similar treatment use for BED as for AN and BN in that country. In contrast, Fairburn and colleagues (2000) reported that in England as few as 8% of individuals with BED ever received any treatment for an eating disorder, compared with 40% of individuals with BN. In an examination of costs charged through a health maintenance organization in the United States, Dickerson et al. (2011) found that less than 25% of individuals identified with BED had received any healthcare services related to eating or weight management, with an average cost of $72/year per patient. Given the prevalence of BED among individuals seeking weight loss, it seems likely that many individuals with the disorder seek treatment for their weight problems rather than psychological treatment for their eating behaviors.

Studies of treatment use do not provide a direct measure of treatment needs. In a study of adolescents in the United States, Merikangas et al. (2011) estimated that only one in five individuals who had suffered from an eating disorder ever used mental health services. In Striegel-Moore et al.'s (2000) study, only 0.06% of female patients in the National Insurance Claims database were treated for eating disorders. This rate is much lower than the 12-month prevalences of AN and BN (see Chapter 1), suggesting that the majority of women with eating disorders receive no treatment.

Individuals may not receive the treatment they need because of lack of recognition of the disorder, lack of money, lack of access to treatment, lack of interest, or any combination of these factors. Lack of recognition that an eating disorder is present is a major barrier to treatment. For example, while recruiting women with recurrent binge eating or BED for a self-help program, Dickerson et al. (2011) found that only 4% of the women had been identified in their health insurance records as having an eating disorder. In addition, even when an

eating disorder is identified, patients do not receive coverage for the level of care they need (Klump, Bulik, Kaye, Treasure, & Tyson, 2009). Lack of insurance coverage has been linked to the misconception that eating disorders are life choices rather than diseases with a genetic basis like schizophrenia or bipolar disorder (Klump et al., 2009).

Review of treatment use data thus reveals that most individuals with eating disorders never receive any form of treatment. To some extent, this deficiency reflects the imbalance between the large number of individuals who suffer from eating disorders and the small number of clinicians available to help them (Levine & Smolak, 2001). That imbalance has led many researchers to argue for the importance of preventing eating disorders (Austin, 2000; Levine & Smolak, 2001), the topic of the next chapter. For patients with access to treatment, however, a wide range of options may be available. The following section discusses different types of treatment that are used for eating disorders.

Treatment Modalities

Inpatient Treatment

Inpatient treatment is the most intensive and expensive treatment for eating disorders. The primary rationale for inpatient treatment is to achieve medical safety in a controlled environment. For individuals who seem unable to interrupt their disordered eating behaviors despite imminent health risks, inpatient treatment provides a safe environment where they will be prevented from hurting themselves. Most often, inpatient treatment is used for individuals with AN who are dangerously underweight (see Figure 9.1) because of the severe medical consequences of starvation (see Chapter 8). Many patients, however, consider inpatient treatment the least desirable form of treatment and resist entering it. This was true of Emily.

Case Study: Emily

In addition to seeing a doctor to monitor her medical condition, Emily began seeing a psychologist and a nutritionist. However, she continued to lose weight over the first four weeks of treatment. In therapy, Emily admitted that she was afraid of becoming fat as a result of treatment and that she had been increasing her weight loss efforts to protect against this potential outcome. Emily's weight was now critically low, and her treatment team feared that she might go into cardiac arrest. They informed her that she needed inpatient treatment. Emily pleaded that that she had not been "truly trying" and that she would be able to gain weight on her own. However, her treatment team informed her that doing so would be unsafe because the process of regaining weight could be dangerous once her weight dropped below a certain point. Reluctantly, Emily agreed to enter an inpatient treatment program for eating disorders. She hated the program, hated the loss of autonomy, hated being woken at 5:30 a.m. every morning to have her weight and vital signs checked, hated the nursing staff, hated the other patients, hated being monitored all the time, hated being forced to eat, and hated being forced to take part in "stupid groups." However, Emily slowly gained weight. As this happened, she found that it was easier to read and concentrate on conversations and that some of the other patients and nursing staff were actually "interesting." As she neared her target weight for discharge, Emily felt a combination of eagerness to be free from the

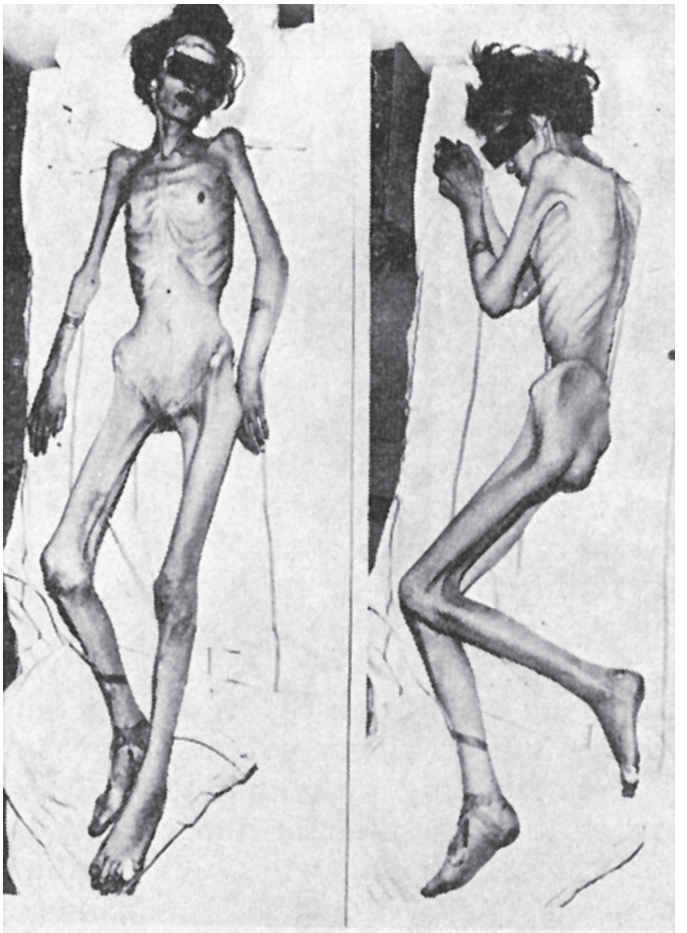


FIGURE 9.1 Patient hospitalized for anorexia nervosa in attempt to prevent her death. Source: Valanne, E. H., Taipale, V., Larkio-Miettinen, A. K., Moren, R., and Ankee, M. (1972). *Anorexia Nervosa: A follow-up study*. Psychiatria Fennica, 267.

antiseptic environment of the inpatient unit and fear of leaving what had become a safe place where it didn't matter whether she wanted to eat or not because it was not her decision.

Inpatient treatment also is employed for patients who purge very frequently, because of the dangerous electrolyte imbalances that accompany this symptom. Finally, inpatient treatment is used both to prevent suicide attempts and after suicide attempts. Depending on the severity of the attempt, inpatient treatment may be used to achieve medical stability in addition to ensuring safety from additional attempts.

Case Study: Valerie

Valerie had been working with her therapist for approximately six months and had also been on antidepressant medications. She felt better during her therapy sessions, but the feelings generally didn't last for long after she left a session. She felt bad returning week after week without having reduced her purging frequency. Her therapist had discussed healthier

methods for weight management with her, including exercising. But Valerie found it difficult to fit that into her work schedule. They also discussed other things Valerie could do when she felt the urge to vomit, such as calling and talking to a friend or taking a hot bath—but when Valerie wanted to purge, she felt so awful about herself that she couldn't imagine anyone wanting to talk to her and didn't feel like she deserved any kind of pampering. More and more, Valerie was expressing feelings of hopelessness about ever getting better, and she began apologizing to her therapist for wasting her time. Valerie's therapist reassured her that they were not wasting time but did urge her to try things they discussed to get the most out of treatment. One day, Valerie missed her appointment. This was very unusual, because Valerie was always early and attended appointments even when she was feeling under the weather. The therapist called Valerie's home and left a message expressing concern that Valerie hadn't come to her appointment and had not called. Later that day, Valerie's sister returned the message and explained that Valerie had been hospitalized after attempting suicide by taking an overdose of her antidepressant medication and had not yet regained consciousness. The therapist thanked the sister and called the hospital, identifying herself and requesting that the staff let Valerie know that she had called. The staff answered that they would pass on the message but that Valerie would have restricted phone access for a while. Valerie ended up staying in the hospital for two weeks after that attempt. She spent the first 24 hours in a medical unit, because she had experienced tachycardia (fast or irregular heart rate) and was unresponsive to her name after the overdose. After those first 24 hours, she was moved to a psychiatric unit for three days. Valerie was then transferred to an inpatient eating disorders program once the staff concluded that her desperation was related to her inability to control her purging and that the effects of the overdose had been exacerbated by complications due to her extreme purging behavior.

To achieve a controlled environment, inpatient eating disorder treatment programs use several measures. These include medical monitoring of pulse, blood pressure, body temperature, and blood electrolyte levels as well as daily weight checks to determine whether underweight individuals are gaining weight. As in Emily's case, weight gain toward a specific goal is often a condition of discharge. Another shared feature of inpatient programs is monitored meals. These can occur in groups or, for patients who have been put on bed rest, with a nurse present in the room. Patients may also be monitored during their use of the bathroom to prevent purging; this was done with Valerie. In addition to monitoring eating, activity level, and opportunities to purge, inpatient treatment programs often include group sessions for patients to discuss feelings about eating, their bodies, gaining weight, and recovery. Patients may be asked to keep a journal to help them understand their feelings and to facilitate communication of those feelings with others. Group sessions can make use of any of the methods described below in the section titled "Psychotherapy Content and Theoretical Orientations."

Inpatient treatment programs are generally successful in restoring weight for patients with AN and in eliminating binge eating and purging for patients with those symptoms. However, patients vary considerably in their ability to maintain the gains made during inpatient treatment once they are discharged. Some of this variation appears to be attributable to

the nature of the patient populations. It is difficult to assess whether the outcomes reported for hospitalized patients are any better or worse than what would have occurred had they not received inpatient care. Given the medical emergency represented by most hospitalized cases, it would be unethical to do an experiment in which some patients are randomly assigned to inpatient care and others to a wait-list control. At the very least, inpatient care delays death for some patients; for many, it may save their lives and give them the opportunity to achieve eventual recovery.

Because inpatient treatment is employed for patients who are medically unstable, an added benefit of such treatment is the structured environment it provides. As described in Emily's case, this environment relieves many patients of their internal battles about eating by taking the decision to eat or purge away from them. However, patients are usually discharged once they reach medical stability regardless of whether or not they have achieved full recovery or would continue to benefit from remaining in a structured environment. Residential treatment programs offer the benefits of a structured environment to patients who are medically stable.

Residential Treatment Programs

The Renfrew Center of Philadelphia was the first residential treatment clinic for eating disorders in the United States. The program advertises "a safe, non-institutional, nurturing environment" that provides therapy from trained clinicians who specialize in the treatment of eating disorders. Since opening in June 1985 the Renfrew Center has expanded and now includes a second residential center in Florida as well as outpatient clinics in other locations. The documentary *Thin* (2006) presents the courses of treatment offered to four patients through the Renfrew Center, giving insight into the struggles these women face in attempting to overcome their eating disorders, the severity of their illnesses, and the approaches used by the Renfrew program to help them. Renfrew also hosts conferences in which nationally known researchers and clinicians provide continued education and clinical training to health professionals working with eating-disordered patients. In addition to the Renfrew Center, several other residential treatment programs have emerged across the United States over the past three decades. These centers treat many of the most severe and chronic cases of eating disorders.

Residential treatment programs can be private, nonprofit institutions or commercial, for-profit enterprises. They are very expensive, and access to the care they offer is limited by what insurance carriers are willing to pay or by patients' personal ability to pay for treatment. Because residential treatment programs provide healthcare 24 hours a day, their costs are similar to those of inpatient treatment. Frisch, Herzog, and Franko (2006) estimated the cost for one day of inpatient treatment to be $1,000 in the United States. For residential treatment the average cost per day was $956 and the mean duration of treatment was 83 days, resulting in an average cost of $79,348 for patients in such programs.

Another feature common to inpatient and residential treatment programs is the limited ability to determine the success of treatment compared with no treatment or alternative treatments. Residential treatment centers are designed to provide a service. Thus they do not randomly assign patients to different treatment conditions. Instead they attempt to ensure

that each patient receives whatever form of treatment is deemed necessary by the treatment program staff.

Outpatient Treatment

Outpatient care is less expensive than inpatient or residential care, because outpatients do not require room and board. Outpatient treatment can occur at different levels of intensity and frequency, including day programs (also referred to as *partial hospital programs*), evening programs, intensive group therapy, and intensive individual therapy, with weekly, biweekly, or even monthly sessions.

If inpatient and residential treatment involve spending 24 hours a day in treatment, then day programs involve spending 40 hours a week in treatment. In day treatment programs, patients attend treatment in a manner that is similar to going to work. They receive a level of therapeutic intervention similar to that offered by inpatient programs as well as a controlled environment for eating two or three meals plus snacks during the day. They do not, however, receive the same level of medical monitoring, and they spend their evenings and nights at home. Evening programs allow patients to work or care for children during the day and then enter a controlled environment for their evening meal. For some individuals, their jobs provide adequate control over behaviors such as binge eating and purging, and an evening treatment program helps to sustain that structure. For many patients, entering a day or evening program represents a transition from inpatient treatment to further support for maintenance of treatment gains, particularly weight gain in AN.

Intensive group and individual therapy occurs several times per week, and sessions may last for up to two hours. Thus they fill neither the entire day nor the entire evening. Weekly, biweekly, and monthly outpatient sessions are appropriate for patients who have achieved medical stability and who are able to modify their behaviors in response to therapeutic interventions. A traditional schedule for outpatient therapy would be one 50-minute session a week. Regardless of treatment duration or frequency, the content of sessions depends on the theoretical orientation and goals of the treatment.

Levels of Care and Treatment Outcome

Specialized eating disorder programs are unavailable in many geographic areas, and the financial burden associated with specialized, multidisciplinary treatment is significant. Thus many patients and health professionals face a dilemma in trying to identify the best treatment given the needs and resources that are available.

Gowers and colleagues (2010) conducted a randomized controlled trial (see discussion below) in 167 adolescent patients diagnosed with AN comparing outcomes of treatment in a specialized inpatient program, a specialized outpatient program, and routine (nonspecialized) treatment through Child and Adolescent Mental Health Services in England. No patient who was medically unstable was treated as an outpatient, but medically stable patients were assigned at random to treatment such that the selection of inpatient care did not necessarily reflect a medical need. Patients were significantly less likely to accept randomization to inpatient treatment (49% accepted this option) than to accept specialist outpatient (77%) or general outpatient (71%) care. Among patients who engaged in their assigned treatment,

satisfaction was greater for both inpatient and outpatient specialist treatment than for routine outpatient treatment. Overall, there was a positive but slow response to treatment, with 19% of patients having a good outcome at 1 year, 33% having a good outcome at 2 years, and 64% having a good outcome at 5 years. There were no differences in remission across the three treatment conditions. With respect to **cost effectiveness**, specialist outpatient treatment was less expensive than general outpatient treatment, which was less expensive than inpatient treatment. One reason for the cost difference between specialist and general outpatient care was that future hospitalizations were less likely for patients receiving treatment from clinicians with expertise in eating disorders.

Overall, the results of the Gowers et al. (2010) study support the conclusion that specialist outpatient treatment was superior because it was most acceptable to patients, resulted in a high level of satisfaction, produced outcomes that were comparable to those of alternative approaches, and cost less than alternative approaches.

Psychotherapy Content and Theoretical Orientations

This section reviews the logic and content of the different approaches to treating eating disorders that a patient may encounter. Different theoretical orientations emphasize different treatment goals and methods for reaching those goals. These orientations are not specific to eating disorders, nor are they related to any particular treatment modality discussed above. Rather, they reflect ways of understanding mental disorders in general and have been applied to the treatment of a variety of problems. This section also reviews the demonstrated success (or lack thereof) of treatments from each theoretical orientation. The success of a treatment in producing desired changes is referred to as the treatment's *efficacy*.

Psychoeducation

When patients first seek treatment for an eating disorder, they often have several questions about the disorder. Thus **psychoeducation** is included in most forms of intervention and shapes the content of psychotherapy. However, psychoeducation alone is not often thought of as a distinct psychotherapy, and it is not viewed as an adequate intervention (Fairburn, Kirk, O'Connor, & Cooper, 1986).

The basic rationale behind psychoeducation as an aspect of treatment is that for patients to recover from eating disorders, they must understand what the disorders are, what factors are thought to contribute to or exacerbate problem eating, what approaches are thought to decrease problem eating, and the consequences of continued disordered eating. In addition, psychoeducational approaches often include a fair amount of information on weight regulation to combat the wealth of misinformation provided by popular culture. Psychoeducation also usually covers the role of popular culture in promoting specific unobtainable aesthetic ideals.

The following are some particular items of information that might be shared with patients with eating disorders during psychoeducation: "Thin is beautiful" is a relatively recent belief that one may or may not choose to adopt. Images presented in popular media usually

have been altered by postproduction techniques so that they do not represent people as they actually look. In addition to the arbitrary nature of the thin ideal, it is not obtainable by most individuals. The body is designed to defend itself against significant weight loss. When caloric intake drops and weight loss begins, there is also a decrease in the basal metabolic rate, which accounts for the majority of calories consumed by the body. Because of this decrease, a diet that initially allows a patient to lose weight will eventually stop working as well. Further evidence of this weight-defending process includes drops in body temperature, blood pressure, and pulse rate as well as the growth of fine, downy hair (lanugo) on the torso; these changes represent serious medical consequences of starvation. Dietary restriction increases the risk of binge-eating episodes. Binge episodes introduce a large number of calories into the body. Self-induced vomiting and other methods of purging are ineffective means of compensating for binging, because the majority of calories consumed during binge episodes are retained. Finally, individuals who recover from AN weigh more after recovery but feel better about their bodies. Individuals who recover from BN do not weigh significantly more and feel better about their bodies.

Armed with such information, a patient is better equipped to attempt to rid herself of an eating disorder. Unfortunately, understanding what eating disorders are, what factors exacerbate them, what approaches decrease them, and the consequences of continued disordered eating may not provide patients with enough insight, motivation, support, or skills to alter their behaviors. Psychotherapy is typically designed to provide some combination of those factors. The first psychotherapy reviewed, psychodynamic therapy, is focused on enhancing insight.

Psychodynamic Therapy

Psychodynamic therapy seeks to reveal the underlying causes of eating disorders and was among the first therapies regularly employed with patients suffering from eating disorders. Symptoms are viewed as expressions of intrapsychic distress, and the patient may not be consciously aware of the presence of the distress or its source. Hilde Bruch, discussed previously in Chapter 6, was a pioneer in psychodynamically oriented therapy for eating disorders, and her approach has had a significant impact on the way eating disorders are understood and treated. Bruch was a proponent of a fact-finding, noninterpretive, collaborative style of therapy that encourages patients to recognize their own feelings and motivations (Bruch, 1978). Although the patient may not be aware of why she engages in disordered eating behaviors at the initiation of therapy, she has an active role in uncovering and understanding the processes driving those behaviors.

Psychodynamic therapies are nondirective. This means that the therapist does not choose the topics covered in sessions, and sessions are not organized around specific treatment goals. Instead, there is a more general goal: for the patient to explore the meaning behind her symptoms, with the assumption that she will improve as a consequence. Patients often set the agenda for what is discussed in therapy. Early sessions may focus on concrete topics that are of interest to the patient, such as fears of weight gain, food, and calories. The psychodynamically oriented therapist listens to this information to learn more about the patient and build rapport. However, the meaning of symptoms is usually thought to

reside beneath the surface level of the patient's behaviors and conscious fears. The therapist will therefore wait for the patient to turn to topics that seem to account for the high emotional importance given to food, eating, and weight.

The following example illustrates the psychodynamic perspective. An adolescent girl reported that her parents routinely argued at the dinner table. Her symptoms began shortly after her father got up in the middle of a meal and walked out on the family. After 15 months away, the father is now back home, and the girl finds that she can tolerate eating only when he is present. The eating disorder in this case could be interpreted as the patient's attempt to freeze the original dinner in time—refusing to allow the meal to continue until her father returned to the table.

Psychodynamic interventions have received relatively little **empirical support** in the treatment literature. One reason for this lack of clear support is that insight-oriented or psychodynamic interventions are frequently combined with other intervention methods, most often antidepressant medications and behavioral techniques (Brotman, Herzog, & Hamburg, 1988; Norring & Sohlberg, 1991; W. C. White & Boskind-White, 1981). This fact makes it difficult to discern what aspects of treatment contribute to change. In addition, because the therapist does not direct the course of therapy, psychodynamic therapies are not amenable to standardization. Directive treatments, discussed below, have been standardized through the creation of a treatment manual. In the absence of a treatment manual, it is difficult to ensure consistent application of therapy across participants in a given treatment condition. In addition, a nondirective, psychodynamically oriented therapy may have distinct goals that are not captured by, say, reductions in the number of binge and purge episodes per week—a commonly used metric for comparing the efficacies of different forms of treatment.

