Chapter 12

Eating Disorders: Bulimia Nervosa and Binge Eating

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Disordered eating can take a variety of forms, with the core problem behaviors identified as restrictive eating, binge eating, and compensatory behaviors. The current classification system DSM-IV-TR (American Psychiatric Association [APA], 2000) includes two specific diagnoses for eating disorders: Anorexia Nervosa (AN), and Bulimia Nervosa (BN), as well as a nonspecific diagnosis Eating Disorder Not Otherwise Specified (EDNOS) that applies to the wide variety of distressing and impairing patterns that do not meet criteria for a specific disorder. Anorexia Nervosa is discussed separately (in Chapter 13) because its diagnosis and treatment has been dominated by a focus on weight (rather than behavior), and the most important criterion for therapy outcome has been weight restoration. AN has traditionally been viewed as having a fairly distinct etiology compared to BN. However, individuals achieve and maintain low weight through a variety of methods, so the behaviors and attitudes associated with AN overlap considerably with the patterns of binge eating that will be discussed in this chapter.

The primary emphasis here will be the two patterns of binge eating that have been studied most extensively—BN and the EDNOS pattern labeled Binge Eating Disorder (BED). BED is currently a provisional diagnosis but some version is likely to be approved in the forthcoming revision of the DSM, so we refer to BED as a disorder in this chapter. Unlike the binge-purge subtype of AN, neither of these patterns has a weight criterion and neither diagnosis is assigned unless the binge eating behavior occurs.
outside the course of an episode of AN (i.e., low weight). These two maladaptive eating patterns are characterized by: (a) objective binge eating (distressing feelings of loss of control over the process of eating) and/or (b) “compensatory” behaviors (deliberate efforts to minimize intake or minimize its impact on weight). We also address the current diagnostic controversies that are highlighted by the fact that eating behaviors and weight have a complex and uncertain relationship. It is not clear how best to fit behaviors and weight criteria into the diagnostic system, since weight status clearly impacts treatment decisions.

Symptoms and Diagnostic Criteria

**Bulimia Nervosa (BN)**

BN is characterized by recurrent binge eating episodes and recurrent compensatory behavior designed to minimize the impact of those episodes (preventing weight gain). The individual must also highly value body shape and weight, and those must disproportionately factor into the individual’s self-evaluation and self-worth (overconcern with weight and shape). Specific criteria—as described in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*, APA, 2000)—require that the binge episodes be objectively large (objective binge episodes, called OBEs), which is defined as eating more than most people would eat in a similar situation and in a discrete period of time (e.g., two hours). A binge is defined by the subjective experience of loss of control at some point during an eating episode. Defining loss of control has been fairly problematic since most individuals can (and do) stop eating if there is an unexpected external disruption such as another person entering the situation. The general guideline for defining objectively large is a portion at least three times the typical portion size for that food. The OBEs and compensatory behaviors often occur together, forming a distinct binge-purge episode—but that is not always the case and is not a requirement for diagnosis. Nonetheless, both OBEs and compensatory behavior must occur, on average, at least twice a week for three months. Subjective binge episodes (called SBEs)—which may or may not trigger compensatory behaviors—are also frequently reported but those do not count toward meeting the frequency criterion for binge episodes.