The ideal study design for assessing whether a treatment produces recovery is a **randomized controlled trial.** In a study of this sort, researchers randomly assign participants to a treatment or control condition and compare change over time in the conditions. The control condition may consist of no treatment or of an alternative treatment. This approach allows conclusions to be drawn about the impact of treatment on symptom change over time. Using a no-treatment control condition allows one to rule out the possibility that patients would have improved over time without the treatment under investigation. An active treatment control rules out the possibility that patients might have improved over time owing to a **placebo effect** (in which patients improve because they expect treatment to work). Comparing one active treatment to another using a randomized controlled trial is the best approach for testing which specific aspects of a treatment help patients improve.

Studies using randomized controlled trials have found that nondirective, psychodynamic therapies produce less symptom improvement than does cognitive–behavioral therapy (see next section) in the treatment of BN but generally do not yield different results from alternative treatments for AN (Brown & Keel, 2012). More specifically, studies employing random assignment to treatment conditions and consistent application of the therapeutic approach within each condition have found that for the treatment of BN, cognitive–behavioral interventions are superior to nondirective therapy (Kirkley, Schneider, Agras, & Bachman, 1985), short-term focal psychotherapy designed to reveal "underlying difficulties" (Fairburn et al., 1986), psychodynamically oriented supportive psychotherapy (Garner et al., 1993;

B.T. Walsh et al., 1997; Wilson et al., 1999), and psychoanalytic therapy (Poulsen et al., 2014) but have not found such differences for the treatment of AN (Zipfel et al., 2014).

Cognitive–Behavioral Treatments

Cognitive–behavioral therapy (CBT) is a form of directive therapy focused on the patient's present thoughts and behaviors (as opposed to relational features of the patient's rearing environment). In CBT, eating disorder symptoms are viewed as resulting from a combination of distorted cognitions that need to be elicited, challenged, and replaced and of reinforcement, both positive and negative, of behaviors by immediate consequences. Chapter 7 described various distorted cognitions that occur in eating disorders and discussed how disordered eating behaviors are positively and negatively reinforced.

As an example of how CBT approaches distorted cognitions, a cognitive–behavioral therapist might elicit from a patient a statement concerning the importance of being thin, such as "People will love me only if I'm thin." This belief can be challenged in several ways based on the patient's own observations. First, the patient can be asked to view couples in public settings and notice that people across a wide range of weights appear to be in happy relationships. Second, the patient can be invited to question the value of love from an individual who places so much importance on physical appearance. Third, the patient can be asked whether she loves anyone who is not thin (e.g., a grandparent or friend). In the end, the patient may come to adopt a belief that corresponds more closely to reality: "People who truly care about me as a person will love me no matter what I weigh."

One widely used cognitive–behavioral intervention, developed by Christopher Fairburn (1981), is divided into three stages. The first stage involves establishing control over eating with behavioral techniques. These techniques include:

1. Self-monitoring of food intake, binge eating, and inappropriate compensatory behavior with a diary
2. Prescription of a regular pattern of eating at least three meals and two or three snacks per day (going no more than two to three hours without eating)
3. Stimulus control (identifying triggers for binge-eating episodes and avoiding them)
4. Education on weight regulation, dieting, and risks of purging (as described above in the section on psychoeducation).

Figure 9.2 presents a behavioral analysis in which a patient describes the antecedents and consequences of her disordered eating behavior and considers alternative behavioral responses to its likely causes.

The second stage reduces dieting and body image disturbance through a combination of behavioral and cognitive techniques and trains the patient to engage in problem-solving. The third stage works toward maintaining progress and reducing the risk of future relapse. For example, patients are encouraged to recognize and challenge dichotomous thinking so that they can differentiate a lapse from relapse. Eliminating such all-or-nothing thinking allows patients to have slips without returning to the full eating disorder.

Because patients spend most of their lives outside therapy, each stage of Fairburn's approach, and CBT in general, emphasizes work that patients complete outside of sessions,

Antecedents	Behavior	Consequences
Incredibly stressful day at work, trying to make last-minute changes to a program and fix mistakes to meet a deadline	Started eating as soon as I got home to unwind—became a binge episode	Thoughts about work obliterated while eating—completely zoned out while eating After episode, felt bloated, stomach hurt, felt disgusted and disgusting
	Alternative behaviors to deal with feeling stressed:	
	Call a friend to talk about day	Laugh about prospect of submitting program with all mistakes as if they were intentional; decide to get together for dinner
	Go for a walk outside	Thoughts about work dissipate; feel relaxed, a little tired, and hungry for dinner
	Take a long, hot shower or bath	Thoughts about work dissipate; feel warm, relaxed, and ready for dinner

FIGURE 9.2 Behavioral analysis of a binge eating episode. Note: In each case, the alternative behavior is (a) incompatible with eating and (b) achieves the desired consequence of relieving stress from work.

which include homework assignments like maintaining a diary and writing out the stages of problem-solving. Jean used these methods in CBT to help her identify possible solutions to her binge eating.

Case Study: Jean

While reviewing her food diary in her fifth session (see Figure 9.3), Jean identified that her binge episodes typically happened in the evening when she was alone. Her therapist asked her to consider what about this context might be triggering the binge eating. Jean said that she felt like she used food to unwind from work and that when she was alone there wasn't anything to keep her from losing control. Her therapist asked Jean what she felt she needed to unwind from, and Jean explained that she tended to start her workday with a list of things she planned to complete and that when the day ended, she often felt disappointed that she hadn't

Food Journal

Day Monday Date February 18

Time	Food and Drink Consumed	Place	*	V/L	Context and Comments
7:45	1 bowl cereal w/ milk 1 cup decaf coffee	Home			Off to a good start. Determined to do well today.
8:50	1 cup decaf	Office			
10:35	1 apple	Office			
12:30	Strawberry yogurt 1 cup chicken soup	Cafeteria			
7:50	Macaroni & cheese Potato salad	Kitchen	*	V	Ate too much. Feel bad. Once again, I strayed from my diet.
8:10	Diet Sprite	Kitchen			
	Large piece of cheesecake		*		
	10 Oreos		*	V	Feel terrible. I lost control and couldn't stop. I am crying and I feel awful!!!!
	Bag of Doritos (16 oz)		*		
	2 Twix bars		*		
	Diet Sprite		*	V	
	5 scoops ice cream				
10:05	Diet Sprite	Bedroom			Feel tired/calm. Determined to do better tomorrow.
10:40	Diet Sprite	Bedroom			

Column 2: All food and drink consumed should be recorded, including binges. Each item should be written down as soon as it is consumed. Episodes viewed as meals are identified with brackets.

Column 4: An asterisk is recorded when the individuals feels that the food consumed was excessive.

Column 5: Record when self-induced vomiting (V), laxatives (L), or diuretics are used.

FIGURE 9.3 Food journal. *Note:* Adapted with permission of Guilford Publications from Fairburn, C. (1995). Overcoming binge eating. New York: Guilford Press; permission conveyed through Copyright Clearance Center, Inc.

gotten them all done and anxious about falling further and further behind. Her therapist then asked Jean how hungry she was when she got home, and Jean replied that she was very hungry. In her effort to get everything done, she sometimes would skip lunch, meaning that she would not have eaten since breakfast by the time she was getting home in the evening. The therapist then presented Jean with a sheet labeled "Problem-Solving" that listed six steps:

1. Identify the problem as early as possible
2. Specify the problem accurately
3. Consider as many solutions as possible (consider alternative list)
4. Think through the implications of each solution
5. Choose the best solution or combination of solutions
6. Act on the solution.

Jean's therapist then asked Jean to walk through her most recent binge episode using the steps of problem-solving. Initially Jean identified eating too much as the problem, but as

she was pressed to work back, she was able to identify that feeling hungry and stressed out preceded eating too much. Identifying hunger and stress as problems provided an earlier and more specific target for a solution. Jean then wrote down as many solutions to these problems as she could. These included taking an afternoon break to assess and prioritize her work, eating a snack during that break, and unwinding with some alternative to eating when she got home. In the last category, Jean listed watching TV and reading a book. However, her therapist gently challenged her to consider the implications of those solutions and whether they would be incompatible with eating. Jean acknowledged that she often did eat in front of the TV, particularly when watching shows like *The Big Bang Theory,* where the characters seemed to always be eating something. Jean felt that she could read without eating, particularly if she chose a setting outside her home. Based on this discussion, Jean added "in the park" to her list item for reading a book but crossed off watching TV. The therapist then encouraged Jean to continue adding to the list, noting that the more solutions she could generate, the more opportunities she would have to solve the problem of work-related stress. Jean added going for a walk (as a natural extension of taking a book to the park) and calling a friend, and her therapist then offered taking a hot shower or soaking in the tub, noting that very few people eat in the shower.

In the end, Jean's list consisted of the following:

1. Take a break in the mid-afternoon to focus on what she needed to get done and to prioritize what she would finish that day and what she would tackle the next day.
2. During that mid-afternoon break, eat a snack.
3. When she got home, engage in an alternative activity (something other than eating) to help her unwind:
   a. Read a book in the park
   b. Go for a walk
   c. Call a friend
   d. Take a hot shower or bath.

Jean and her therapist then discussed which options or combinations of options represented the best solution. Together they realized that the answer depended on other factors. For example, taking a walk or going to the park would be great solutions in good weather but would not be appealing in bad weather. Similarly, calling a friend was a good solution for getting emotional support when something beyond the usual work-related stress was bothering Jean, but it relied on someone's being available. So Jean and her therapist discussed ways of identifying when that was the case as early as possible, reaching out to friends as early as possible to set up the call, contacting as many friends as she needed to find someone, and coming up with what she would do if no one was available. Then they discussed the context in which taking a hot shower or bath would be the best solution.

Finally, Jean and her therapist agreed that for her homework, she would use this problem-solving approach to tackle the feelings of hunger and stress that appeared to trigger her binge episodes. The therapist framed this assignment as an experiment from which they would both learn whether hunger and stress were actually contributing to Jean's binge

episodes. If the solutions reduced her hunger and stress and Jean didn't binge, that would be strong evidence that those factors were indeed responsible for her binging. If the solutions reduced her hunger and stress but Jean still binged, that would suggest that other factors contributing to her binge episodes needed to be identified and addressed. If the solutions did not reduce her hunger and stress, then Jean and her therapist would need to identify different solutions. The therapist emphasized that problem-solving was a general strategy—a method that could be applied to a wide range of problems that everyone faces in their daily lives.

Numerous studies using manual-based treatments and random assignment to treatment and control conditions support the efficacy of Fairburn's and other forms of CBT in the treatment of BN and BED (Brown & Keel, 2012). A meta-analysis of treatment studies for BN found that CBT was superior to medication for reducing binge frequency, purge frequency, disordered eating attitudes, and depression (Whittal, 1999). In the U.K. the National Institute for Clinical Excellence (2004) gave CBT, delivered in 16 to 20 sessions over four to five months, an "A" grade for treating BN, meaning that the institute deems it the first-line treatment of choice for BN. In addition it was a recommended treatment for BED. Although CBT has been adapted for treatment of patients with AN, it has not demonstrated unambiguous superiority to alternative treatments for this disorder (Brown & Keel, 2012).

Interpersonal Therapy

Interpersonal therapy for BN was originally developed as a control condition for CBT (Fairburn et al., 1993). Although CBT had been observed to be superior to short-term focal psychotherapy (Fairburn et al., 1986), patients in the latter therapy did improve over time. To determine whether this improvement was a consequence of self-monitoring and psychoeducation (therapeutic aspects shared between the interventions), a new control intervention was adapted from an existing interpersonal therapy for depression (Klerman, Weissman, Rounsaville, & Chevron, 1984). The rationale for interpersonal therapy was that patients with BN often report a number of problems in interpersonal domains. These include being conflict avoidant, having internal conflicts between the needs for independence and closeness, and having difficulties with role expectations and social problem-solving (Apple, 1999).

Interpersonal therapy for BN comprises three phases:

1. *Identify interpersonal problems associated with the onset of BN.* This phase involves taking a personal history and examining significant life events, mood and self-esteem, and relationships. Patients are encouraged to associate bulimic symptoms with life experiences and to understand the role of interpersonal problems in the emergence of symptoms. The rationale of interpersonal therapy is covered in this phase, as well as common problem domains. Patients are encouraged to select specific problems on which to focus throughout treatment.
2. *Work on identified interpersonal problems.* Like psychodynamically oriented therapies, interpersonal therapy allows patients to introduce topics to therapy discussions as they

relate to the problem areas defined in phase 1. Techniques such as open-ended questioning, role playing, examining the consequences of change, and encouraging expression of feelings are used to assist patients (Apple, 1999).

3. *Discuss feelings about termination.* This phase involves reviewing gains and anticipating future problems and solutions.

Importantly, patients' eating problems or concerns about weight or shape are not mentioned by the therapist after their initial assessment.

Similar to results from the initial study comparing CBT with short-term focal psychotherapy (Fairburn et al., 1986), CBT was superior to interpersonal therapy at the end of treatment for BN (Agras et al., 2000; Fairburn et al., 1993). However, similar to results from another study that compared CBT with nondirective therapy (Kirkley et al., 1985), these differences were not maintained at follow-up (Fairburn et al., 1993, 1995). Moreover, at 12-month follow-up, patients who had received interpersonal therapy for BN were doing as well as those who had received CBT (Fairburn et al., 1993). Interpersonal therapy has also been found to be effective in the treatment of BED, producing improvements that are comparable to those observed for CBT (Wilfley et al., 2002; Wilson, Wilfley, Agras, & Bryson, 2010).

McIntosh et al. (2005) compared the outcomes in 56 adult patients with AN receiving 20 sessions of one of three treatments: interpersonal therapy, CBT, or nonspecific supportive clinical management. Results suggested that nonspecific clinical management was superior to interpersonal therapy with respect to global outcome. Interpersonal therapy and CBT did not differ with respect to global outcome or weight gain, but patients who received interpersonal therapy had worse restraint scores and were less likely to be rated "significantly improved" than patients who received CBT. At five-year follow-up, no differences were found on any outcome among clinical management, CBT, and interpersonal therapy (Carter et al., 2011). Thus although individuals treated with interpersonal therapy may continue to improve after treatment completion, there is no evidence that this treatment is superior to alternative treatments for AN, BN, or BED, and there is evidence that faster improvements may be obtained with CBT for BN and perhaps with nonspecific supportive clinical management for AN in adults.

Because CBT and interpersonal therapy appear to reduce eating disorder symptoms through different methods, some researchers have examined whether interpersonal therapy may help patients who do not respond to CBT (Agras, Telch, Arnow, Eldredge, & Marnell, 1995; J. E. Mitchell et al., 2002). However, neither patients with BN (J. E. Mitchell et al., 2002) nor patients with BED (Agras et al., 1995) who failed to respond to CBT showed increased success following a shift to interpersonal therapy. A particular problem observed with interpersonal therapy in one study was a high dropout rate (J. E. Mitchell et al., 2002), suggesting that patients may become demoralized when initial treatment does not work, similar to what happened in Valerie's case.

Researchers have therefore begun focusing on developing treatments that enhance CBT by adopting components of other interventions. These include dialectical behavior therapy, emotion-focused therapy, and acceptance and commitment therapy, as well as eating disorder–specific interventions such as enhanced cognitive–behavior therapy

(CBT-E) for eating disorders (Z. Cooper & Fairburn, 2011), emotion acceptance behavioral therapy for AN (Wildes, Marcus, Cheng, McCabe, & Gaskill, 2014), and integrative cognitive–affective therapy for BN (Wonderlich et al., 2014). Collectively, these approaches are referred to as *third-wave treatments*.

Third-Wave Treatments for Eating Disorders

Dialectical behavior therapy was originally designed to treat individuals with a personality disorder characterized by poor impulse control, self-destructive behavior, interpersonal difficulties, and significant mood fluctuations. Because many of these features are present in patients with eating disorders—particularly those with bulimic symptoms—researchers adapted this treatment for eating disorders.

Traditional dialectical behavior therapy is provided as a combination of individual CBT focusing on symptoms in order of decreasing urgency (e.g., harmful purging behavior is addressed before the fear of never finding a romantic partner) and skills training groups that focus on building strength in four areas:

1. **Mindfulness**—the ability to be aware of internal feelings and external demands in a way that combines emotions with intellect in making wise choices
2. **Distress tolerance**—the ability to develop safe ways of coping with painful emotions without resorting to impulsive (and often self-destructive) behaviors that ultimately increase emotional pain
3. **Interpersonal effectiveness**—the ability to relate to people in a way that meets personal needs, the needs of the other person, and the need for self-respect
4. **Emotional regulation**—the ability to experience emotions without having extreme fluctuations that interfere with life function.

Dialectical behavior therapy was successful in treating BN in one controlled study (Safer, Telch, & Agras, 2001), and results of two uncontrolled studies suggested that it may be useful in the treatment of both BN and BED (Palmer et al., 2003; Telch, Agras, & Linehan, 2000). Wisniewski and Kelly (2003) have reported that this treatment can be applied to eating disorders with relatively modest revisions, such as including focusing on eating disorder behaviors in the treatment goals and adding a nutrition skills module to the skills training groups (see the section on nutritional counseling below).

Early clinical trials of other third-wave interventions have provided promising results but have come from uncontrolled studies (Chen et al., 2015; Dalle Grave, Calugi, Doll, & Fairburn, 2013; Fairburn et al., 2013; Wildes et al., 2014; Wnuk, Greenberg, & Dolhanty, 2015) or have not demonstrated advantages uniquely associated with third-wave interventions (Juarascio et al., 2013; Wonderlich et al., 2014). Thus these interventions appear to be useful but not necessarily superior to the original interventions they were intended to surpass.

Many of the problems patients with eating disorders have in interpersonal function are thought to emerge within the context of poor family functioning (see Chapter 6). Thus interventions focused solely on the individual may fail to address a significant source of

difficulties—the interpersonal context in which these problems emerge. Because AN is associated with a younger age of onset than BN or BED, this limitation is particularly relevant for AN patients, who are often still living at home. Family therapy seeks to address interpersonal function within the family as a system and is the only treatment for AN that has demonstrated superiority to alternative treatment in adolescent patients.

Family-Based Therapy

Family therapy is most often used when patients are children or adolescents living at home. As described in Chapter 6, traditional family systems therapy involved evaluating the entire family as the patient (Minuchin et al., 1978). A newer approach to family-based therapy invites family members to be members of the treatment team rather than viewing family members as patients (le Grange, 1999). Following a study by Russell, Szmukler, Dare, and Eisler (1987) indicating the superiority of a family treatment developed at the Maudsley Hospital in England for younger eating disorder patients compared to individual supportive therapy, the Maudsley model of family therapy has received increased attention and empirical support in the treatment of adolescents with AN.

Like CBT and interpersonal therapy, the Maudsley model of family therapy has three phases:

1. *Refeeding the client*. Therapists support and reinforce parents' efforts to refeed their child and encourage parents to form a united front. Meanwhile, siblings are encouraged to be supportive of the patient; this aspect of therapy reinforces appropriate boundaries between parental and sibling subsystems, as described in Chapter 6. Families are encouraged to devise their own plans for refeeding.
2. *Negotiations for a new pattern of relationships*. Once patients show willingness to participate in refeeding and achieve weight gain, weight gain with the least amount of conflict is allowed. Although symptoms remain central to this phase of treatment, other family issues are introduced to therapy. Only issues that affect the parents' ability to ensure the patient's weight gain are covered, and only to the extent that they are relevant to the patient's symptoms.
3. *Termination*. After the patient has achieved a healthy weight, the focus shifts to encouraging a healthy relationship between patient and parents. This step is particularly important, because up to this point the patient's illness has formed the basis of family interactions. Reflecting patients' ages, this phase often covers themes of increased autonomy, appropriate family boundaries, and preparation for children's departure from home (Dare, Eisler, Russell, & Szmukler, 1990).

Controlled studies have supported the efficacy of this intervention in adolescent patients with AN (le Grange, Eisler, Dare, & Russell, 1992; Lock et al., 2010; Robin et al., 1999; Russell et al., 1987). For example, Lock and colleagues (2010) randomly assigned 61 adolescent patients with AN to family-based therapy and 60 patients to a psychodynamically oriented adolescent-focused therapy. Overall, both treatments were acceptable to patients, with 85% accepting the condition to which they were randomly assigned (an even

better acceptability rate than that for specialized outpatient treatment in the Gowers et al., 2010, study described above). Full remission occurred in a higher percentage of patients who received family-based therapy than of patients receiving adolescent-focused therapy at 6 months (40% vs. 18%) and 12 months (49% vs. 23%). In addition, significantly more patients in adolescent-focused therapy than in family-based therapy required hospitalization to maintain a minimally safe weight during the course of treatment (37% vs. 15%). At the end of treatment, patients who received family-based therapy had achieved significantly higher BMIs and reported significantly lower disordered eating attitudes and behaviors; however, these differences diminished at 6- and 12-month follow-up. Overall, results across studies indicated that family-based therapy is superior to psychodynamically oriented individual therapy in adolescents with AN. Moreover, sustained benefits of family-based therapy were found in a five-year follow-up study (Eisler et al., 1997) of the original Russell et al. (1987) study.

In considering factors that may contribute to the efficacy of family-based therapy, it is worth noting that by actively engaging parents in refeeding their children, this treatment offers the beneficial structured environment of inpatient or residential treatment in patients' own homes. In this context, shifting control of eating from the environment to the child can be more gradual and can be matched to the child's growing ability to feed herself. In addition, twin studies indicate that family environment has a significant influence on disordered eating attitudes and behaviors in young children; this finding may explain why family-based therapy has been found to be superior for the treatment of children and adolescents with AN but not adults with AN (Russell et al., 1987).

Treatment Mediators and Moderators

The preceding sections reviewed evidence regarding the efficacy of treatments. In addition to wanting to know which treatments work best in helping patients recover, researchers are interested in understanding why treatments work and for whom they work best.

Answering the first question—why does a treatment work—is important for discriminating key components of a treatment that are required for its success from parts of the treatment that might be eliminated without reducing efficacy. Doing this requires identifying **mediators** of treatment response. A mediator is a link in a causal chain of events. For example, CBT is designed to reduce dietary restraint, and reductions in dietary restraint are intended to reduce risk for binge eating. Thus reductions in dietary restraint as a consequence of CBT constitute a mediator for reductions in bulimic symptoms. Supporting this model, a significant reduction in dietary restraint by the fourth week of CBT contributed to reduced binge and vomiting frequency after treatment (Wilson, Fairburn, Agras, Walsh, & Kraemer, 2002). Identification of treatment mediators makes it possible to improve treatments. Unfortunately, few mediators of treatment response have been identified. A recent review (Lock, 2015) identified no mediators of treatment response in treatment studies of children and adolescents.

Answering the second question—in whom does treatment work best—involves identifying **moderators** of treatment response. A moderator is a factor that contributes to a

treatment's being more successful in some people than others. Unlike a mediator, it is a difference between people rather than a change within a person that explains why a treatment worked. For example, among adolescents treated for AN, greater obsessive–compulsive symptoms were associated with a better response to longer-duration family-based therapy in the format that focuses on the family system (Lock, 2015). Similarly, when patients had greater eating disorder severity, they showed a more robust response to family-based therapy as compared with individual therapy (Lock, 2015). Together, these findings suggest that patients with less severe symptoms may do equally well in different forms of treatment (regardless of differences in duration or focus), whereas those with the most severe illness benefit most from family-based therapy.

Nonpsychotherapy Interventions

Many patients with eating disorders benefit from multimodal, team-based approach to treatment involving a combination of psychotherapy, nutritional counseling, medication, and medical management. The following sections discuss the roles of nutritional counseling and medication in the treatment of eating disorders. While nutritional counseling alone would not be recommended for the treatment of eating disorders, evidence suggests that symptoms of BN may remit with medication alone (Goldstein et al., 1995).

Nutritional Counseling

Despite the great attention they pay to calories and food intake, many individuals suffering from eating disorders lack basic knowledge of what constitutes a well-balanced diet. This unawareness is somewhat understandable given that most people do not follow optimally nutritious diets. For example, up to 75% of girls aged 9–13 years consume less than the recommended daily amount of calcium, and this figure increases to 90% for females aged 14–30 (U.S. Department of Agriculture, 2009). Further, patients with eating disorders often have skewed understandings of guidelines for daily caloric intake. Thus nutritional counseling is an important aspect of treatment for eating disorders. The goals of nutritional counseling are to "attain medical stability, to normalize eating behaviors and weight, and to reestablish a healthy relationship with food" (Brunzell & Hendrickson-Nelson, 2001, p. 217).

In addition to providing basic nutritional information, dieticians work with patients to reduce fears of forbidden foods, eliminate food myths, and help them recognize feelings of hunger and fullness. Dieticians explain that calories in food come from carbohydrates, protein, and fats. Along with vitamins, minerals, and water, these sources of calories represent nutrients that are used to build the body, regulate its functions, and provide energy. Carbohydrates represent the primary source of energy for the body and should contribute 50–65% of the calories consumed in a day. Protein is important for building and maintaining organs, muscles, skin, hair, antibodies for fighting illnesses, enzymes, and hormones. It is also important for regulating water balance in the body. Protein should constitute 10–20% of total daily caloric intake. Finally, dietary fat provides essential fatty acids, carries

fat-soluble vitamins, and contributes to the maintenance and function of body tissue. Fat should contribute 20–30% of the calories consumed in a day. In treatment, patients are gradually introduced to more varied diets that include recommended proportions of calories from carbohydrates, protein, and fats.

The relationships among food intake, energy expenditure, and weight are often reviewed in some detail during nutritional counseling so that patients can act as informed consumers. Dieticians can also instruct patients in behavioral techniques to plan for eating in social situations as well as for responding to urges to binge or to restrict food intake.

Case Study: Jamie

Jamie began meeting regularly with a dietician in addition to taking antidepressant medications. In one of the most illuminating sessions the dietician showed Jamie a picture (see Figure 9.4) demonstrating how he could easily consume the same number of calories in a single fast-food meal as he might take in by consuming healthier food over the course of the day. Jamie had always thought that reducing his caloric intake would leave him feeling starving, but as he compared the amounts of food, he realized that he could actually eat more food if he switched to more complex carbohydrates and increased his intake of fruits and vegetables. The dietician also worked with him to identify recipes that would allow him to prepare foods that would taste good in addition to being good for him.

FIGURE 9.4 Comparison of 1,980 kcal in a fast food meal versus 1,962 kcal consumed across the day as breakfast, lunch, and dinner.

Medication

Several medications have been used to treat AN, BN, BED, and night eating syndrome. These include antidepressants, mood stabilizers, opioid antagonists, tetrahydrocannabinol (the primary component of marijuana), antipsychotics, and stimulants (Brown & Keel, 2012). The atypical antipsychotic olanzapine has shown success in improving weight-related outcomes in adult patients with AN in randomized **double-blind placebo-controlled studies** (Attia et al., 2011; Bissada, Tasca, Barber, & Bradwejn, 2008; Brambilla et al., 2007). Antidepressants have emerged as the medications of choice in the treatment of BN (Brown & Keel, 2012). Fluoxetine (commonly known by the trade name Prozac), a selective serotonin reuptake inhibitor (SSRI), is the only drug to have U.S. Food and Drug Administration approval for treating BN (American Psychiatric Association, 2000). In contrast, another, non-SSRI antidepressant, bupropion (trade name Wellbutrin), is not recommended for the treatment of eating disorders, because of evidence that it may increase the likelihood of seizures in this population (Diamond, 2002). Some data support the efficacy of SSRIs in the treatment of BED (McElroy, Guerdjikova, Mori, &Melia, 2012) and night eating syndrome (O'Reardon et al., 2006).

Case Study: George

George couldn't be sure if he was receiving the active medication in the treatment study (see Chapter 4), because no one he spoke to could tell him, including the physician who followed him for side effects and adjusted the dosage. However, George felt certain that he was taking the active medication, not the placebo. At first, whatever he was taking made him a little lightheaded and caused diarrhea. However, this phase quickly passed, and George noticed that he felt more focused at work and that when he ate he felt full much more quickly. As a study participant, he was not allowed to seek any additional treatment. However, this restriction wasn't a problem for him, because he was sleeping better, did less night eating, was losing weight, and didn't have to spend hours a week in therapy sessions or meetings or preparing special diet meals for himself. His only question was what would happen when the study was over. The physician who was meeting with him said that this would be up to George. If he was in the medication condition, he could either remain on the medication or stop. If he was in the placebo condition, he could either ask to receive the medication or not. Because the treatment was new, no information was available concerning the medication's long-term efficacy. So there was no way of knowing whether improvements achieved with the medication would be maintained.

For George, and for many patients with night eating syndrome or BED, achieving weight loss is as important a treatment goal as achieving control over eating. Although it might seem that eliminating disordered eating behaviors would lead to reduced weight, this is not necessarily the case. Thus in addition to treatments targeting disordered eating behavior, several trials are underway to address weight loss for BED. Whether these studies use behavioral weight loss programs or medication, findings suggest that the treatments produce the changes that they are designed to produce (McElroy, Guerdjikova, Mori, Munoz, & Keck, 2015). That is, treatments that target disordered eating behaviors achieve reductions in disordered eating behaviors, and treatments that target reductions

in weight achieve reductions in weight. Thus far no single treatment has emerged that has achieved both goals in patients with BED though combinations of treatments may hold promise in the future.

Conclusion

Several efficacious treatment approaches have been identified for BN. These include cognitive–behavioral therapy, interpersonal therapy, and antidepressant medication. Several treatments that work for BN also appear to be successful in treating BED. In contrast to results for BN and BED, treatments with demonstrated efficacy for adults with AN are limited. For adolescents with AN, the Maudsley model of family-based therapy appears to provide the best available treatment.

Despite evidence of efficacious treatments, most individuals with eating disorders do not appear to receive those treatments. Individuals with eating disorders may be reluctant to seek treatment. Such reluctance can be caused by fear of becoming fat, shame over disordered eating behaviors, or an inability to recognize the dangers of eating disorders. In addition, there have been reports of problems with insurance coverage for patients who do seek treatment for an eating disorder. Finally, even when individuals seek treatment with insurance coverage, they often are not offered treatments with demonstrated efficacy (A. M. Simmons, Milnes, & Anderson, 2008; von Ranson, Wallace, & Stevenson, 2013). In a survey of randomly selected psychologists, the majority reported that they had never received training in the use of manual-based, empirically supported treatment approaches for eating disorders and that they used methods other than CBT or interpersonal therapy when working with eating-disordered patients (Mussell et al., 2000). In a survey of eating disorder specialists, by contrast, most had received training in evidence-based treatments, and training was a significant predictor of whether or not clinicians used these treatments (A. M. Simmons et al., 2008). Thus in addition to continued efforts to identify efficacious treatments for eating disorders, greater efforts are needed to disseminate information about and training for those interventions (Wilson, Grilo, & Vitousek, 2007).

At this time, multimodal, team-based approaches are recommended for treating eating disorders. A typical treatment team would include a physician to monitor medical well-being, a dietician, a therapist, and, potentially, a psychiatrist for medication management. The use of a multidisciplinary team ensures that there will be appropriate expertise to address the complexity of eating disorders given their impact on physical, emotional, cognitive, and social well-being.

Key Terms

Cognitive–behavioral therapy
Cost effectiveness
Dialectical behavior therapy
Distress tolerance

Double-blind placebo-controlled study
Emotional regulation
Empirical support
Interpersonal effectiveness
Interpersonal therapy
Mediator
Mindfulness
Moderator
Placebo effect
Psychodynamic therapy
Psychoeducation
Randomized controlled trial

10

Prevention

As noted in Chapter 9, the number of individuals suffering from eating disorders far exceeds the availability of treatment. For this reason, a number of experts in the field have focused on prevention. Simply put, effective prevention would save time, money, and, most importantly, suffering.

This chapter starts by reviewing different theoretical models, or paradigms, of prevention. It then describes different levels of intervention and provides specific examples of eating disorder prevention programs within each level. Evidence of these programs' efficacy is reviewed for each level. The chapter ends with an examination of challenges for prevention research and future directions for the important work of preventing eating disorders.

Prevention Paradigms

Just as different theoretical orientations contribute to the development of psychotherapies, different theoretical orientations contribute to the development of prevention programs. Resulting paradigms of prevention reflect different ways of conceptualizing health and how it is maintained.

One common model is known as the **Disease-Specific Pathways Model** (Levine & Smolak, 2001) or **Disease Prevention Paradigm** (Rosenvinge & Borresen, 1999). Programs using this model seek to identify and then modify the specific risk factors that contribute to the etiology of eating disorders. For example, in the Disease Prevention Paradigm, a girl would be encouraged to develop a positive body image to prevent her from developing an eating disorder. Thus the success of a prevention program within this model depends on the accurate identification of specific risk factors and the ability to modify them.

A variation of the Disease Prevention Paradigm is the **Nonspecific Vulnerability-Stressor Model** (Levine & Smolak, 2001). As with the Disease Prevention Paradigm, programs using the Nonspecific Vulnerability-Stressor Model seek to identify and modify risk factors that contribute to the etiology of eating disorders. However, rather than focusing on specific risk factors thought to relate uniquely to the onset of eating pathology, this model addresses general risk factors that contribute to the etiology of many related

problems (see Chapter 4 for a discussion of general versus specific risk factors). For example, in the Nonspecific Vulnerability-Stressor Model, a girl would be encouraged to develop a positive self-image to prevent her from developing problems such as depression and eating disorders.

The **Health Promotion Paradigm** (Rosenvinge & Borresen, 1999) overlaps with the Nonspecific Vulnerability-Stressor Model in seeking to maximize overall health. However, the Health Promotion Paradigm emphasizes *protective factors* rather than risk factors. Whereas a risk factor promotes illness when present and does nothing when absent, a protective factor promotes wellness when present and does nothing when absent. Rosenvinge and Borresen (1999) have argued for using the Health Promotion Paradigm instead of the Disease Prevention Paradigm because the specific risk factors for eating disorders are not well understood and because focusing on information related specifically to eating disorders emphasizes the very things one is attempting to prevent. For example, under the Disease Prevention Paradigm, a girl who never gave much thought to her weight or shape might become more focused on them during a program concerned with body image.

In addition to emphasizing protective factors, the Health Promotion Paradigm advocates interventions designed for communities as well as individuals. In other words, the targets for change include community action as well as the behavior of a given individual in a community. For example, in the Health Promotion Paradigm, schools would be encouraged to promote valuing individual differences with regard to race, sex, and weight. This intervention would seek to reduce racism, sexism, and *weightism* (overvaluation of thinness and denigration of fatness). The focus would be on system-wide actions that influence health as well as on individual students' decisions about how to treat people who differ across a range of physical characteristics. While the goal of instilling the value of diversity among schoolchildren is not specifically related to the goal of preventing eating disorders, promoting health in the general population can have the consequence of preventing illness, including eating disorders, in individuals.

Another model of prevention that looks beyond the role of individual factors is the **Empowerment-Relational Model** (Levine & Smolak, 2001). This model is rooted in feminist theory, and programs using it seek to empower girls to transform their environments. Thus while the target for change is the environment, the agent of change is the individual girl. For example, in the Empowerment-Relational Model, a girl would be encouraged to create her identity based on her skills rather than her physical appearance and to actively challenge social messages that objectify women and girls.

Reviewing the above examples, one can see variations in the methods used to prevent eating disorders. However, these examples exaggerate the differences among the models. In practice, prevention programs tend to combine aspects of each model. There is no contradiction between having schools promote the value of diversity and encouraging girls to develop a positive self-image that includes what their bodies can do as an aspect of positive body image. The following section provides a brief overview of themes and content common to several prevention programs and relates them to the models described above.

Prevention Themes and Content

Across different types of prevention programs, certain themes are commonly emphasized and reflected in program content. Consistent with the Disease Prevention Paradigm, these themes tend to reflect attempts to directly address specific risk factors that emerge from the current sociocultural context (see Chapters 5 and 6). For example, many programs attempt to prevent or reduce weight preoccupation and dieting because these are considered risk factors for the development of eating disorders. To achieve these ends, programs educate participants on the arbitrary nature of the thin ideal, determinants of body size, nutritional needs, consequences of dieting, and consequences of disordered eating.

In addition to focusing on specific risk factors for eating disorders, several programs also seek to promote resilience by addressing more general factors related to mental health. Consistent with the Nonspecific Vulnerability-Stressor Model, programs address methods for improving self-esteem, stress management, assertiveness, and problem-solving skills. Each of these skills increases girls' resilience to environmental risk factors stemming from society (see Chapter 5) and their immediate social contexts (see Chapter 6).

Consistent with the Health Promotion Paradigm, several programs offer guidance on healthy eating and exercise habits to provide students with healthy options for weight control. This intervention is based on the premise that having accurate information about nutrition and exercise helps protect adolescents both from developing an eating disorder and from becoming obese. No prevention program, of course, endorses weight control for the purpose of trying to achieve an unrealistic and unhealthy thin ideal.

Finally, many prevention advocates seek to address change at a broader sociocultural level. Prevention programs have attempted to increase students' awareness of discrimination against the overweight and obese so that they will challenge weightism as readily as they would challenge racism (Steiner-Adair et al., 2002). They also have sought to improve students' awareness of how peer pressure and teasing increase body dissatisfaction and unhealthy weight loss behaviors and to incorporate these messages into broader anti-bullying campaigns. In addition to helping girls recognize, challenge, and reject social messages about the importance of being thin in their immediate social environments, such programs advocate changing the messages presented in popular culture. They have initiated dialogues with magazine editors, advertising agencies, companies that market products to women, and government representatives. Consistent with the Empowerment-Relational Model, the goal is to get students actively involved in changing the social environmental factors that may contribute to eating disorders.

Implementing Prevention and Levels of Intervention

After determining the goal of, audience for, and content of a prevention program, its designers must decide how and when to deliver it. In general, prevention means stopping an event before it occurs; this has been referred to as *primary prevention*. However, individual differences in when problems begin as well as challenges in identifying when problems begin (e.g.,

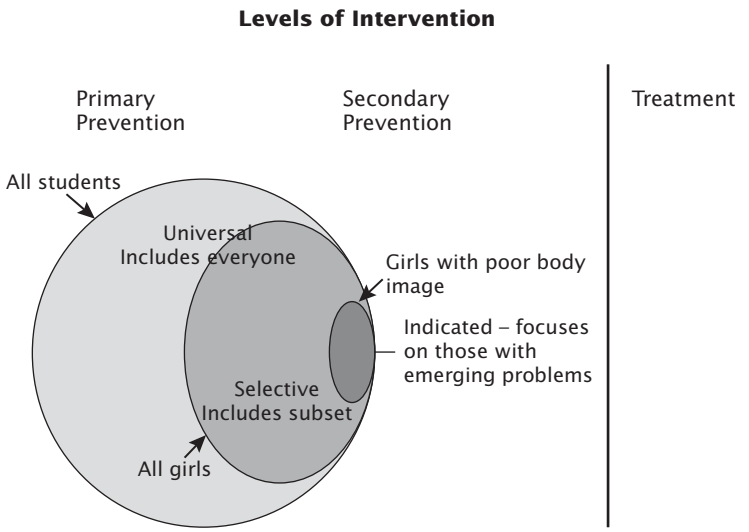


FIGURE 10.1 Levels of Intervention for Prevention Programs.

do they begin as early as infant feeding experiences?) make this a lofty goal. Instead, prevention programs can be divided into three categories based on the types of population they address: universal, selective, and indicated (see Figure 10.1). **Universal prevention programs** work with a general population, such as students in a school system. **Selective prevention programs** focus on individuals who are at increased risk for eating problems (e.g., only the girls in a school). **Indicated prevention programs** are aimed at individuals who are already reporting problems with their eating attitudes and behaviors.

Indicated prevention programs work toward the goal of *secondary prevention*—stopping emerging problems from developing into more serious problems. The distinction between a treatment to reduce weight preoccupations in order to improve body image and an indicated prevention to reduce weight preoccupation in order to prevent eating disorders is not necessarily captured by what happens *during* each type of intervention. Instead, it lies in their goals and measured outcomes. The treatment program would be successful if weight preoccupation decreased, whereas the indicated prevention program would be successful only if weight preoccupation decreased and future risk for eating pathology was reduced. Thus the bar for establishing the success of a prevention program is quite high, because to be considered effective, it must affect a distal outcome.

The following sections describe specific examples of prevention programs designed for each level of intervention (universal, selective, and indicated) and review evidence of their success (or lack of success) in altering knowledge, attitudes, and behaviors related to disordered eating.

Universal Prevention Programs

Planet Health was a universal prevention program that followed the Health Promotion Paradigm (Austin, Field, Wiecha, Peterson, & Gortmaker, 2005). Researchers at the

Harvard School of Public Health collaborated with the wellness coordinator for Boston Public Schools. Together they developed an innovative school-based curriculum aiming to prevent childhood obesity by promoting knowledge and specific behavioral changes that would contribute to a healthier lifestyle. The program used a two-year curriculum for the sixth and seventh grades that was integrated into the teaching of four major subjects (language arts, math, science, and social studies) as well as physical education. For example, information about nutrition and exercise provided the context for math problems so that students would both learn the topics specific to the subject area (e.g., algebraic equations) and learn to apply learned skills in making choices about food and activity. Planet Health also emphasized four behavioral changes:

1. Increase daily consumption of fruits and vegetables
2. Decrease (but not eliminate) daily consumption of high-fat foods
3. Reduce time spent watching TV or on the computer ("screen time")
4. Increase moderate and vigorous physical activity.

To test the efficacy of the program, 10 schools were randomly assigned to either the intervention condition, in which students followed the Planet Health curriculum, or the control condition, in which the standard curriculum was followed.

With regard to the targeted behaviors, both girls and boys in the program demonstrated increased fruit and vegetable consumption and reduced television viewing compared with children in the control schools (Austin et al., 2005). In addition, obesity prevalence in girls decreased by half in the intervention schools compared with the control schools, and reduction in time watching TV predicted decreases in obesity in girls. More relevant to the focus of this chapter, however, the program also reduced risk for disordered eating. Among girls in control schools, 6.2% reported that they began purging as a means to control their weight or shape at 21 month follow-up. In contrast, only 2.8% of girls in the intervention schools had started purging for these purposes at follow-up (Austin et al., 2005). These results suggest that an educational program that taught girls to engage in a healthier lifestyle both reduced their risk for obesity and reduced the likelihood that they would turn to unhealthy and extreme weight control methods. Unfortunately, boys in the intervention program did not demonstrate similar reductions in obesity risk; the reason for this difference is unclear. Further, girls who participated in Planet Health maintained the benefits from the program over follow-up (Austin et al., 2007). Data at three-year follow-up indicated that reduced TV viewing time played a specific role in decreasing girls' subsequent use of purging and diet pills (Austin et al., 2012).