**Subtypes**

The two subtypes of BN are based on the primary type of compensatory behavior employed: purging (self-induced vomiting or misuse of laxatives, diuretics, or enemas) or nonpurging (fasting or excessive physical activity). Some individuals rely primarily on one method—such as self-induced vomiting—but many utilize several methods and their use patterns vary over time. In clinical settings, the purging subtype of BN is reported more frequently than the nonpurging subtype: 80 to 90% of treatment seeking individuals with BN report engaging in vomiting behaviors, while one-third of individuals with BN report using laxatives after binge eating (APA, 2000). However, data suggest that the nonpurging subtype may be more common (or at least as common as the purging subtype) in the general population (Favaro, Ferrara, & Santonastaso, 2003). If such individuals have not become overweight due to their binge eating, they may not view their problem as a potentially treatable disorder and they are generally less motived to see a psychotherapist, body image issues are typically quite evident. As the purging subtype is associated with the total intake of food, it is a more acknowledged and recognized form of the disorder. The pattern of purging is a key feature of the diagnosis of BN. As the pattern of purging becomes more evident, the purging subtype is diagnosed. Common triggers include interpersonal stress, broken relationships, disordered beliefs about comfort or control, and shame. In more like a ha becomes very purge. Due addition to fertility, 2014; Polivy & Herman, 2004). Individuals Fairburn & HA that they consi either wanting weight if they to stop binge. While the init
motivated to seek treatment. Individuals with the nonpurging subtype of BN report less severe psychopathology than those who purge—especially in terms of mood disturbance, body dissatisfaction, and physical abnormalities (APA, 2000)—although they are typically quite distressed about their weight and tend to be heavier than individuals with the purging subtype. Individuals who meet most but not all of the criteria (e.g., frequency) are considered to have subclinical BN, although officially they would receive a diagnosis of EDNOS.

Clearly identifiable binge/purge episodes tend to occur when the individual is alone. The behaviors are often quite secretive and are typically associated with high levels of shame and embarrassment. Oftentimes binges are comprised of high fat, high calorie foods such as pastries or ice cream, but a binge may be large amounts of healthy foods. Individuals often restrict their food intake between binge/purge episodes, and they often binge on the same foods they are trying to restrict. Meals are frequently skipped. The most typical pattern is a small to no breakfast or lunch, with intake postponed as long as possible in the day. Frequency of eating is minimized as the individual is trying to limit total intake and feels at risk for binge or overeating whenever they do eat. Both type and amount of food may be severely limited by a set of rules—for example, only healthy foods, no fat, no processed foods, no meat. Binges are often unplanned (normal eating turns into overeating and then binge eating), at least early in the course of the disorder.

As the pattern crystallizes, it becomes more common that individuals fight the urge to binge and purge. They are then very vulnerable when they are alone and have the opportunity to binge and purge. When the urge to binge builds, the person typically plans to binge at a time when they will not be detected. Some individuals then experience a period of relief from the urges and can refrain for several days. Evenings are especially high-risk times for many individuals with BN (Deaver, Miltenberger, Smyth, Meidinger, & Crosby, 2003).

Common triggers for binges/purges include emotions (both positive and negative), interpersonal stressors, presence of tempting food, feeling like a dieting rule has been broken, body image dissatisfaction, and skipping meals or getting too hungry. Eating disordered behaviors often serve to distract individuals from unpleasant emotions, to comfort or soothe the individual, to numb unpleasant emotions, or to provide a sense of control. However, after binging/purging individuals typically report feeling intense guilt and shame. In more chronic cases, the binge-purge pattern is so well established it feels more like a habit rather than a response to current distress. Nonetheless, the individual becomes very distressed when they attempt to refrain from either the binge eating or the purging. Due to the high levels of distress that accompany binge/purge symptoms in addition to fears about weight, individuals with bulimia are more likely to seek treatment voluntarily than are women with AN (Fairburn & Harrison, 2003; Klein & Walsh, 2004; Polivy & Herman, 2002). However, individuals typically struggle to stop on their own for a long period of time, often as long as 5 to 10 years before they seek treatment.

Individuals with BN are typically within the normal weight range (APA, 2000; Fairburn & Harrison, 2003), though many are at the higher levels of that range—levels that they consider "overweight." Regardless of their actual weight, these individuals are either wanting to be considerably thinner or they are convinced that they will gain weight if they stop their compensatory behaviors. Thus, while they are highly motivated to stop binge eating, they are ambivalent about stopping the compensatory behaviors. While the initiation of purging behaviors may initially be associated with some weight
loss, most individuals find that this effect is not maintained, and many report some weight gain over time because the frequency of binge eating tends to escalate once individuals know they have a way to compensate. As one client stated,