In light of these findings as well as the discussion of risk factors in Chapters 4 and 5, a key aspect of Planet Health's success may be reducing behavior (TV viewing) that increases risk in two ways. TV viewing both exposes girls to media images that promote the thin ideal, and it involves inactivity that increases their risk for weight gain, increasing discrepancy from this ideal.

Like Planet Health, many universal prevention programs have been initiated in schools because they provide an ideal audience. Given their educational setting, these programs have

often emphasized psychoeducation (see Chapter 9). Not surprisingly, studies of such programs have often found improved knowledge among participants (Dalle Grave, De Luca, & Campello, 2001; Kater, Rohwer, & Levine, 2000; Levine, Smolak, & Schermer, 1996), and several have reported improved attitudes about body image (Dalle Grave et al., 2001; Kater et al., 2000; Varnado-Sullivan et al., 2001; Wade, Davidson, & O'Dea, 2003). However, unlike Planet Health, many of these programs have not been found to lead to improvement in behaviors (Austin, 2000; Levine & Smolak, 2001; Littleton & Ollendick, 2003). This finding raises questions concerning what has kept these other programs from having a more positive impact.

One possible reason for the limited success of universal prevention programs is the short durations of interventions. With the exception of Planet Health, interventions have ranged from 3 sessions (Buddeberg-Fischer & Reed, 2001) to 10 sessions (Kater et al., 2000; Levine et al., 1996). Given the multiple factors contributing to the development of eating disorders (see Chapters 4–8), it would be miraculous if a 10-session intervention could prevent them. The limited duration of interventions reflects constraints on incorporating new curriculum material at schools already being pressured to meet ever-increasing demands with ever-decreasing resources. For example, several prevention programs have relied on teachers to conduct sessions (Smolak, Harris, Levine, & Shisslak, 2001). Although health-related school staff support the inclusion of prevention material in school curricula (Neumark-Sztainer, Story, & Coller, 1999), this support does not necessarily translate into full adherence by teachers in implementing the program. For example, in the universal prevention program of Levine et al. (1996), 31% of teachers did not deliver a one-hour lesson on media analysis, and 50% devoted five minutes or less to explaining a key point in the lesson.

Thus time constraints in school settings likely contribute both to the low number of sessions and to incomplete delivery of information. Such time constraints limit the use of interactive techniques that are more effective than traditional teaching methods (Stice and Shaw, 2004). A key finding from the three-year follow-up of Planet Health participants was that the best outcomes were observed for girls in schools where there was active teamwork among staff, where more lessons were devoted to reducing TV viewing, and where staff participated in programs that emphasized the goal of reducing TV viewing (Austin et al., 2012). This result suggests that universal programs can be highly effective if there is a strong partnership with the schools in which the programs are conducted.

Selective Prevention Programs

Becker, Bull, Schaumberg, Cauble, and Franco (2008) examined a selective prevention program that was implemented with college women in sororities (hence a selected group) among whom the level of risk for eating disorders could fully vary (hence not indicated group). The program was designed to reduce internalization of the thin ideal and so followed the Disease-Specific Pathways Model in targeting a specific risk factor for the development of eating disorders.

Becker and colleague's intervention made use of cognitive dissonance theory. According to the theory (which has been borne out in numerous experimental studies), cognitive dissonance occurs when individuals are confronted with a conflict between what they believe

and how they are acting. To minimize the internal contradiction, their beliefs will shift to become consistent with their overt behaviors, in part because public behaviors cannot be easily undone. As the theory was applied in Becker et al.'s study, participants identified the thin ideal and then engaged in public behaviors that actively opposed this ideal.

For the intervention, Becker and colleagues trained members of the Tri-Delta sorority to be peer leaders who led groups of their fellow sorority members through two sessions with accompanying homework. The total active intervention time was approximately four hours, not including homework exercises. During the first session, participants identified costs of pursuing the thin ideal, discussed the unattainability of the thin ideal and who benefits from it (the fashion and diet industries, not them), and were assigned homework. The homework consisted of a mirror exposure exercise in which they identified positive aspects of themselves to discuss with the group, a behavioral exposure exercise in which they did something they would normally avoid because of body image concerns (e.g., exercise in shorts instead of baggy sweatpants), and writing a letter to their younger self providing encouragement and support to resist the thin ideal. In the second session they shared with the group the positive qualities they identified during the mirror exposure exercise and read the letters they wrote out loud to the group. If they agreed to it, their readings of the letters also were recorded so that they could upload them to YouTube. This last component increased the public nature of their active repudiation of the thin ideal. They also took part in role-playing exercises about how to resist peer pressure to conform to the thin ideal, shared personal experiences with such peer pressure, problem-solved about what they would say if they were in such situations again, created a top-10 list of reasons to resist the thin ideal, and, consistent with the Empowerment-Relational Model, generated ideas for challenging the thin ideal within their communities, including their sorority, campus, town, and society. Finally, they committed to continuing the mirror exposure and behavioral exposure homework assignments and to engaging in self-affirmation exercises in which they would focus on their positive qualities.

To test the efficacy of this program, Becker et al. (2008) randomly assigned sorority sisters to either the cognitive dissonance intervention described above or a media advocacy control program. The media advocacy program included much of the same psychoeducational material as the cognitive dissonance intervention but did not include any component in which participants had to personally act in ways that opposed the thin ideal. Thus while improvements might be observed in the media advocacy condition as a consequence of increased awareness of the origins and harmful consequences of the thin ideal, that condition lacked a key component expected to alter internal beliefs so as to affect subsequent behaviors.

Participants in the cognitive dissonance condition experienced significantly greater decreases in body dissatisfaction, dietary restraint, and bulimic symptoms than participants in the media advocacy condition, and these effects were maintained at eight-month follow-up (Becker et al., 2008). In addition, while media advocacy produced improvements in a subgroup of participants with higher levels of risk for disordered eating at baseline (thus resembling an indicated prevention group), the cognitive dissonance program produced improvements in both women with high and women with low baseline eating disorder risk. This is a key finding for supporting the use of this program as a selective intervention.

Like the Becker et al. (2008) program, selective prevention programs often focus on girls or women, because they are at greater risk than boys or men for developing eating disorders. Selective prevention programs have included participants ranging in age from 10 years (Piran, 1999) to 25 years (Phelps, Sapia, Nathanson, & Nelson, 2000), hence extending to young women who are older than is typical of universal prevention programs. Because several selective prevention programs have included participants past the age at which risk for onset of eating disorders begins, it is not entirely accurate to depict these programs as focusing just on participants among whom no problems have yet emerged. Nevertheless, selective prevention programs do not target participants specifically on the basis of having problems with body image or disordered eating attitudes or behaviors. Students with eating disorders were allowed to participate in the Becker et al. (2008) study but were excluded from data analyses of the efficacy of the intervention. Thus this still constituted a prevention rather than treatment study.

Several controlled selective prevention studies have found improvements in knowledge and attitudes associated with intervention compared with a control condition (Moreno & Thelen, 1993; Steiner-Adair et al., 2002; Stewart, Carter, Drinkwater, Hainsworth, & Fairburn, 2001; Withers, Twigg, Wertheim, & Paxton, 2002), and these improvements have been maintained when assessed at follow-up (Moreno & Thelen, 1993; Steiner-Adair et al., 2002). As with universal prevention programs, only some selective prevention programs have produced improvements in behavior (Austin, 2000; Levine & Smolak, 2001; Littleton & Ollendick, 2003), and some programs have failed to find any improvements in the intervention group compared with controls (Baranowski & Hetherington, 2001; Martz & Bazzini, 1999; Martz, Graves, & Sturgis, 1997; McEvey & Davis, 2002). Frequently, this failure was due to the presence of improvements in both the intervention and control groups (Baranowski & Hetherington, 2001; McEvey & Davis, 2002).

Stice, Shaw, and Marti (2007) completed a meta-analysis of eating disorder prevention programs and found that although over half of programs successfully reduced eating disorder risk factors, less than a third demonstrated efficacy in reducing future disordered eating. Selective programs were more successful than universal programs, and programs that focused on women were more successful than programs that included both women and men. Finally, programs that included interactive components, such as role-playing challenges to the thin ideal from peers, were more likely to achieve reductions in risk, as were those that consisted of more than one session or included participants older than 15 years. These findings underscore the numerous components of Becker et al.'s (2008) intervention that likely contributed to its overall success.

One factor that may facilitate the success of selective programs focusing on females over the age of 15 is that these are precisely the indviduals who will have higher body image concerns at baseline. Statistical tests are sensitive to differences or changes, and there is more room for individuals who begin with elevated concerns about weight or shape to show a significant decrease in these concerns. Moreover, to the extent that individuals with elevated weight and shape concerns are at greatest risk for developing eating disorders, they represent a prime target group for efforts to reduce eating disorder risk. The next section reviews programs that adopt this exact approach.

Indicated Prevention Programs

Indicated prevention programs are specifically designed for people who already have problems with body image or eating. Participants are often college-age women who report significant body image disturbance. These programs often exclude individuals with AN or BN, because one cannot prevent an event after its onset; however, some participants may be diagnosable with OSFEDs (see Chapter 1). Among prevention programs, therefore, indicated prevention programs bear the greatest resemblance to treatment, and several of these programs include techniques originally developed in treatment studies.

Celio and colleagues (2000) investigated two indicated prevention programs that target women on the basis of body image disturbance. College women who reported high levels of body dissatisfaction took part in one of the two programs, Student Bodies and Body Traps, or in a wait-list control condition. Women with AN or BN were excluded from the study. Student Bodies is an Internet-based program that combines psychoeducation with cognitive–behavioral therapy exercises (this is an example of how preventions can include treatment techniques). Body Traps is a classroom intervention with a focus on psychoeducation.

The basic designs of the two programs are as follows:

Student Bodies
Three face-to-face sessions (over an eight-week period)
Weeks 1 and 2: Orientation to program
Week 6: Group discussion of body image dissatisfaction
Academic readings (one or two articles per week)
Written reflections in response to academic readings (1–2 pages)
Online readings on body image, exercise, nutrition, and eating disorders; cognitive–behavioral exercises
Online body image journal (at least one entry per week [suggested])
Discussion group messages (at least two messages per week, one in response to a group member)

Body Traps
Eight two-hour class meetings (over an eight-week period)
Lecture or guest speaker
Group discussion
Academic readings (one or two articles per week)
Written reflections in response to academic readings (1–2 pages)

Thus in both interventions, participants are asked to complete weekly readings and a weekly reflection paper and to take part in group discussions (which occur online for Student Bodies). Both programs cover four topics: eating disorders, healthy weight regulation, nutrition, and exercise, divided into eight weekly programs: body image, eating disorders, weight regulation, diet check, general nutrition, food item analysis, exercise, and diary.

The primary differences between the programs are that Student Bodies takes place online and Body Traps in a classroom and that Student Bodies includes weekly cognitive–behavioral exercises (e.g., observing real women in public places and comparing them with magazine models) while Body Traps remains psychoeducational. In addition, Student Bodies encourages communication among participants in between online group discussions. Celio and colleagues hypothesized that both programs would reduce body dissatisfaction and disordered eating compared with the control condition and that those improvements would be maintained at follow-up.

Participants in Student Bodies reported fewer weight or shape concerns at the end of the program than controls did, but no differences were found between controls and participants in Body Traps. At six-month follow-up, participants in Student Bodies continued to report fewer weight or shape concerns, fewer eating concerns, and less dietary restraint than controls did. Again, no differences were found between controls and participants in Body Traps (Celio et al., 2000). The Student Bodies results are particularly notable because differences attributable to the prevention program were maintained at follow-up and included a measure of behavior—dietary restraint. Subsequent studies with the Student Bodies program replicated the reductions in weight concerns as well as finding decreases in a global measure of disordered eating, drive for thinness, and bulimic symptoms compared with wait-list controls; moreover, improvements in weight concerns, disordered eating, and drive for thinness were maintained at 12-month follow-up (C. B. Taylor et al., 2006).

Results of indicated prevention studies have generally been promising. Participants in indicated prevention programs have reported improvements in knowledge and attitudes following intervention (Celio et al., 2000; Franko, 1998; Stice, Chase, Stormer, & Appel, 2001; Stice, Trost, & Chase, 2003; Winzelberg et al., 1998; Zabinski et al., 2001). These improvements were greater than those reported by controls and were maintained at follow-up (Franko, 1998; Taylor et al., 2008; Winzelberg et al., 1998). In addition, as was seen with Student Bodies (Celio et al., 2000; Taylor et al., 2006), there is evidence of improved behavior compared with controls at follow-up.

Conclusion

Across outcomes, both indicated prevention programs (Stice & Shaw, 2004) and selective prevention programs (Stice et al., 2007) produce better effects than universal programs. This difference may reflect treatment efficacy rather than prevention efficacy. Rosenvinge and Borresen (1999) have argued that the limited results for universal programs reflect the limited understanding of factors that increase the risk of eating disorders. This challenge calls for more research into risk factors for eating disorders as well as for the creation of programs that do not rely on altering specific risk factors for their influence.

A second challenge in work on eating disorder prevention is that some improvements seen immediately after intervention dissipate over the course of follow-up (Becker et al., 2008; J. Pearson, Goldklang, & Striegel-Moore, 2002; Stewart et al., 2001; Stice, Trost, & Chase., 2003; Taylor et al., 2006; Withers et al., 2002). The greatest constraint on the scope of prevention is the limited time available to work with children. As described above, the

durations of programs have generally been quite limited, with some programs providing as few as three (Buddeberg-Fischer & Reed, 2001; Rocco, Ciano, & Balestrieri, 2001) or five sessions (Baranowski & Hetherington, 2001; Paxton, 1996; Wade et al., 2003). Indeed, two prevention studies used a videotape as the intervention (Moreno & Thelen, 1993; Withers et al., 2002). Given that girls reported watching 3.2 hours of television a day in one study (Tiggemann & Pickering, 1996), it becomes clear why one 22-minute video would not produce a lasting influence on attitudes after one month (Withers et al., 2002), much less throughout the period of risk for developing an eating disorder. Across programs, number of sessions is associated with program effectiveness (Stice & Shaw, 2004). Thus modest interventions of limited duration may produce modest changes of limited duration. The solution is to increase the resources for implementing prevention programs.

A third challenge in prevention work has come from findings of similar improvements in experimental and control groups (Baranowski & Hetherington, 2001; Stice, Trost, & Chase, 2003). Although it is good to find improvement, it is difficult to interpret its meaning when no group differences are found. Improvement could reflect nonspecific benefits of encouraging healthy eating. It could reflect a phenomenon known as *regression to the mean*, in which individuals with more extreme scores on a measure of disordered eating would be expected to score closer to the mean when retested. It could reflect that individuals with problems improve just because "time heals all wounds." Alternatively, improvement among all participants in a prevention study may reflect important self-selection biases. For example, only individuals who are motivated to improve would likely volunteer for studies of indicated prevention programs, and this motivation might account for improvements over time regardless of whether participants were placed in the intervention or the control group.

The prevention programs discussed above, and even the paradigms within which prevention programs are conceived, have clearly focused primarily on sociocultural factors described in Chapter 5 (media images, body image, and dieting) and have made use of a peer context to exert positive social influences in the immediate environment. Prevention programs have focused to a lesser extent on some of the general risk factors described in Chapter 4. Thus one way of thinking about prevention versus treatment is that prevention tends to focus on social factors and to a lesser extent on some psychological factors, while treatment spans the full range of social, psychological, and biological factors. The emphasis on social factors in prevention programs reflects the public health focus of prevention as a discipline. However, this emphasis rests on two implicit premises: first, that social factors have the greatest influence on the development of eating disorders, and second, that social factors are the easiest to change. Perhaps another reason for the limited success of prevention programs thus far is an unmet need to critically evaluate these premises.

A roundtable discussion of experts convened by the National Institute of Mental Health generated the following recommendations for improving future programs for preventing eating disorders:

1. Develop common definitions of symptoms, syndromes, risk factors, and outcomes to better assess progress in epidemiology and prevention trials.
2. Encourage the integration of basic social science research in prevention approaches.

3. Encourage research on neural mechanisms of eating disorders at the animal level. Foster cross-discipline interactions among animal experimentalists, clinicians, and other researchers in the field.
4. Develop guidelines for assessing the scientific merit of eating disorders prevention trials.
5. Develop approaches to assess and minimize iatrogenic effects (for example, producing the disordered behaviors the program attempts to prevent).
6. Encourage research in biology, personality traits, family and social groups, and societal norms and values, all of which influence the development of eating disorders.
7. Increase awareness that eating disorders are a public health problem and that prevention efforts are warranted.
8. Adopt an approach that considers the public health impact of these disorders.

Pearson et al., 2002; pp. 237–238 *reprinted with permission of John Wiley & Sons, Inc.* Copyright 2002 by Wiley Periodicals, Inc.

These recommendations would expand prevention paradigms to encompass the full range of etiological risk factors for eating disorders, from neuronal communication to cultural constructions of gender. They also reflect some of the impediments to developing efficacious prevention programs, which range from deficits in understanding the exact causes of eating disorders to societal stigmatization of eating disorders as problems that people choose to have (Crisp, Gelder, Rix, Meltzer, & Rowlands, 2000; Stewart, Keel, & Schiavo, 2006). Such ill-informed beliefs reduce support for eating disorder prevention and need to be debunked to enhance the successes of this important endeavor.

Key Terms

Disease Prevention Paradigm
Disease-Specific Pathways Model
Empowerment-Relational Model
Health Promotion Paradigm
Indicated prevention program
Nonspecific Vulnerability-Stressor Model
Selective prevention program
Universal prevention program

11

Long-Term Course and Outcome

This chapter examines the long-term course and outcome associated with eating disorders. **Outcome** refers to how well patients are doing at some point after they were initially encountered (e.g., what percentage of patients have recovered from their eating disorder). **Course** refers to the path patients have taken between when they were first encountered and when the outcome is assessed. Course and outcome are related to one another, but they are not the same thing. Two patients could have the same outcome (e.g., recovery) but could have taken very different courses to that outcome.

Although research on long-term course and outcome is largely descriptive, it has the potential to improve the diagnosis, understanding, and treatment of eating disorders. Differences in course and outcome across eating disorders can reveal valid distinctions among disorders (Kendell, 1989). Accurate distinctions among disorders will ultimately help to reveal their etiologies. For example, valid definitions are required for identifying genes that increase the risk of eating disorders (see Chapter 8). Understanding both the natural course of the illness and its course following intervention also is essential to evaluating whether treatment works. Because of differences in the times when specific eating disorders were first identified (ranging from 1873 for AN to 2005 for purging disorder), the availability of information on their outcomes varies significantly.

This chapter reviews and then compares data on the long-term courses and outcomes of AN, BN, and BED. In addition, it provides preliminary findings regarding the outcome of purging disorder. The following outcome domains are covered: mortality, recovery, relapse, crossover, and prognostic factors (i.e., variables that predict outcome).

Anorexia Nervosa

Mortality

Anorexia nervosa has been associated with one of the highest risks of premature death among psychiatric disorders (E. C. Harris & Barraclough, 1998). Approximately 5% of

patients diagnosed with AN have died by follow-up across outcome studies (Steinhausen, 2002; Sullivan, 1995). To understand how much the risk of death is increased by AN, studies use a **standardized mortality ratio** (SMR). This is the ratio of the observed number of deaths in a specific group (e.g., patients with AN) to the expected number of deaths in a matched population; values significantly greater than 1.0 reflect an elevated risk of death. Standardized mortality ratios reported for AN have ranged from 4.4 to 12.0 (Franko et al., 2013; Keel et al., 2003; Löwe et al., 2001; Patton, 1988). A recent meta-analysis of studies examining mortality rates in AN estimated that SMR was 5.86 across studies (Arcelus, Mitchell, Wales, & Nielsen, 2011). One study found that elevated death in AN was observed during the first 10 years of follow-up and then decreased (Franko et al., 2013).

In addition to deaths linked to the physical consequences of starvation (Keel et al., 2003), AN is associated with increased risk of death by suicide. One in five patients with AN commits suicide (Arcelus et al., 2011). A recent meta-analysis of suicide in AN provided an SMR of 31 (Preti, Rocchi, Sisti, Camboni, & Miotto, 2011), meaning that individuals with AN were 31 times more likely to die by suicide than were other individuals matched on gender, age, and socioeconomic status. An examination of the ways AN patients have committed suicide showed the use of highly lethal methods that would have been fatal regardless of the attempter's physical health (Holm-Denoma et al., 2008).

Examining the literature on outcome suggests that causes of death in AN have shifted away from factors related to starvation and toward suicide. This shift may reflect several trends. For example, the diagnostic criteria for AN shifted from requiring a loss of 25% of prior body weight in the *DSM-III* to weight 15% below that expected in the *DSM-III-R* (similar to the *DSM-IV* threshold) to current guidelines that would include individuals with a BMI less than 18.5 kg/m^2 in the *DSM-5*. Reflecting this change, epidemiological data have demonstrated that the weights of patients seeking treatment for AN have increased over time (Eagles, Johnston, Hunter, Lobban, & Millar, 1995). Thus a smaller proportion of AN patients may now be at risk for death by starvation. The shift in causes of death also may reflect that many patients now receive treatment earlier in the course of the illness. Finally, there may have been improvements in techniques employed to refeed undernourished patients.

Predictors of a fatal outcome in AN include low weight (Franko et al., 2013; W. Herzog, Deter, Fiehn, & Petzold, 1997; Patton, 1988), poor psychosocial functioning (Engel, Wittern, Hentze, & Meyer, 1989; Franko et al., 2013; Keel et al., 2003), longer duration of follow-up (Keel et al., 2003; Steinhausen, 2002), and severity of alcohol use disorders (Franko et al., 2013; Keel et al., 2003). Low weight likely contributes to mortality because of the severe medical consequences of starvation (see Chapter 8). Poor psychosocial functioning may contribute to the risk of death by suicide. Longer duration of follow-up likely reflects the cumulative deleterious effects of starvation on physical and mental health. Alcohol use disorders may contribute to suicide risk, and the risk of death by alcohol poisoning may be elevated in individuals with low weight and excessive alcohol consumption (Keel et al., 2003). Factors associated with suicide attempts in AN include purging behaviors, depression, substance abuse, and a history of physical or sexual abuse (Franko & Keel, 2006).

Recovery

Recovery tends to be a prolonged process that takes several years for many patients with AN (D. B. Herzog et al., 1999; Nilsson & Hagglof, 2005; Steinhausen, 2002). At 12-month follow-up, only 9.1% of patients have been found to be in remission (Milos, Spindler, Schnyder, & Fairburn, 2005). However, at two to five years follow-up, remission from AN across studies is approximately 45% (Agras, Crow, Mitchell, Halmi, & Bryson, 2009; Clausen, 2008; Fichter & Quadflieg, 2007). At long-term follow-up, approximately 53% of individuals from community-based samples have achieved remission (Wentz, Gillberg, Anckarsäter, Gillberg, & Råstam, 2009), while estimates among treatment-seeking samples are higher, with approximately two-thirds of individuals achieving remission (calculated as weighted average of 69% from the results of Eisler, Simic, Russell, & Dare, 2007; Fichter, Quadflieg, & Hedlund, 2006; Halvorsen, Andersen, & Heyerdahl, 2004; Nilsson & Hagglof, 2005). Collapsing recovery rates across studies, Steinhausen (2002) reported that approximately 46% of patients recover, 33% improve but remain symptomatic, and 20% remain chronically ill.

Relapse

Relapse occurs when a person has achieved remission from a disorder and the disorder returns. Morgan and Russell (1975) reported that 51% of patients hospitalized for AN required readmission over the course of follow-up. D. B. Herzog and colleagues (1999) reported that 40% of women with AN who achieved full recovery later relapsed. Finally, Strober, Freeman, and Morrell (1997) reported that approximately 30% of women who achieved weight recovery during hospitalization relapsed after discharge, with higher relapse rates over follow-up among women who were considered only partially recovered (9.8%) compared to those who were fully recovered (0%). The latter data indicate that with a more stringent definition of recovery (or remission), one finds a decreased risk of relapse.

Overall, over a third of women who achieve weight recovery will later lose weight and relapse into AN. Continued weight and shape concerns significantly increase the risk of relapse (Keel, Dorer, Franko, Jackson, & Herzog, 2005). A common pattern among patients with AN is to experience improvement in weight (a sign of recovery) as a consequence of the development of binge-eating episodes—a sign of crossover, discussed next.

Crossover

Crossover is the transition from having one eating disorder to having another eating disorder. Thus as an outcome, it is distinct from both recovery and relapse. Most women with ANR develop symptoms of binge eating and purging over time (Eddy et al., 2002; Eddy et al., 2008). This finding is consistent with the hypothesis that dietary restriction increases the susceptibility to binge eat (Polivy & Herman, 1985). If binge eating and purging continue at low weight, then the person suffers from ANBP. If binge eating results in weight gain and the binge–purge behaviors continue at normal weight, then the person has crossed over from AN to BN. Between 2% and 34% of individuals with AN transition to a different eating disorder diagnosis (Castellini et al., 2011; Fichter & Quadflieg, 2007; Fichter et al., 2006; Milos et al., 2005; Nilsson & Hagglof, 2005).

Crossover from AN to BN is more common for patients initially diagnosed with ANBP (Eddy et al., 2008) and is more common with longer durations of follow-up (Agras et al., 2009; Fichter & Quadflieg, 2007). Additional predictors of crossover from AN to BN include unipolar depression, substance abuse, and the absence of an OCD diagnosis (Castellini et al., 2011). Anorexia nervosa is 4 to 30 times more likely to remain stable than to cross over to a diagnosis of BN (Agras et al., 2009; Ben-Tovim et al., 2001; Fichter & Quadflieg, 2007; Milos et al., 2005). In the studies reviewed for this book, no cases of crossover were observed from AN to BED (Agras et al., 2009; Castellini et al., 2011; Fichter & Quadflieg, 2007).

Thus AN does exhibit some diagnostic crossover to BN but not to BED. However, stability is more common for AN than crossover is, highlighting the chronicity of the disorder.

Prognostic Factors

Prognostic factors are features that predict the likely course or outcome of a disorder (also referred to as *prognosis*). Figure 11.1 presents prognostic factors from a comprehensive review of outcome in AN (Steinhausen, 2002). While no prognostic factor has been unambiguously associated with outcome, some tentative conclusions can be drawn from the pattern of findings. For example, 13 studies suggest that an older age of AN onset is associated with a worse prognosis, and 14 studies suggest no significant association. Only 2 studies, however, suggest that an older age of onset is associated with a favorable prognosis. Similarly, 14 studies suggest that a longer duration of symptoms before treatment is associated with a poor prognosis in AN, and 7 studies suggest no significant association. No studies suggest that a longer duration of symptoms is associated with a favorable prognosis. Longer duration of treatment and need for inpatient treatment also predict worse outcome in AN (Keel & Brown, 2010).

Thus the weight of the evidence suggests that an older age of onset and a longer duration of illness prior to treatment and greater severity of symptoms are associated with lower recovery rates. These findings may indicate that an early age of onset is predictive of a better course and outcome only if the delay before seeking treatment is short.

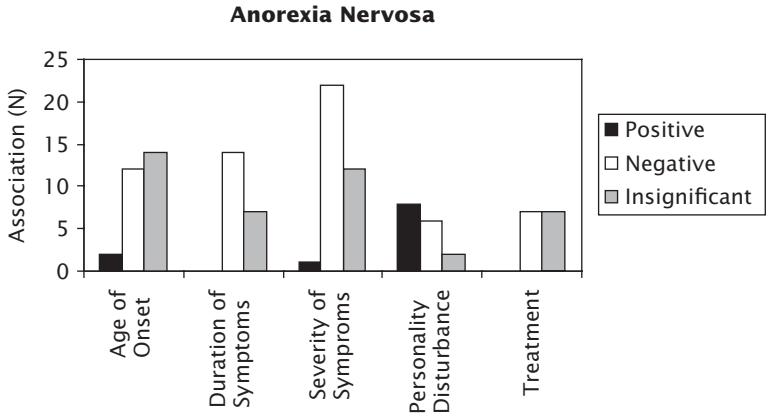


FIGURE 11.1 Prognostic indicators of a favorable course or outcome in AN. *Note:* Created by the author with information from: Steinhausen, H. C. (2002). The outcome of anorexia nervosa in the 20th century. *American Journal of Psychiatry*, 159, 1284–1293.

Bulimia Nervosa

Mortality

Until recently, it was believed that BN was not associated with an increased risk of death (Keel & Mitchell, 1997; Keel et al., 2003), in stark contrast to AN. However, Crow and colleagues (2009) established that risk of fatal outcome was significantly elevated in BN (SMR = 1.57) through a death record search of 906 patients with the disorder—the largest study of mortality from eating disorders in the literature. The proportion of deaths observed in this sample (3.9%) was similar to that observed in a subsequent study (3.3%; Franko et al., 2013); however, the smaller sample size of 60 BN patients in the latter study reduced its ability to detect whether this figure differed significantly from what would be expected in the absence of BN. In addition to establishing an increased risk of death in BN, Crow et al. (2009) demonstrated a specific increased risk of death by suicide, such that patients with BN were more than six times as likely to die by suicide as individuals matched on age, gender, and socioeconomic status (SMR = 6.51).

Two recent meta-analyses further supported that BN is associated with increased risk of death (SMR = 1.93; Arcelus et al., 2011) and death by suicide (SMR = 7.5; Preti et al., 2011). These meta-analyses have also borne out early observations of differences in mortality between AN and BN (Keel et al., 2003). Although both AN and BN are associated with increased risks of death and death by suicide, these risks are greater in AN than in BN (Arcelus et al., 2011; Preti et al., 2011). Predictors of suicide attempts in BN include a history of substance use disorder, laxative abuse, symptoms of Cluster B personality disorders, and childhood sexual abuse (Favaro, Santonastaso, et al., 2008; Franko et al., 2004).

Recovery

Prospective longitudinal studies of the course of BN suggest that the majority of patients recover from their eating disorder at some point during follow-up (Keel & Brown, 2010). Combining results across outcome studies with follow-up durations of approximately five years, Keel and Mitchell (1997) reported that approximately 50% of individuals with BN recover and maintain their recovery, 30% are improved but maintain partial syndromes, and 20% continue to meet full criteria for BN. Longer follow-up studies suggest that approximately 65–70% of BN patients achieve remission (Fichter & Quadflieg, 2004; Grilo et al., 2007; Keel, Gravener, Joiner, & Haedt, 2010; Keski-Rahkonen et al., 2009), while 10% follow a chronic course (Collings & King, 1994; Keel, Mitchell, Miller, Davis, & Crow, 1999; Steinhausen & Weber, 2009). However, as duration of follow-up increases, women become more likely to suffer from an OSFED rather than meeting full criteria for BN (Keel & Brown, 2010).

Case Study: Jean

Jean responded well to treatment and was free from symptoms within four months of starting therapy. She was very concerned about relapsing, however, because she knew how easily this could happen. Her therapist encouraged her to join a support group for patients who had recovered from eating disorders. The therapist felt that being in the group might

help Jean appreciate how far she had come and provide her with support when she felt pressure to diet or lose weight. One of the most important achievements of therapy was helping Jean recognize that she was at a healthy weight without dieting. In addition, Jean's therapist encouraged her to reveal her eating problems to her boyfriend so that he could provide support. Jean stayed in the support group for approximately eight months. She formed two close friendships there and continued to socialize with the members. In fact, one of the group members was a bridesmaid at Jean's wedding. Jean found that the older she became, the less she cared about her weight and shape. This was particularly true after she had her first child. She was concerned that she would gain too much weight during her pregnancy and this might trigger her eating disorder, but she didn't. She actually liked the way her body looked during pregnancy, and afterward she was too busy with her baby daughter to worry about her weight. In addition, Jean was committed to setting a good example for her daughter, Eva. She wanted Eva to have high self-esteem and accept her body exactly as it was. Toward this goal, Jean finally confronted her mother when she commented that Eva was eating too much. Jean told her that Eva's appetite and weight were perfectly healthy and normal for a girl of her age, and that such critical comments were neither helpful nor welcome. Ten years after treatment, Jean remained happily married with two children and was working as an office manager. She occasionally ate too much and felt fat when she was under stress. However, her eating disorder never returned.

Relapse

Rates of relapse have ranged from 26% to 55% across follow-up studies of BN (Fairburn et al., 2000; Fichter & Quadflieg, 1997; D. B. Herzog et al., 1999; Keel & Mitchell, 1997; Olmsted, Kaplan, & Rockert, 2005). Similar to what is observed with AN, more stringent definitions of recovery are associated with lower relapse rates (Field et al., 1997; Olmsted, Kaplan, & Rockert, 1994, 2005). This pattern raises the question of whether studies are assessing relapse or simple symptom fluctuation. That is, are patients truly remitting and relapsing or are their symptom levels simply fluctuating around diagnostic thresholds? Despite considerable variations in definitions of remission or recovery (Keel, Mitchell, Davis, Fieselman, & Crow, 2000), relapse rates converge around 30% across most studies (Keel & Brown, 2010; Keel & Mitchell, 1997). Predictors of relapse in BN include poor psychosocial functioning and overconcern with weight or shape (Keel, Dorer, et al., 2005).

Crossover

Keel, Mitchell, Miller, Davis, and Crow (2000) reported that over time women with BN were more likely to continue to suffer from BN than to cross over to either AN or BED. However, approximately 10% of women with BN were found at long-term follow-up to have crossed over to purging disorder, characterized by recurrent purging in the absence of binge episodes at normal weight. The likelihood of suffering from purging disorder at long-term follow-up did not differ significantly from that of suffering from BN. Further, women were significantly more likely to suffer from purging disorder than from BED. Thus when crossover occurs in BN, it is often to purging disorder.

Supporting these findings, other studies indicate that the most common pattern of crossover for BN is migration to purging disorder or to a subthreshold form of BN, with crossover rates ranging from 1.2% to 26% (Castellini et al., 2011; Fichter & Quadflieg, 2007; Milos et al., 2005). It can be challenging to distinguish crossover to a partial syndrome of BN from partial remission from BN; over the course of 7-year follow-up, up to 83% of BN patients met criteria for an OSFED that could also be defined as partial remission from BN (Eddy et al., 2008). Crossover from BN to AN occurs in 1–14% of individuals (Ben-Tovim et al., 2001; Castellini et al., 2011; Eddy et al., 2008; Keel, Dorer, et al., 2005; Keel, Mitchell, Miller, et al., 2000; Milos et al., 2005). The majority of this migration is from BN to ANBP, with crossover from BN to ANR being rare (Eddy et al., 2008). However, BN is 7 to 10 times more likely to remain stable than to cross over to AN (Agras et al., 2009; Ben-Tovim et al., 2001; Fichter & Quadflieg, 2007). Despite the shared symptom of binge eating, crossover from BN to BED appears to be relatively uncommon, ranging from 0.7% to 19% across studies (Bogh, Rokkedal, & Valbak, 2005; Fichter & Quadflieg, 2004, 2007; Keel, Mitchell, Miller, et al., 2000; Stice, Marti, Shaw, & Jaconis, 2009). Indeed, BN was 49 times more likely to remain stable than to cross over to BED in one study (Fichter & Quadflieg, 2007).

Thus while BN does exhibit some crossover, diagnostic stability is more common than crossover to either BED or AN.

Prognostic Factors

Figure 11.2 presents prognostic factors updated from a comprehensive review of outcome in BN (Keel & Mitchell, 1997). Few prognostic factors have been replicated across studies (Steinhausen & Weber, 2009). Similar to the pattern observed for AN, six studies reported that a longer duration of symptoms before presentation was associated with a poor prognosis in BN, and three studies found no significant association. However, no studies reported that a longer duration of symptoms was associated with a favorable prognosis. Similarly, six studies suggested that personality disturbance was associated with a poor prognosis, four studies

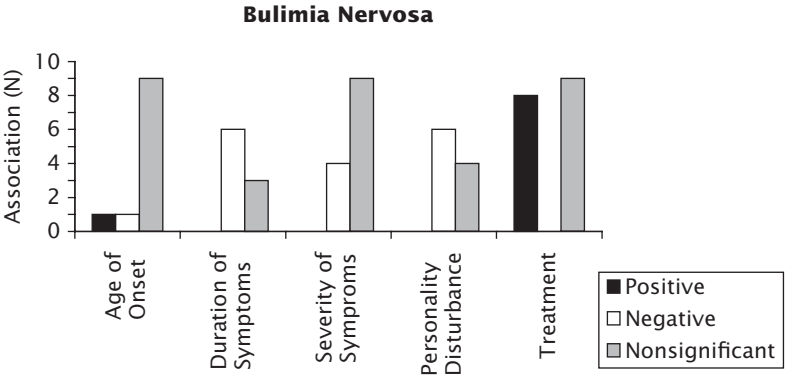


FIGURE 11.2 Prognostic indicators of a favorable course or outcome in BN. *Note:* Created by the author with information from: Keel, P. K., & Mitchell, J. E. (1997). Outcome in bulimia nervosa. *American Journal of Psychiatry*, 154, 313–321.

found no significant association, and no studies reported that personality disturbance was associated with a favorable outcome. These results thus indicate that both longer duration of illness prior to treatment and personality disturbance may decrease the likelihood of a positive response to treatment.

Overall, treatment seems to either improve or have no impact on BN outcome. Keel and Mitchell (1997) found that studies with a shorter duration of follow-up tended to find a favorable effect of treatment on outcome, while studies with a longer duration of follow-up tended to find no significant association between treatment and outcome. Few studies have evaluated the long-term impact of treatment on recovery in BN. At five- to nine-year follow-up, Fairburn et al. (1995) found that both cognitive–behavioral therapy and interpersonal therapy were associated with a better outcome than was behavioral therapy. However, most other authors have failed to find an impact of treatment on long-term outcome in BN (Keel & Brown, 2010).

How can we reconcile the short-term treatment efficacy of cognitive–behavioral therapy and antidepressant medications for BN (Peterson & Mitchell, 1999) with the rather bleak picture of their long-term impact (Keel et al., 1999)? The key may be in evaluating the long-term *course* rather than the long-term *outcome*. If the outcome represents the final destination, then the course represents the path taken to that destination. Two course patterns could account for the distinct pictures of treatment impact provided by short-term and long-term outcome studies. First, increased relapse rates over time in women who responded to treatment could reduce the long-term efficacy of initially successful interventions (see Figure 11.3). Second, eventual recovery among women who did *not* receive treatment (or the best treatment) could obscure the ongoing benefits of treatment (see Figure 11.4), so that treatment could speed recovery without showing an impact on long-term outcome (Keel, Mitchell, Davis, & Crow, 2002). Data support the latter explanation. Miller et al. (2004) reported that women who received cognitive–behavioral therapy achieved recovery sooner and maintained it longer than did women who received only anti-depressant medication or no treatment. Furthermore, relapse rates did not differ across treatment conditions.

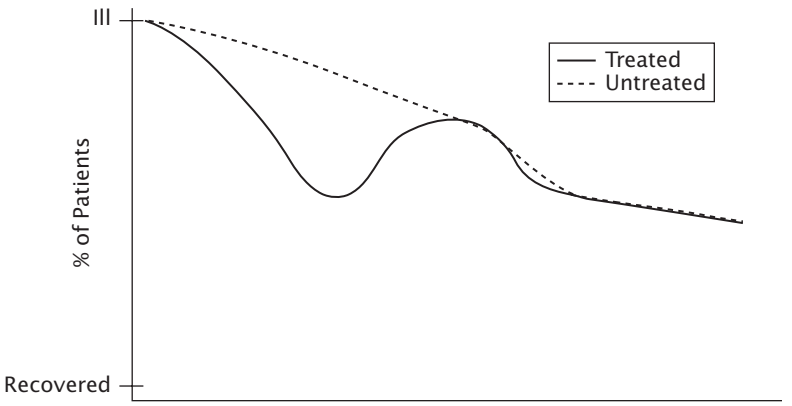


FIGURE 11.3 Increased rates of relapse in a treated group explain the lack of treatment effect at long-term follow-up.

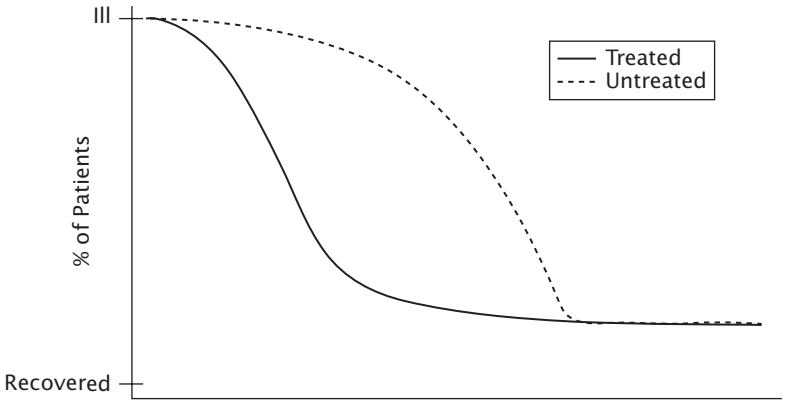


FIGURE 11.4 Eventual recovery in an untreated group explains the lack of treatment effect at long-term follow-up.

Binge-Eating Disorder

Since the introduction of BED in the *DSM-IV* (American Psychiatric Association, 1994), a number of treatment studies of the disorder have been published, and several have provided short-term and intermediate follow-up data (Agras, Telch, Arnow, Eldredge, & Marnell, 1997; Cachelin et al., 1999; Ciano, Rocco, Angarano, Biasin, & Balestrieri, 2002; Devlin, Goldfein, Carino, & Wolk, 2000; Peterson et al., 2001; Ricca et al., 2001). Less is known, however, about the long-term course and outcome of BED. At this point, four studies (Agras et al., 2009; Fairburn et al., 2000; Fichter et al., 1998; Fichter, Quadflieg, & Hedlund, 2008) have characterized the course and outcome for BED at four or more years following first presentation for the disorder, and two of these articles represent successive follow-up assessments of the same cohort (Fichter et al., 1998, 2008). Only two deaths have been reported among patients with BED, both in the same cohort (Fichter et al., 1998, 2008), yielding a crude mortality ratio of 3% and a SMR of 2.29 (which was not statistically significant owing to the small sample size). A meta-analysis of suicide risk in eating disorders found no reports of suicide in patients with BED (Preti et al., 2011).

At 4-year follow-up, Agras et al. (2009) found that 82% of individuals diagnosed with BED achieved remission, a higher likelihood of remission than in AN (57%) or BN (47%). Similarly, over a 5-year follow-up, Fairburn et al. (2000) found that 82% of BED patients had improved or recovered. In contrast to these findings, Fichter et al. (1998) reported remission in 57.4% of patients treated for BED at 6-year follow-up, and this figure increased only to 67% at 12-year follow-up—quite similar to the remission observed for BN at the same interval (Fichter et al., 2008). Fairburn et al. (2000) reported that relapse occurred in 4–10% of individuals recovered from BED across various points of follow-up. Predictors of relapse include being male and reporting a higher frequency of experiencing a loss of control over eating when eating a normal amount of food (Castellini et al., 2011).

Case Study: Jamie

Despite his reservations about joining a group that consisted mostly of women (see Chapter 3), Jamie joined the Monday evening group his counselor had suggested after football season was over. The group followed a self-help guide for eliminating binge eating. When the group discussed triggers for binge-eating episodes, Jamie was surprised to find many of his experiences mirrored in those of other group members. He also found it helpful to learn approaches that had worked and had failed for others. As the weeks passed, he found that he was able to contribute more examples of approaches he had taken during discussions of problem-solving and of identifying alternatives to binge eating. In particular, as the weather got warmer, he started a walking group with a couple of the women in the group. Walking provided him with an alternative approach for dealing with stress, and he enjoyed the camaraderie of spending time with others working toward a shared goal. He realized that his shift from playing team sports to computer games had deprived him of an important social element that he felt brought out the best in him. Moreover, within both the Monday evening group and the walking group, which eventually grew to include eight people from both Jamie's neighborhood and the Monday group, Jamie's natural leadership abilities were valued and he felt accepted, even though his weight remained very high. After 12 weeks, Jamie was completely free of binge-eating episodes and was feeling better about himself than he had in years.

Although a majority of individuals with BED recover, some experience crossover to another eating disorder. The most common pattern of crossover is to a subthreshold form of BED, with rates ranging from 3% to 18% across studies (Fairburn et al., 2000; Fichter & Quadflieg, 2007), or to BN, with rates ranging from 3% to 15% (Castellini et al., 2011; Fairburn et al., 2000; Fichter & Quadflieg, 2007). This pattern suggests that the likelihood of partial remission is similar to the likelihood of progression to more symptoms. Indeed, Fichter and Quadflieg (2007) found that in their sample, crossover to BN was more likely than continuing to meet criteria for BED. Across follow-up studies of BED, none have reported crossover to AN (Castellini et al., 2011; Fairburn et al., 2000; Fichter & Quadflieg, 2007).

Predictors of a worse outcome in BED have included having more interpersonal problems (Hilbert et al., 2007), a history of sexual abuse, and the presence of other mental disorders (Fichter et al., 2008). However, no predictors of outcome have been replicated across studies, and there are no data supporting long-term impact of treatment on outcome in BED (Keel & Brown, 2010).

Purging Disorder

Despite purging disorder's very recent recognition as a distinct syndrome (Keel, Haedt, & Edler, 2005) and its even more recent inclusion in the *DSM-5* as a named form of OSFED (American Psychiatric Association, 2013), outcome data support its clinical significance in terms of chronicity and mortality. Koch, Quadflieg, and Fichter (2014) conducted a nine-year follow-up of 225 patients diagnosed with purging disorder between 1999 and 2005.

Because purging disorder had yet to be recognized at the start of this period, Koch et al. retrospectively applied Keel and Striegel-Moore's (2009) research diagnostic criteria for purging disorder to data in patients' medical records. Overall, 5% of patients had died over follow-up, representing a significantly increased risk of premature death (SMR = 3.9). In a comparison of mortality across eating disorders, Koch, Quadflieg, and Fichter (2013) found a significantly greater risk of death in purging disorder than in BN and no significant difference between purging disorder and AN.

A prospective community-based six-month follow-up of 23 women with purging disorder (Keel, Haedt, & Edler, 2005) found that 13% achieved partial or full remission. Based on combined results from two school-based longitudinal studies (Allen, Byrne, Oddy, & Crosby, 2013; Stice et al., 2009), approximately 75% of individuals with purging disorder achieve remission over follow-up of intermediate length (less than five years). However, studies examining illness status more than five years after presentation suggest lower recovery rates, with less than half of individuals with purging disorder achieving remission (Allen et al., 2013; Koch et al., 2013). The data of Koch et al. (2013) indicated that purging disorder has a more favorable outcome than AN.

Little is known about relapse in purging disorder; however, one longitudinal study of adolescents (Stice et al., 2009) indicated a lower likelihood of relapse in purging disorder (5%) than in BN or BED (41% & 33%, respectively). However, purging disorder was more likely to have a later onset (during the final year of observation) than the other disorders in the study, minimizing the opportunity to observe relapse following remission. Thus additional research is needed to draw more definitive conclusions.

Case Study: Valerie

After Valerie's hospitalization for her suicide attempt, her therapy sessions focused primarily on her feelings of despair and her belief that others would be better off without her in their lives. Valerie was overcome by a sense of being a burden to others, particularly her sister, who had taken time off from work during Valerie's hospitalization. The most the therapist could achieve in the sessions was obtaining Valerie's promise that she would not try to kill herself without first calling or reaching out to someone for help; very little time was spent focusing on her purging behaviors. As spring approached, Valerie's mood seemed to improve, and she seemed more focused in sessions. Her therapist began suggesting that perhaps they could return to their work on reducing her purging. Valerie agreed with her therapist that if she could stop purging she would feel like more of a worthwhile person and that her repeated use of vomiting and laxatives seemed to be a way of punishing her own body for existing. However, while the therapist was at an eating disorder conference in the United States, she received an email. Valerie was dead. According to the autopsy report, she had been driving under the influence of alcohol and had crashed her car. It was unclear whether the crash was an accident or deliberate. The absence of skid marks suggested that Valerie had not used the brakes to try to save herself, but her blood alcohol level was so high that it was unclear if she was conscious at the time of her death.

The tragic end to Valerie's life resembles that of many patients with eating disorders who die by suicide as well as of other individuals who commit suicide. According to the

interpersonal theory of suicide (Joiner, 2005; Van Orden et al., 2010), individuals who have high levels of hopelessness, feel strongly that they are a burden on others, and have acquired the capability to commit suicide through a series of provocative and painful experiences are at highest risk for acting on thoughts of ending their own lives. Purging is a risk factor for suicide attempts across eating disorders, supporting the concept that as individuals engage in repeated acts of self-harm, whether through purging, cutting, or other means, they gradually lower the barrier to committing the ultimate act of self-harm.

Comparison of *DSM-5* Eating Disorders

Anorexia Nervosa and Bulimia Nervosa

Both within studies and across studies, AN is associated with a significantly increased risk of premature death compared with BN or BED. Suicide attempts are more common in AN than in BN (Franko et al., 2004). Within studies, AN also has been associated with lower rates of recovery than BN.

Relapse rates for AN and BN are similar, but the relationship between relapse and chronicity likely differs between the disorders. Because a high percentage of women with BN recover at some point during follow-up, the chronic course of the disorder appears to be characterized by a pattern of remission and relapse. The smaller percentage of women who recover from AN means that its chronic course is marked by a more steady presence of symptoms. This difference may reflect the nature of the core features of these syndromes. For AN, the core symptom is the absence of a behavior: eating. For BN, the core symptoms are two behaviors: binge eating and purging. Thus to develop AN, an individual must avoid eating consistently. In contrast, a woman can develop BN by having relatively normal eating behaviors interrupted by episodes of abnormal eating.

Patterns of crossover also differ markedly between AN and BN. Approximately 10–50% of women with AN cross over to having BN, and approximately 30% of women with BN report a prior history of AN. Conversely, crossover from BN to AN is relatively rare. This pattern likely reflects consequences of the core features of each disorder. Intense dietary restriction and weight loss likely increase the risk of developing binge-eating episodes. Conversely, large binge-eating episodes likely protect against low weight. Indeed, comparisons of the subtypes of AN suggest that engaging in binge eating and purging is associated with higher weight.

AN and BN appear to have different prognostic indicators. However, this difference is likely a function of the difference in the numbers and sizes of outcome studies for the two disorders. An early age of onset has been associated with an improved prognosis in AN but has not been associated with the prognosis in BN. Currently, the most effective treatments for AN involve family interventions (see Chapter 9). These are likely to be more useful for young adolescent patients who live with their parents. Thus little is known about the effective treatment of older patients with AN. In contrast, BN often develops during late adolescence and young adulthood. Successful treatments include cognitive–behavioral therapy and antidepressant medications, which are likely to work well across the developmental range from late adolescence to middle age.

Longer duration of symptoms at intake predicts a worse outcome for both AN and BN. Severity of symptoms appears to be associated with the prognosis for AN but not BN. This difference may reflect the fact that chronicity in AN is associated with a steady course, whereas chronicity in BN is associated with considerable symptom fluctuation. A single measure of symptom severity in BN would then be an unreliable indicator, limiting its ability to predict BN outcome. By contrast, the overall duration of illness captures the length of time a patient has struggled with an eating disorder regardless of course fluctuations.

Personality disturbances have been associated with a worse prognosis for both AN and BN. However, the nature of the relevant personality disturbances differs between the two disorders. Obsessive–compulsive personality features have been associated with a worse prognosis in AN (Steinhausen, 2002). Conversely, borderline personality features and marked impulsiveness have been associated with a worse prognosis in BN (Keel & Brown, 2010; Keel & Mitchell, 1997). These findings likely reflect differences in the core symptoms of these disorders. Anorexia nervosa is marked by rigid adherence to dietary restriction, BN by recurrent loss of control over eating. As described above, these differences are also reflected in the disease courses among those individuals who remain chronically ill.

The impact of treatment on disorder outcome differs between AN and BN. Although treatment has not been significantly associated with long-term outcome in BN (Keel & Brown, 2010; Keel et al., 1999), it has been associated with a differential course (Miller et al., 2004). Specifically, treatments that have demonstrated efficacy for BN appear to speed recovery. In AN, few treatments are associated with long-term outcome. There is evidence that a longer duration of inpatient treatment is associated with a worse outcome in AN (Steinhausen, 2002). This association likely reflects the effect of increased disorder severity on treatment use (Keel, Dorer, et al., 2002). One study found that family-based treatment was superior to individual treatment for AN at five-year follow-up (see Chapter 9). Controlled studies of new treatments have been initiated for AN, but more time is needed to evaluate the long-term impact of these treatments on AN course and outcome.

Bulimia Nervosa and Binge-Eating Disorder

In contrast to BN, the risk of a fatal outcome may not be elevated in BED. However, obesity in BED may contribute to increased mortality with increasing duration of follow-up. Comparison of recovery in BN and BED has produced inconsistent results. Fairburn and colleagues (2000) found that rates of recovery were higher for BED than for BN, but Fichter and colleagues (1998, 2008) failed to find any differences in outcome. This difference may reflect how investigators recruited their participants. Fairburn et al. studied women recruited from the community, while Fichter et al. recruited participants from an inpatient treatment program. Arguably, women receiving inpatient treatment for BED are likely to have a more severe illness than women with BED in the community. Thus selection factors that lead patients to seek inpatient treatment may explain the greater similarity of outcomes between BN and BED in Fichter et al.'s study. Binge-eating disorder shows a higher likelihood of crossing over to BN than of remaining stable, whereas BN is significantly more likely to remain stable than to cross over to BED. No crossover has been observed between AN and BED.

Taken together, studies of outcome and course of eating disorders suggest that these disorders may reside on a continuum of severity, with AN representing a particularly severe expression of eating pathology, BN demonstrating intermediate but highly variable severity, and BED appearing to be least severe disorder in terms of chronicity and mortality. However, conclusions about the severity or chronicity of BED are tentative, given the limited data on its intermediate and long-term outcome.

Conclusion

As noted at the beginning of the chapter, having data on long-term course and outcome could improve the diagnosis, understanding, and treatment of eating disorders. Course and outcome data support the predictive validity of distinguishing among AN, BN, and BED and formed the primary basis on which the *DSM-5* eating disorders workgroup identified them as distinct disorders of eating. These disorders appear to be associated with different rates of mortality, recovery, and crossover as well as with distinct sets of prognostic indicators (including different treatment responses).

Fewer studies have assessed the long-term outcome of BED than of AN and BN, and even fewer have examined the natural history of BED at long-term follow-up. Indeed, almost all longitudinal studies of eating disorders are based on treatment-seeking samples, even though the majority of women with eating disorders may not seek treatment (see Chapter 9). Using treatment-seeking samples introduces certain biases into study results. For example, follow-up studies based on treatment-seeking samples may present more dire descriptions of outcome than would be obtained with community-based samples. Alternatively, treatment may increase the rate of recovery (Fairburn et al., 1995) or reduce the time to recovery (Keel, Mitchell, Davis, & Crow, 2002; Miller et al., 2004).

Factors that are associated with the maintenance of eating disorder symptoms may play a role in the initiation of eating disorders. Evidence that obsessive–compulsive personality features are associated with prognosis in AN while impulsive and borderline features are associated with prognosis in BN indicates an opportunity to discover risk factors that may be specific to each disorder. Improved understanding of the etiology of eating disorders holds promise for their prevention. In addition, improved understanding of factors that maintain illness is crucial for the development of effective treatments.

Key Terms

Course
Crossover
Outcome
Prognostic
Relapse
Standardized mortality ratio

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Conclusion

Eating disorders are a serious form of psychopathology. They are associated with great distress, impairment in life functioning, and significant medical risk. Among the many forms of mental disorders in the *DSM-5* (American Psychiatric Association, 2013) and *ICD-10* (World Health Organization, 1998), few demonstrate such a clear influence of culture on the prevalence and presentation of symptoms. Yet eating disorders are not restricted to modern Western culture. Similarly, few disorders show the imbalanced gender ratio presented by eating disorders. Yet eating disorders are not restricted to women. Epidemiological patterns provide some hints to the etiology of these disorders without fully explaining why one individual develops an eating disorder and another does not. Moreover, eating disorders represent a fascinating example of social, psychological, and biological factors conspiring to produce a specific kind of mental illness. This chapter summarizes information presented in this book in the context of information originally presented in the case studies and briefly examines current debates within the field. The chapter ends with a consideration of potential directions for future research endeavors.

Case Studies Revisited

Before considering how the case studies relate to the state of knowledge in eating disorders, it is worthwhile to remind ourselves of their initial descriptions. Reading these case studies a second time, at the end of this book, provides an opportunity to notice details and examples that reinforce material presented in Chapters 3 through 11 that may have been missed upon first reading.

Emily, a Case of Anorexia Nervosa

Emily, a 19-year-old sophomore at a large state university, bluntly stated that she had been "forced into treatment" by her school. Emily made it clear that she thoroughly resented the university's interference in her private life, since she had top grades in all classes and was clearly fine. Emily saw no reason to be in therapy or any sort of treatment. At 5 feet 10 inches tall and 109 pounds, Emily had a BMI of 15.7 kg/m^2, well below the 5th percentile for someone the same age and in the severe range for an adult with AN. Emily's college roommates were extremely worried, because she had needed to be taken to the emergency room after fainting in the dining hall. When asked about the incident, Emily said that she had lost track of the time, hadn't eaten all day, and had become lightheaded after an afternoon run. However, Emily asserted that this was very unusual behavior and that she always had a high-energy snack before exercising. In fact, Emily said she always carried food because of a tendency to be hypoglycemic and, in contrast to what people thought, was eating all the time.

When asked what she would eat during a given day, Emily described having cereal for breakfast, snacking throughout the day, having a salad for lunch, snacking throughout the afternoon, and then eating a full dinner. On further questioning, Emily reported eating one packet of plain instant oatmeal made with spring water for breakfast in her room. The snacks consisted of celery sticks, carrot sticks, or sugar-free gum. Lunch was a "huge" plate of salad greens without dressing from the dining hall's salad bar. Dinner was the only meal that varied from one day to the next. Emily might eat a skinless chicken breast with half of a baked potato and a green vegetable. Occasionally, she ate half a cup of pasta with tomato sauce and vegetables added from the salad bar. On days when the dining hall served nothing she liked, Emily ate two slices of bread with cottage cheese spread over each slice and tomatoes on top—she likened this to pizza—along with a large salad. Based on this self-report, Emily's average daily caloric intake was estimated at less than 500 kilocalories (kcal). In comparison, average daily energy needs for a woman of the same age would fall between 1,500 and 1,800 kcal, depending on daily activity level.

Emily stated that she didn't eat red meat because she didn't like the idea of eating cute, furry animals. In fact, Emily didn't care for meat as a food group but made sure to always include protein in her diet because it was important for muscle development. Emily considered muscle development important because of feelings of constantly struggling with a "lopsided" body. Emily described herself as having a "classic pear shape," with shoulders and arms that were too thin and sticklike, and rotund hips, thighs, and buttocks. To improve her muscle definition, Emily exercised rigorously, running every afternoon between classes and dinner. On weekends, Emily added weight training. She had read that metabolism increased both during and after exercise and felt that this pattern increased the probability that dinner would fuel her body rather than being stored as fat. Emily was terrified of becoming fat.

Emily's concerns about weight had emerged during middle school. Always tall for her age, Emily felt like an "amazon" after entering puberty, because she towered over all of her classmates, including the boys on the football team. In an effort to fit in, she began to diet and lose weight. Emily's mother attributed the weight change to a loss of "baby fat," and friends expressed admiration of her self-control. Emily once was approached in the shopping

mall, asked if she had ever considered becoming a model, and given the card of a modeling agency. Although flattered, Emily did not pursue this opportunity because she planned to go to a good college, then go to law school, and eventually become a judge. A career in modeling, Emily felt, would be a waste of her intellect because it required people to focus on superficial things like appearance.

Jean, a Case of Bulimia Nervosa

Jean was a 27-year-old secretary who lived with her boyfriend of two years. She was 5 feet 4 inches tall and weighed 138 pounds. Jean came in for treatment because of a return of eating problems that she thought had ended in college. In college, Jean had experienced binge-eating episodes and had engaged in self-induced vomiting. Jean spent a great deal of time trying to hide these behaviors from roommates and from family when home during breaks. However, her roommates confronted Jean after a particularly bad episode in which she had gone to the bathroom to vomit four times within a two-hour period.

Treatment had allowed Jean to stop binge eating and purging on a regular basis. Jean continued to have occasional slips—times when she felt she had eaten too much and purged to avoid weight gain. However, these occurred rarely, and sometimes Jean prevented herself from vomiting after "eating too much." About a year and a half ago, Jean had noticed that she was gaining weight and could no longer fit into the same-size jeans she had worn since high school. Jean couldn't bring herself to buy larger jeans because she couldn't feel good about herself unless she fit into that specific size. She decided to diet and go to the gym more regularly. At first, the new fitness routine worked, and Jean lost approximately seven pounds. At a weight of 125 pounds, Jean felt great and was more likely to want to go out with friends and to flirt with and get attention from men.

However, when Jean and her boyfriend started living together, she had a hard time resisting the tempting foods he kept in the kitchen. While living alone, Jean had never had cookies, ice cream, or potato chips in the house, because these had been common triggers for binge-eating episodes. Now these foods were always around. At first, Jean simply resisted eating them because they were not part of her diet. However, one night, while her boyfriend was out with his friends, Jean ate an entire bag of potato chips and finished off a package of cookies and three-quarters of a gallon of ice cream. Disgusted with herself and in pain from consuming so much food, Jean made herself throw up. Afterward, she went to the store to replace the food. To hide the fact that she had bought new food, Jean used the garbage disposal to get rid of some of the new ice cream and cookies so that the packages looked as they did before. Jean vowed not to eat any more of these "dangerous foods" and told herself that this was just a slip. However, the next week, when Jean was alone in the apartment, the same cycle happened again. She would binge and purge only when alone, because that was when the impulse became irresistible.

Jean was now binging and purging several times a week, even leaving work early to get home to binge and purge before her boyfriend arrived. Jean had regained the lost weight and had found that her weight was creeping above its pre-diet level. She then redoubled her efforts at dieting, as well as using self-induced vomiting, to counteract the effects of the binge episodes. Jean even began vomiting when eating normal amounts of food, because it felt

necessary to eat as little as possible to get rid of the unwanted weight. She felt disgusted with herself. As Jean's weight increased, she felt worthless and revolting.

Jamie, a Case of Binge-Eating Disorder

Jamie's problem was simply stated: "I eat too much. For some people, it's alcohol; for some it's cocaine. For me it's food." This had always been true; even when Jamie was a small child, a whole box of Twinkies was a single serving. For a junior high school bake sale, Jamie's mother had baked a cherry pie. The pie, Jamie's favorite dessert, was gone before the start of school that day. When the teacher asked for the dessert, Jamie lied to conceal the gluttony. There had been many times like this throughout childhood—episodes of eating all of something rather than just one serving. However, because Jamie was tall and athletic, that big appetite was often a source of pride rather than embarrassment. In fact, everyone on the athletic teams ate large amounts of food, so Jamie didn't feel unusual most of the time. Jamie didn't realize there was any eating problem until the end of college, when job interviews started. It was the first time Jamie needed to buy a suit but couldn't fit into any of the sizes offered in the normal department store. Jamie was embarrassed by having to go to a special store that stocked larger sizes.

At 35 years of age, Jamie currently weighed 360 pounds, despite several diets and weight loss programs. Jamie was frequently able to lose some weight on these programs; the greatest loss had been 50 pounds, down from 280 to 230 pounds. However, as at all other times, the weight had come back—and more. Jamie denied eating when not hungry but acknowledged eating to the point of being uncomfortably full. Jamie felt that this was because, when hungry or with favorite foods, eating occurred at one rate: "as much and as quickly as possible." Jamie said it was like being a "food addict;" there was no way to stop until all of the food was gone. For example, Jamie would consume three "value meals" from the local fast-food restaurant in the car on the way home from work. Eating alone in the car, Jamie said, was "the best," because "I can just zone out." Terrible guilt followed these episodes, because Jamie knew that eating so much junk food contributed to the weight problem—and could lead to heart problems. However, Jamie didn't like salads, vegetables, or fruit because they were bland and boring. "I wish I felt about fast food the way that I feel about salads because then I would be thin as a rail." Recognizing the existence of an eating problem that occurred every day—often throughout the day—Jamie wanted to know if there was any medication that would cause weight loss or make it easier not to eat. Based on all of the TV advertisements for such products, Jamie felt like a good candidate for medication.

Valerie, a Case of Purging Disorder

Valerie wasn't sure if she had a "real" eating disorder. She never identified with the magazine stories about skeletal actresses terrified of becoming fat or with TV movies about desperate teens who seemed to have perfect lives but who secretly gorged on food to stuff their feelings down and then vomited to void their emotional pain. Valerie knew that what she did wasn't "normal" and understood that it might be dangerous—the blood in her vomit worried her – but she wasn't sure if she had an "eating disorder." The "aha" moment came when a link on "purging disorder" drew her attention while searching the Internet for information about

vomiting blood. On reading the associated article and viewing a brief video clip, Valerie found that they matched what she had been doing for the last eight years. Valerie started scouring the Internet for more information, eventually finding an e-mail address for the person in the video clip and composing a message—a combination of affirmation that the disorder was real and a request for help. Valerie paused, wondering if she really wanted to attach her name to an admission of what she had been doing and if the person in the clip would even read or respond to the message. Finally, Valerie hit "send," exhausted from eight years of trying to convince herself that if no one ever noticed or asked about the vomiting, it must not be that big of a deal.

This was the message Valerie sent:

> I just found an article on Purging Disorder and wanted to know if you needed any one for your research. I think I've had this Disorder for several years. I had a bad case of mono in secondary school and lost over 4 kilograms while I was sick. I didn't want to gain the weight back. So, I tried to eat only what I had been eating whilst sick—chicken noodle soup, saltine crackers, and water. That worked for a while, but then my parents were worried that something else was wrong, and I started trying to eat normally around them so they wouldn't worry. I remember the first meal I got rid of—it was my mum's Shepards Pie. I felt so sick afterwards, like I had eaten the whole dish. I felt bloated and gross and was convinced that after weeks of living on chicken noodle soup and crackers, I had ruined it all with one dinner. So, I went to the bathroom, locked the door, turned on the shower, and threw up in the toilet until my stomach felt completely empty and clean. I felt relieved and in control again. I don't throw up every time I eat. Sometimes I can eat and be fine. Sometimes even a small amount of food has to be gotten rid of. But I never have huge binges, and I've never starved myself, and I've never gotten very thin. I am the thinnest that I've ever been, and everyone tells me that I look great and that I shouldn't worry about my weight. Of course, they don't know what I do to keep the weight off. What frightens me most is that if I eat normally and don't purge, then I would get really fat. But I'm also frightened that I'll never be able to eat normally, and I don't know when I'll stop this. I'm hoping that you can tell me what to do.

George, a Case of Night Eating Syndrome

George originally sought help through a sleep clinic because of trouble sleeping. His snoring was so bad that he no longer was able to sleep in the same bed as his wife. George described spending half of the night sleeping on the couch and half of the night up and feeling exhausted the next day. Results of sleeping tests indicated that George suffered from sleep apnea, which was likely made worse by weight: at 45 years old, George was 5 feet 11 inches tall and weighed 260 pounds. He was given a special mask that would ensure sufficient oxygen flow while sleeping. In addition, George's doctor explained how even modest weight loss and regular physical activity could improve sleep and quality of life. George felt like the sleep problems contributed to the weight problems, because frequently on waking up

at night, he would get something to eat to help him feel sleepy. Once full, George would lie down on the couch in front of the TV and eventually fall asleep. The next morning, he didn't feel hungry at all.

The doctor asked more about George's eating patterns. George reported generally skipping breakfast, in order to get as much sleep as possible before going to work, and if he ended up getting into work late, skipping lunch as well to make up the time. George started feeling hungry around mid-afternoon. Thus he was always hungry for dinner and usually had a big meal. George joked that it was as if his whole body was on the night shift. More nights than not, George would have a second or third meal in the middle of the night, when everyone else in the house was asleep. He would eat the leftovers from dinner, finish with a bowl of cereal, and then, if still up and hungry, might make some scrambled eggs and toast. In George's mind this was like having breakfast before getting to sleep, since he wouldn't be hungry for it in the morning.

After listening to George describe this pattern of eating, the doctor suggested seeing a specialist in the eating disorders clinic of the hospital. George was skeptical about going, because he wasn't an underweight teenage girl. However, the doctor explained that his colleagues were studying a condition called night eating syndrome and might be able to help figure out a way to get George's eating on schedule while the team at the sleep clinic helped him get back to sleeping through the night.

Current Knowledge and Ongoing Debates

The *DSM-5* (American Psychiatric Association, 2013) formally recognizes three eating disorders: AN (exemplified by Emily), BN (exemplified by Jean), and BED (exemplified by Jamie). However, most individuals who suffer from eating disorders do not fit into one of these three categories. These individuals would be diagnosed with an OSFED or an unspecified feeding or eating disorder. Studies often restrict inclusion to individuals who meet standard definitions for an eating disorder to increase reliability and the likelihood of replicating results. To the extent that OSFEDs and unspecified feeding or eating disorders represent subthreshold diagnoses that exist on a continuum with AN, BN, and BED, the causes, consequences, and effective treatment of these other disorders may be adequately addressed by research on defined eating disorders.

Thus one debate within the field of eating disorders is whether these disorders exist on a continuum or represent distinct categorical entities. Proponents of the continuum view point to similarities among individuals who are vulnerable to developing eating disorders, to the symptom overlap among disorders, and to the high longitudinal crossover between eating disorders. Emily and Jean both fear weight gain and engage in dietary restriction, and Jean and Jamie both experience uncontrollable urges to binge eat. Emily is underweight, Jean is normal weight, and Jamie is overweight. In several ways, these cases do appear to exist on a continuum of disordered eating attitudes and behaviors.

This dimensional view of eating disorders is a central premise of the Research Domain Criteria framework proposed by the National Institute of Mental Health (Cuthbert & Insel, 2013), which was introduced in Chapter 1. Rather than starting with categories of

mental illness, the RDoC approach starts with core constructs that explain individual differences from normality to pathological extremes in domains relevant for understanding psychopathology (see Figure 1.1 in Chapter 1). Rather than mapping symptoms onto diagnoses such as AN, BN, or BED, the RDoC approach seeks to understand behavioral features that cut across diagnostic boundaries, such as binge eating, by asking what genes, neural circuits, neurochemicals, hormones, behaviors, and self-reported experiences come together to produce the feature in question. Work in my own lab, for example, has pursued the explanation that binge eating emerges from an overactive drive to eat (high approach motivation) and underactive satiation (longer-term responsiveness to reward). This approach encourages researchers to pose broader questions that apply to a larger portion of individuals suffering from eating disorders and to frame questions in a way that identifies neurobiological processes that contribute to pathology. The ultimate hope is that understanding basic processes that contribute to symptoms will lead to the development of treatments.

There is evidence of discontinuity among AN, BN and BED as well, and this evidence indicates a need for caution in assuming continuity between these disorders and OSFEDs. For example, AN, BN, and BED show different historical and cultural patterns, suggesting that they likely have distinct etiological features. Further, each syndrome is associated with specific biological aberrations. To the extent that different factors contribute to AN, BN, and BED, these disorders do not exist on a continuum. In addition, BN responds to treatment with cognitive–behavioral therapy, interpersonal therapy, and antidepressant medication, whereas AN does not, and cognitive-behavioral therapy is a superior treatment for BN whereas it is not clearly better than alternative treatments for BED. Finally, AN, BN, and BED are associated with distinct courses and outcomes.

The implications of the debate over continuum versus categorical views are far-reaching. Much of the research on risk factors and prevention, as well as experimental analog studies, examines disordered eating attitudes and behaviors rather than specific eating disorders. The extent to which data from studies comparing high and low scorers on continuous measures of dietary restraint or bulimic symptoms are relevant for understanding AN and BN may be limited by the extent to which the continuum model of eating disorders is valid. To address the presence of syndromes not captured by the diagnostic criteria for AN, BN, or BED, the *DSM-5* introduced named examples within OSFEDs, including purging disorder (exemplified by Valerie) and night eating syndrome (exemplified by George). This development paves the way for studies to examine whether these syndromes reside on a continuum with existing eating disorders or represent distinct entities.

Chapter 2 explored the boundaries of eating disorders by describing conditions that are closely related to eating disorders without actually being eating disorders. There is clear overlap between the features of feeding disorders and obesity and those observed in eating disorders. For example, both ARFID and AN are characterized by restricted food intake. However, AN requires the presence of body image disturbance, while ARFID requires the absence of body image disturbance. Obesity is common among individuals with BED, and binge eating has been found to contribute to weight gain. However, not all individuals with BED are overweight (Keel et al., 2011), and most obese individuals do not have BED. The case studies in Chapter 2 exemplify key differences between feeding disorders

and eating disorders with respect to the gender of sufferers and age of onset, as well as highlighting the extent to which obesity is a physical condition that emerges from relatively commonplace behaviors rather than from the pathological processes that underlie mental disorders. Nevertheless, more research is needed to understand feeding disorders and their overlap with eating disorders. Future work may reveal that a subgroup of individuals with ARFID represent an early phenotype of AN. Placement of the feeding disorders in the same chapter as eating disorders within the *DSM-5* may facilitate work examining continuity between them.

Chapter 3 examined the stereotype that eating disorders predominantly affect White girls. This chapter revealed that eating disorders do not spare any demographic group and can occur in men and in individuals across a range of ethnic and racial groups. Jean is a Korean American woman who experienced strong pressure to lose weight from her mother, who was born in Korea. Thus eating disorders are not restricted to White individuals any more than they are restricted to Western cultures. While eating disorders are more common in women than men, these disorders may go undetected in men when they do not conform to the typologies presented by women. Jamie and George are distressed by their weight but may not be viewed as having body image disturbance, because their concern seems appropriate given the health risks associated with obesity. A diagnosis of BED does not require body image disturbance, which may explain why this disorder has a less skewed gender distribution than observed for other eating disorders. If it were decided that body image disturbance was a central feature of all eating disorders, this might decrease the number of individuals diagnosed with BED and particularly decrease the number of men diagnosed with BED.

Within the eating disorders field, there is debate about whether diagnostic criteria should be expanded to become more inclusive and more representative of the population with eating problems. Proponents of expanded criteria note that we tend to treat what we define. Thus definitions that exclude certain portions of the population from diagnosis also exclude them from treatment. Others have called for a more parsimonious approach to defining eating disorders that results in the fewest possible categories. These individuals note that there are risks inherent in pathologizing behaviors that do not represent an actual disorder. Considerable debate surrounded, for example, the introduction of BED as an official eating disorder in the *DSM-5*. The people who opposed it pointed to the symptom overlap between depression and BED, the high levels of depression in patients with BED, the similar gender distributions of depression and BED, and the fact that BED responds to many treatments with demonstrated efficacy in treating depression. They questioned whether it was ultimately more helpful or more harmful to assign a diagnosis of BED to an obese person already diagnosed with depression. In light of these concerns, it is important to consider that diagnostic criteria do not simply reflect who suffers from eating disorders but can actively create gender and ethnic differences in the distributions of these disorders. One consequence of deciding that BED is not an eating disorder would be a dramatic reduction in the representation of men and Black women among individuals diagnosed with an eating disorder. And certainly recognition of muscle dysmorphia as an eating disorder rather than a form of body dysmorphic disorder would increase the number of men counted in the eating disorder category.

Chapter 3 also reviewed findings suggesting that body image disturbance may be a more central feature in BN than in AN. This difference is not apparent in the case histories of Emily and Jean. In both women, body image disturbance appears to be the motivation for disordered eating. However, the disorders may differ in the extent to which body image concerns represent a *cause* versus an *interpretation* of disordered eating. Thus another significant debate within the eating disorders field is whether or not weight phobia represents a central feature of AN. In response to this debate, the *DSM-5* eliminated the requirement that individuals explicitly report a fear of gaining weight or becoming fat, instead letting engaging in behaviors to prevent weight gain serve as an indicator of the criterion. This change has been controversial (Brown, Holland, & Keel, 2014; Thomas, Vartanian, & Brownell, 2009). Proponents of the view that weight concerns are a central feature of AN point to the fact that increasing incidence of AN has coincided with increased idealization of thinness and to the role that weight concerns play in many patients' initial food restriction and continued refusal to eat. In addition, studies in the United States and Canada suggest that individuals with AN without an intense fear of fat simply look like individuals with a less severe version of AN at presentation and follow-up (Thomas et al., 2009). Because these observations suggest that weight concerns are associated with outcome in clinically meaningful ways, they support including this symptom in the definition of AN.

This debate is almost impossible to resolve by assessing individuals in modern Western cultures. In Western culture, weight concerns represent a meaningful symptom of AN. That is, a patient's expression of terror over gaining weight reflects the intensity of her disorder—even if her weight concerns represent an interpretation of self-starvation rather than the true cause. Thus varying levels of weight concern are analogous to varying levels of being underweight: Both represent differing levels of symptom severity. Because outcome studies suggest that symptom severity is associated with outcome in AN, it would be surprising if women with more severe weight concerns did not experience worse outcomes than women with less severe weight concerns do. A more valid comparison can be made in non-Western cultures that offer alternative explanations for self-starvation. What might be expressed as terror over gaining weight in one context may be expressed as terror over feeling sick in another. The intensity of the expression—that is, whether it is terror or just discomfort—can be similar even if the focus of the fear differs by cultural context. In non-Western cultures, differences in severity have not been found between AN patients with and without weight concerns, suggesting that weight concerns may not be a central feature of the disorder. Nonetheless, in a culture with ubiquitous idealization of thinness and denigration of fatness, there are individuals who do develop AN because of these messages. A question then remains: How many of them might have developed AN without those messages?

Chapter 4 reviewed methods for examining risk factors for eating disorders, including cross-sectional studies of correlates of disorders, longitudinal studies, experimental studies, and studies of natural phenomena. The limitations of each method were also discussed. For example, dieting appears to be a common correlate of eating disorders in all of our case studies. This pattern suggests that dieting may serve as a risk factor for eating disorders. However, dieting appears to be a response to obesity in both Jamie and George, illustrating why correlations do not prove causation.

Whether dieting represents a risk factor for eating disorders or an important and safe intervention for the prevention of obesity is probably one of the more heated debates in the field of eating disorders. Proponents of dieting note that obesity is increasing in the United States, particularly in certain subpopulations. As a consequence, certain weight-related diseases are increasing as well. For example, type 2 diabetes is increasing among adolescents as a consequence of increasing obesity rates. Thus from a public health perspective, weight loss appears to be an important goal for many adolescents. However, a number of individuals in the eating disorders field feel that the emphasis should be not on weight loss dieting but on healthy eating and physical activity. These individuals believe that focusing on a number such as BMI encourages attitudes that lead to eating disorders, and they argue that the emphasis on weight reflects societal biases against overweight individuals. They point to the limited long-term efficacy of weight loss dieting and the extent to which such dieting sets people up for failure, risks decreasing their self-worth, and may contribute to the onset of binge eating. However, data on dieting as a risk factor for eating disorders are based largely on studies of normal-weight populations, so they may not generalize to people who are significantly overweight.

Chapters 5 and 6 examined two forms of social influence that may contribute to the development of eating disorders. At the societal level, media messages reinforce the importance of thinness and the denigration of fatness. A common thread in all our case studies is the perceived importance of not being fat. This belief appears to contribute to self-starvation in Emily, self-induced vomiting in Jean and Valerie, and significant distress over binge eating in Jamie and distress over night eating in George. Families and peers may contribute to the development of eating pathology by serving as conduits of such messages. Even psychodynamic and systems-oriented theories take into account the presence of disturbed parental eating patterns, suggesting that parents may directly model behaviors that reinforce the thin ideal. Disturbed parental eating patterns also may reflect the influence of genetic factors. Thus genetic and environmental factors may be correlated and enhance the influence of familial factors on eating disorders. Although genetic and environmental factors likely work in concert to increase the risk of eating disorders, nature-versus-nurture debates still occur.

Despite the general consensus that a biopsychosocial model is necessary for improving understanding the causes of eating disorders and for advancing their treatment and prevention, experts disagree on what focus will provide the "next big break." Proponents of biological research feel that molecular genetic and neuroimaging studies represent the future and note the limited impact of psychosocial investigations. Proponents of psychosocial research feel that biological approaches have limited relevance for understanding disorders that appear to be dramatically influenced by psychosocial factors and that such approaches risk biological reductionism. Each side points to examples of shoddy methodology and poorly supported conclusions in the other's area. Indeed, one can find examples of poorly conducted research in any area of inquiry. Still, it would be an error in logic to conclude from specific examples of poor research that an entire area of inquiry has little value.

Chapter 7 described psychological factors that appear to contribute to the onset and maintenance of eating disorders. Individuals with eating disorders appear to share cognitive distortions such as dichotomous thinking, selective abstraction, and overemphasis on the

importance of weight and shape. For example, Emily, Jean, and Valerie follow food rules in which some foods are "good" (e.g., salad) and other foods are "bad" (e.g., ice cream). In addition, the symptoms of eating disorders appear to represent a vicious cycle in which processes of positive and negative reinforcement maintain disturbed patterns. Emily's dietary restriction and both Jean's and Valerie's self-induced vomiting are negatively reinforced by their fear of gaining weight, and Jean, Jamie, and George find themselves irresistibly drawn to the rewarding effects of forbidden foods. Personality may provide the predisposition for thinking and behaving in ways that contribute to the development of eating disorders. Like Emily, individuals who develop ANR are characterized by being perfectionistic, rigid, and reserved. Individuals with bulimic symptoms are characterized by being impulsive and emotionally labile. Perfectionism also characterizes individuals who develop BN. Thus it is possible for Jean to have perfectionist strivings to maintain a certain body type but to fail to meet these rigid standards because of problems with impulse control.

Within the eating disorders field, an interesting question has been raised concerning whether personality provides a predisposition to eating pathology or is a defining aspect of such pathology. Some researchers have argued that personality is so closely involved in the development of eating disorder subtypes that personality features should be included in the definitions of these subtypes. Thus, for example, extreme perfectionism and rigidity would be symptoms on a par with self-starvation in defining ANR. These researchers have argued that identifying stable personality traits that existed before and continue after an eating disorder holds promise for understanding the true causes of eating pathology by identifying a stable phenotype. Other researchers have noted the significant instability and inconsistency of personality findings for most eating disorders other than ANR. For example, problems with impulse control appear to remit with the remission of bulimic symptoms, suggesting that these problems may not reflect personality traits at all. Thus personality disturbance may play a more central role in understanding the pathology of ANR than it does for other eating disorders.

Chapter 8 introduced biological factors thought to contribute to the risk of eating disorders, as well as biological correlates and consequences of these disorders. Dysregulation of serotonin (5-HT) has been described as a correlate of eating disorders, particularly BN. Neuropeptides that regulate weight and feeding also have been implicated in the etiology of eating disorders. Genes appear to play an important role in eating disorder etiology. Molecular genetic studies have focused especially on genes related to 5-HT function, obtaining results that support a role for these genes in eating disorders. Taken together, these biological factors may influence the body's responses to food restriction and its defenses against starvation. Differences between Emily's and Jean's reactions to dietary restriction—emaciation in the former and binge eating in the latter—may represent the absence in Emily of a biological weight-defending mechanism that is present in Jean. Similarly, Valerie, despite engaging in more extreme behaviors to control her weight than Jean, does not lose substantial amounts of weight. Interpretation of biological correlates is complicated by the possibility that they may be biological consequences of an eating disorder. Because eating is central to life, disruptions in food intake and extreme methods of weight control lead to serious medical consequences.

Thus one debate in the eating disorders field has centered on the validity of examining biological differences between controls and patients recovered from an eating disorder. Some researchers have argued that biological differences found between recovered patients and controls reflect stable predisposing factors that contribute to the onset of the disorder in question. Others have argued that these differences may reflect consequences of the eating disorder that remain, like a scar, after the disorder remits. Moreover, comparisons of brain function (or any biological factor) between controls and recovered patients raise an interesting logical paradox. If a factor is related to the onset of an eating disorder, why would the disorder go into remission when the factor is still present? In contrast, if the factor is present only when the disorder is present, it could mean that the factor was caused by the disorder. The best approaches to resolving these issues involve prospective longitudinal studies. However, current technologies for evaluating brain function and other biological factors are invasive and expensive, making it difficult to include them in large prospective longitudinal studies.

Chapter 9 provided information about the treatment of eating disorders. Several efficacious treatments have been identified for BN and BED. Jean had a history of successful treatment response and ultimately achieved lasting recovery from BN following treatment. Efficacious treatments for BN and BED include cognitive–behavioral therapy, antidepressant medication, and interpersonal therapy. These interventions have efficacy in treating depression and anxiety disorders as well. In contrast, the most efficacious treatment identified for AN thus far is a family-based intervention. Several factors may explain why one treatment does not seem to work for all eating disorders, including age differences among individuals with different eating disorders and differences in the extent to which the disorders are ego-syntonic (as in Emily) versus ego-dystonic (as in Jean and Jamie). When a disorder is ego-syntonic, this means that the person afflicted by the disorder views the illness or its symptoms as being consistent with who he or she is as a person. This poses a challenge for patients' motivation in treatment because asking them to change their attitudes and behaviors may be experienced by patients as asking them to change who they are. In contrast, ego-dystonic disorders are experienced as highly distressing to patients, which increases their motivation to work in treatment towards recovery. Because purging disorder is a newly identified OSFED, there are no evidence-based treatments for this condition. Thus when Valerie sought help for her eating disorder, her therapist had to develop or adapt a treatment. Unfortunately Valerie did not have the chance to recover, because her life ended too soon. In contrast, George responded well during the medication study and experienced dramatic improvements in his sleep, mood, energy, and appetite.

Beyond the issue of treatment efficacy is that of treatment effectiveness—how well treatments work in the real world. There are debates about why clinicians often do not use empirically supported, manual-based treatments in working with patients with eating disorders. From the researcher's perspective, failure to use such treatments reflects clinicians' lack of knowledge and training, and the solution is to work harder to disseminate information from treatment research. From the clinician's perspective, treatment studies lack ecological validity, because patients in those studies are not representative of the larger population seen by clinicians. Quite often, controlled treatment studies restrict inclusion to patients

who meet full criteria for AN, BN, or BED; have no current problems with substance use; and do not report suicidal intentions. Clinicians rarely encounter such uncomplicated cases. A second concern of clinicians is the perceived rigidity of manual-based treatment. They fear that using a manual will reduce their ability to establish rapport with patients, with the possible result that an ambivalent patient will decide to leave treatment prematurely. Clearly there are valid concerns on both sides of the debate. Patients have the right to expect that a clinician will provide them with the treatment most likely to promote recovery. Clinicians have the right to expect that researchers will conduct research on treatments that can be successfully implemented in the real world.

Chapter 10 reviewed efforts to design efficacious programs to prevent eating disorders. This chapter has changed the most from the first to the second edition of this book. At the time of the first edition, few prevention programs had demonstrated much impact on disordered eating behaviors. Now, several prevention programs have shown substantial benefits in reducing future onset of serious eating disorders. The designs of studies testing these programs also provide a rigorous approach to testing etiological models for eating disorders. For example, finding that an intervention designed to increase physical activity decreased risk for obesity and purging in girls supports results of retrospective, follow-back designs regarding the role of greater weight in increasing risk for bulimic symptoms. Similarly, evidence that an intervention that reduces internalization of the thin ideal also reduces disordered eating symptoms supports an etiological role for body image disturbance in the development of eating pathology.

Chapter 11 reviewed the courses and outcomes of eating disorders. Anorexia nervosa is associated with a worse prognosis than BN in terms of mortality rate and recovery rate, and BN is associated with a worse prognosis than BED. Jean's response to treatment and her ultimate recovery represent the most likely outcome for someone with BN. Because Emily had an older age of onset (young adulthood versus early adolescence) and required inpatient treatment, her prognosis is less favorable. She is less likely to respond to a family-based intervention, and alternative treatments with proven efficacy are lacking. Based on preliminary outcome data for BED, Jamie has a good chance of recovery, but he may face a lifelong struggle with weight control. Limited data on course and outcome for purging disorder and night eating syndrome contributed to the decision by the *DSM-5* eating disorders workgroup to place these disorders within the OSFEDs rather than introducing them as official eating disorder diagnoses.

Probably the greatest debate in long-term outcome research has to do with the long-term efficacy of treatment. Though some studies have provided limited evidence that treatments affect long-term outcome in AN or BN, most suggest that treatment does not predict long-term outcome. One approach to resolving this issue is to examine the long-term impact of treatment not just on outcome but on course. Such examinations have indicated that some efficacious treatments speed recovery. However, the failure of treatments to show an impact on long-term outcome challenges researchers to develop more powerful interventions. The need for better interventions is underscored by the significant proportion of patients who do not respond to even the most efficacious treatments available. Cognitive–behavioral therapy, for example, is the first line of treatment recommended for BN, yet only 30% of patients who enter CBT achieve recovery.

Future Directions

Because the field of eating disorders is still relatively young, heated debates surround almost every topic related to these disorders. However, the field is expanding rapidly. In 1981 the first journal to focus on eating disorders, the *International Journal of Eating Disorders*, was established. Since then, six more journals dedicated to the subject have emerged: *Eating Disorders: Journal of Treatment and Prevention* in 1993, *European Eating Disorders Review* in 1993, *Eating and Weight Disorders* in 1996, *Eating Behaviors* in 2000, *Body Image* in 2003, *Advances in Eating Disorders: Theory, Research, and* Practice in 2013, and the *Journal of Eating Disorders* in 2013. A new generation of researchers specializing in eating disorders has joined the ranks of junior faculty in medical schools and colleges around the world, giving students greater opportunities to study eating disorders and eventually specialize in the field. To promote public awareness and support for research on and treatment of eating disorders, a coalition of eating disorder associations has emerged, and the National Institute of Mental Health has a designated officer to focus on the development and funding of research on these disorders. The advances thus far and the opportunity to make significant contributions in the near future make this an exciting time in the eating disorders field.

The field's expansion provides multiple avenues along which future research can develop. One such avenue is expanding controlled treatment studies to include individuals with OSFEDs rather than limiting studies to individuals with full-criteria eating disorders. Another avenue is incorporating features from other efficacious interventions into existing evidence-based treatments such as cognitive–behavioral therapy. In addition to new psychosocial interventions that are under examination, new medications are being developed and tested, and the combination of medication and therapy in stepped-treatment programs is being explored. Empirical studies of the development of eating disorders among immigrants from non-Western nations to the United States are being initiated. Studies of neurocognitive function in eating disorders are increasing as neuroimaging technology develops further, and multinational collaborative molecular genetic studies are underway. The National Institute of Mental Health has established a focus on translational research to promote work across traditional boundaries between basic science and clinical research and to encourage communication among investigators in these different lines of research.

Thus rather than curtailing certain research avenues, current debates serve to keep several lines of investigation open simultaneously. In closing, what remains unknown in the field of eating disorders represents not so much a failure of past research as a challenge for the future.

Glossary

5-HT: see *serotonin*.

12-month prevalence: the percentage of a population affected by a condition over a one-year period.

Acculturative stress: distress experienced during the process of adapting to a new culture.

Affective disinhibitor: an emotional trigger for loss of restraint over eating.

Alexithymia: inability to identify one's emotional states; confusion in differentiating among internal states.

Allele: one of several alternative forms of the same gene.

Amenorrhea: loss of menstrual periods for three consecutive months.

Analog studies: experimental studies testing a hypothesized relationship between an independent and a dependent variable in which one or both are analogous to the topic of study.

Arcuate nucleus: a portion of the hypothalamus implicated in the regulation of food intake and body weight.

Ascetics: individuals who engage in extreme abstinence or self-control.

Association studies: molecular genetic studies that examine whether there is an association between a specific gene and the presence of a specific trait.

Body Mass Index: metric for evaluating body weight that is calculated as weight in kilograms divided by the square of height in meters.

Brain-derived neurotrophic factor: protein that contributes to the growth, development, and maintenance of neurons.

Candidate gene study: study designed to examine whether individuals with a particular disorder are more likely to have a certain allele or combination of alleles for a specific gene than are individuals who do not have the disorder.

Central coherence: ability to "see the big picture" or to derive overall meaning from details.

Cholecystokinin: peptide that is released in the small intestine following food ingestion and is associated with eliciting a feeling of satiation.

Cognitive–behavioral therapy: a form of directive therapy organized around the theory that disorders consist of reinforced behaviors to which there are healthier alternatives and of irrational beliefs that need to be elicited, challenged, and replaced.

Cognitive disinhibitor: a cognitive trigger for loss of restraint over eating.

Cognitive distortions: thoughts that do not correctly reflect reality.

Complex inheritance: form of heredity in which the combined action of multiple genes controls the expression of a phenotype.

Constraint: a dimension of personality representing the tendency to inhibit impulses and to show caution, restraint, and conventionalism.

Conversion disorder: a disorder characterized by expression of physical distress in place of suppressed psychological distress.

Correlation: concurrent association between two variables.

Cost effectiveness: extent to which an intervention provides the best outcome for the lowest price.

Course: the path a patient's illness follows on its way to an outcome.

Crossover: transition from having one eating disorder to having another.

Deoxyribonucleic acid (DNA): a sequence of nucleotides that stores genetic information.

Dialectical behavior therapy: a form of cognitive–behavioral therapy, originally designed to treat patients diagnosed with borderline personality disorder, that focuses on building skills in mindfulness, distress tolerance, interpersonal effectiveness, and emotional regulation.

Diathesis: a vulnerability factor for developing a mental disorder.

Dichotomous thinking: black-and-white thinking; a pattern of thinking in which things are categorized as either good or bad.

Disease Prevention Paradigm: theoretical approach to prevention based on identifying and then reducing specific risk factors for a disease.

Disease-Specific Pathways Model: see *Disease Prevention Paradigm*.

Disengagement: condition in which boundaries between family subsystems are too strong

Distress tolerance: the ability to develop safe ways of coping with painful emotions without resorting to impulsive (and often self-destructive) behaviors that will ultimately increase emotional pain.

Dizygotic twins: twins who share, on average, 50% of their genes.

Dopamine: a neurotransmitter that appears to inhibit food intake by acting on receptors in the lateral hypothalamus and that is involved in experiencing reward.

Double-blind placebo-controlled study: a study design in which participants are assigned (usually at random) either to active treatment or to inactive treatment that resembles active treatment in order to rule out the possibility that participants in active treatment improve because they expect treatment to be effective.

Downregulation: a process by which neurotransmitter receptors become less sensitive due to increased neurotransmitter availability in the brain.

Ecological validity: how well results of an experimental study reflect real-world situations.

Edema: fluid retention.

Efficacy: ability to produce change.

Emotional regulation: the ability to experience emotions without having extreme fluctuations that interfere with life function.

Empirical support: with respect to treatment, evidence of efficacy from research studies.

Empowerment-Relational Model: theoretical approach to prevention based on empowering girls to transform their social environments so as to eliminate sources of risk.

Enmeshment: condition in which boundaries between family subsystems are too weak.

Epidemiology: the study of the occurrence of disorders within populations.

Equal-environments assumption: the assumption that members of twin pairs reared together share 100% of their home environment, regardless of twin type.

Etiology: the causes and origins of mental and physical disorders.

Experimental studies: studies in which an independent variable is manipulated to determine its effect on a dependent variable.

Family systems model: theory that all families are made up of subsystems (e.g., spousal, parental, and sibling) involving different roles and responsibilities.

Functional magnetic resonance imagining (fMRI): a method used to link activity in brain regions or circuits during lab-based tasks to emotions, thoughts, or behaviors.

Genome: the entire set of genes present in an organism.

Genome-wide association study: a study evaluating genes across an entire genome to determine whether specific alleles are linked to the presence of a condition.

Genotype: the specific combination of alleles that an individual has for a given gene.

Ghrelin: a peptide released from the stomach that stimulates hunger.

Glucagon-like peptide 1 (GLP-1): a peptide released from the gut after eating that influences the experience of satiation and meal termination.

Harm avoidance: a dimension of temperament representing a tendency to avoid punishment by inhibiting behavior.

Health Promotion Paradigm: theoretical approach to prevention based on identifying and then increasing protective factors at both individual and community levels to promote wellness.

Heritability estimate: a percentage representing how much genes contribute to the development of a disorder in a given group of people.

Heterozygous: having two different alleles for a gene.

Homozygous: having two identical alleles for a gene.

Hypothalamic–pituitary–adrenal (HPA) axis: a system linking the hypothalamus, the pituitary gland, and the adrenal glands that is implicated in responses to stress.

Hypothalamic–pituitary–gonadal (HPG) axis: a system linking the hypothalamus, the pituitary gland, and the gonads that is involved in the release of sex hormones and in maturation.

Hypothalamus: a brain structure that plays a central role in appetite and weight control.

Incidence: the number of new cases of an illness per 100,000 people per year.

Indicated prevention program: a secondary-prevention program designed for individuals already demonstrating features of the full condition to be prevented.

Interoceptive awareness: a personality measure of ability to recognize one's own feelings and internal states.

Interpersonal effectiveness: the ability to relate to people in a way that meets personal needs, the needs of the other person, and the need for self-respect.

Interpersonal therapy: a form of therapy organized around the theory that disorders arise from problems patients have in negotiating their relationships with others.

Intrapsychic: occurring within the mind.

Leptin: a neuropeptide that provides a negative feedback loop in the brain's control of weight and eating.

Lifetime prevalence: the overall percentage of people who have had a specific disorder at some point in their lives.

Longitudinal studies: studies involving repeated measurements of individuals or groups followed at different points over time.

Mediator: variable that represents a link in a causal chain, such that a first event causes a change in the mediator, and the change in the mediator causes a change in outcome.

Mendelian inheritance: form of heredity in which the action of a single gene controls the expression of a phenotype.

Meta-analysis: an analysis of data from multiple studies to determine overall trends and significance.

Mindfulness: the ability to be aware of internal feelings and external demands in a way that combines emotions with intellect in guiding wise choices.

Moderator: a factor that influences the association between two other variables. For example, a factor that contributes to a treatment being more successful in some people than others would be a moderator.

Monozygotic twins: twins who share approximately 100% of their genes.

Muscle dysmorphia: a condition characterized by viewing one's body as much smaller than it really is, causing significant distress and leading to efforts to increase muscle mass.

Naturalistic investigations: studies in which natural events are observed.

Negative emotionality: a dimension of personality representing the tendency to experience negative mood states (e.g., sadness, anxiety, and anger).

Negative reinforcement: the phenomenon in operant conditioning in which a behavior's likelihood increases because not engaging in the behavior has an undesirable consequence.

Neurasthenic disorders: a class of disorders commonly diagnosed in the late 19th and early 20th centuries in which patients complained of fatigue and physical symptoms such as headache, muscle pain, and problems with hearing or vision.

Neuron: a major type of brain cell.

Neuropeptide Y: a neuropeptide whose activity increases food intake and is inhibited by leptin.

Neuropeptides: a class of chemicals that activate specific areas of the brain but are physically larger than neurotransmitters.

Neurotransmitters: chemicals in the brain that facilitate communication between neurons.

Nocturnal eating: eating that occurs at night, particularly after a person wakes at night.

Nonorganic failure to thrive: a condition in which young children fail to make necessary weight gains and no biological reason can be found for their low weight.

Nonspecific Vulnerability-Stressor Model: theoretical approach to prevention based on identifying and then reducing general risk factors for illness.

Norepinephrine: a neurotransmitter that has roles in both increasing and decreasing food intake and is involved in physiological responses to stress.

Novelty seeking: a dimension of temperament representing a tendency to pursue rewards.

Nucleus accumbens: region of the brain that is part of the dopaminergic reward pathway (from the nucleus accumbens to the ventral tegmental area) and is implicated in experiencing reward and learning environmental sources of reward.

Nuisance variable: anything that might create differences between conditions in a study but is irrelevant for understanding the problem under investigation.

Obesogenic: contributing to the development of obesity.

Objectification: process in which individuals are viewed as objects (things) rather than as agents (people).

Operant conditioning: a type of learning in which associations between behaviors and reinforcement (positive or negative) or punishment influence the likelihood that those behaviors will recur.

Outcome: the condition of a patient assessed at some point after initial assessment.

Paraventricular nucleus: section of the hypothalamus, named for its location outside of the third ventricle.

Peptide YY (PYY): a peptide released from the gut after eating that influences satiety and delay before eating again after a meal.

Persistence: a dimension of temperament representing a tendency to continue behavior that is no longer rewarded.

Personality: a stable way in which individuals perceive, react to, and interact with their environments that is influenced by both biology and experience.

Pharmacological disinhibitor: a psychoactive substance that causes loss of restraint over eating.

Phenocopy: a condition in which a phenotype occurs in the absence of the genotype with which it is associated.

Phenotype: the observable manifestation of a genotype.

Placebo effect: improvement that occurs because an individual expects treatment to help her or him get better.

Point prevalence: The percentage of a population affected by a condition at a given time.

Positive emotionality: a dimension of personality representing the tendency to enjoy and actively engage in work and social interactions.

Positive reinforcement: the phenomenon in operant conditioning in which a behavior's likelihood increases because engaging in the behavior has a desirable consequence.

Positron emission tomography (PET): a method for studying the activity of particular chemicals in areas of the brain.

Proband: an individual exhibiting a trait whose inheritance is to be studied.

Prognostic: predictive of a likely course or outcome.

Psychoanalytic: referring to a theory of psychopathology as arising from unconscious intrapsychic conflicts.

Psychodynamic therapy: a form of nondirective therapy organized around the theory that disorders arise from internal conflicts of which the patient may not be aware, with the goal of facilitating improvement by promoting insight.

Psychoeducation: factual information provided in the context of therapy to educate patients about their disorders.

Psychogenic vomiting: vomiting for which there is no physical explanation.

Punishment: the phenomenon in operant conditioning in which a behavior's likelihood decreases because engaging in the behavior has an undesirable consequence.

Pyloric sphincter: a muscle at the base of the stomach that contracts and relaxes to control the rate at which food passes from the stomach to the small intestine.

Randomized controlled trial: study design in which participants are assigned at random to a treatment or control condition in order to rule out the influence of selection factors on response to treatment.

Receptor: structure in neurons that allows them to receive neurotransmitter signals.

Reduced penetrance: a condition in which a genotype does not always lead to the corresponding phenotype.

Refeeding syndrome: a life-threatening condition caused by reintroducing too much food too quickly to a person in a state of starvation.

Relapse: return of a full disorder after remission has been achieved.

Representativeness assumption: the assumption in twin studies that patterns observed in twins are representative of patterns observed in the nontwin population.

Resting metabolic rate: the rate at which the body consumes energy in the form of calories when it is not exercising.

Retrospective recall bias: the influence of factors in the present on memory for the past such that memory is less accurate.

Reuptake: process by which a neuron reabsorbs neurotransmitters it has just released before they can bind to receptors on another neuron.

Reverse anorexia: see *muscle dysmorphia*.

Reward dependence: a dimension of temperament representing a tendency to continue rewarded behavior.

Ribonucleic acid (RNA): a sequence of nucleotides transcribed from DNA that is used to build a chain of amino acids into a protein.

Selective abstraction: a cognitive process in which part of something comes to represent the whole and carries undue influence in the evaluation of the whole.

Selective prevention program: a prevention program designed for specific segments of the population that are at increased risk for certain problems.

Serotonin (5-HT): a neurotransmitter that plays an important role in the regulation of mood, appetite, and impulse control.

Set-point theory: the theory that the body has, as a result of evolution, behavioral and biological mechanisms for maintaining a certain weight.

Socioeconomic status: a combined index of educational and professional attainment and income.

Somaticize: to express a mental conflict as a physical condition.

Standardized mortality ratio: ratio of the observed number of deaths in a specific group to the expected number of deaths in a matched population.

Temperament: a biologically based predisposition to experience certain emotional and behavioral responses.

Transmission disequilibrium test (TDT) study: a study that compares the frequency of allele transmission to offspring with a disorder from parents who are heterozygous for the corresponding gene with the frequency expected if there were no association between the allele and the disorder.

Tryptophan: an amino acid that is required to make serotonin.

Twin concordance: the similarity within twin pairs for a specific trait.

Universal prevention program: a prevention program designed for a general population.

Upregulation: a process by which neurotransmitter receptors become more sensitive as a result of sustained low concentrations of a neurotransmitter in the brain.

Vagus nerve: a cranial nerves with both motor and sensory functions, including stimulating smooth muscle fibers in the stomach and intestine and receiving sensations from the abdomen.

Variable expressivity: a condition in which a genotype leads to variable phenotypes.

Ventral tegmental area: region of the brain that is part of the dopaminergic reward pathway (from the nucleus accumbens to the ventral tegmental area) and is implicated in experiencing reward and learning environmental sources of reward.

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Additional Resources

BOOKS

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WEBSITES

Proud2Bme ("an online community created by and for teens. . .. with the goal of promoting positive body image and encouraging healthy attitudes about food and weight"): http://proud2bme.org
ED Bites ("the latest tasty tidbits in eating disorder science"): http://edbites.com
Science of Eating Disorders ("making sense of the latest findings in eating disorders research"): http://www.scienceofeds.org

FILM

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About the Author

Pamela K. Keel, PhD, is a Professor in the Department of Psychology at Florida State University. Dr. Keel conducts research on the nosology, biology, epidemiology, and longitudinal course of bulimic syndromes and has authored over 200 papers and two prior books on eating disorders. Within her NIH-funded program of research, Dr. Keel defined and characterized purging disorder as a potentially new eating disorder, resulting in its inclusion in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition* (*DSM-5*). Dr. Keel is a Fellow of the American Psychological Association, the Association for Psychological Science, and the Academy for Eating Disorders. She also served as President for the Academy for Eating Disorders (2013-2014) and the Eating Disorders Research Society (2009-2010).

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