*This disorder turns out to be a cruel trick. You think you have found a solution for occasional overeating. You believe you are only going to resort to purging occasionally, but you end up eating more, feeling more out of control, and purging more and more often. The solution stops working but you are afraid to stop purging. (Craighead, personal communication, 2006)*

**Medical Complications of Bulimia**

Medical complications arise most often from the purging behaviors—for example, self-induced vomiting and laxative abuse (Mitchell & Crow, 2006)—especially at high levels of use. In a moderately severe case the frequency of purging is limited to a few times a week, but in the most severe cases an individual may purge several times a day or purge after any food is eaten. Extended episodes that include multiple periods of eating and then purging are not uncommon. Persons with BN may develop fluid or electrolyte abnormalities (leading to potentially fatal arrhythmias), esophageal complications, GI symptoms, renal system problems, menstrual irregularities, thyroid dysfunction, and Russell’s sign (scarring or calluses on the top of the hands from repeatedly inducing vomiting) (APA, 2000; Klein & Walsh, 1996; Mitchell & Crow, 2006). However, many individuals show few medical indicators for extended periods of time. Dental concerns (dental enamel erosion, gum disease) and enlarged parotid glands (chipmunk-like cheeks) are often the only obvious signs of chronic vomiting. Dentists are now being trained to assess for purging when atypical dental problems are evident. Laboratory tests (salivary amalase) can be used as an indicator of inflammation of the parotid glands and positive results may mean the person is purging, but this test is not a specific indicator so it is not currently widely used. BN is very difficult to detect early in the course of the disorder if a person is motivated to hide their problem. Social difficulties related to atypical eating patterns, social isolation, unexplained absences, secretive behavior, missing food (or money), and excessive bathroom use are more often the observable signs that a person’s problems have reached clinically significant levels.

Decreased stomach motility can contribute to a patient’s complaints that they feel very full after eating normal or small amounts of food (making it more difficult to resist urges to purge). Preliminary evidence suggests that individuals with BN have blunted postprandial cholecystokinin (CCK) release, which may contribute to their difficulty feeling satisfied with normal amounts of food. Keel, Wolfe, Liddel, De Young, and Jimerson (1997) reported findings suggesting that this blunted release may be fairly specific to those who eat large amounts when they binge. Women who purged after normal to small amounts of food were not significantly lower than controls on CCK release.

Over time some individuals find certain methods of eating (or types of foods) that make it easier to self-induce vomiting. Using those strategies escalates the frequency of both bingeing and purging. Some individuals find they can now induce vomiting simply by flexing their stomach muscles. Other find that inducing vomiting becomes more and more difficult. A few individuals resort to using ipecac to induce vomiting, a method which is medically very dangerous as it can lead to irreversible damage to heart muscle tissue. Individuals with diabetes are at particular risk for medical problems as they can misuse their medication as a form of compensatory behavior. Many women with BN seek treatment for eating disorders (2006). It is no surprise that parents lack confidence in the

**Binge Eating**

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**Medical Problems**

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seek treatment from a medical doctor for physical concerns before they consider seeking psychological treatment for the disorder (Crow & Peterson, 2003; Mitchell & Crow, 2006). It is not clear if people fail to seek treatment due to stigma, reluctance to tell parents, lack of financial resources, or failure to perceive BN as a treatable problem.

**Binge Eating Disorder (BED)**

BED is diagnosed when an individual reports OBES (same as defined for BN), but no regular, inappropriate (unhealthy) compensatory behaviors that warrant a diagnosis of BN. Another difference is that overconcern about weight and shape is not a required criterion for BED, although marked distress regarding binge eating is required. Overconcern is generally reported as part of the clinical picture but this is less clearly a distinguishing aspect of the disorder as the majority of those with BED are overweight (or obese) or have appropriate concerns associated with their medical problems. Also, many do not endorse the excessively thin weight goals that are common in BN. Individuals with BED often report low levels of restrictive eating between binge episodes so they have less clearly delineated episodes of binge eating than those with BN (where the end of an episode is more clearly marked by the compensatory behavior). With BED, binges can be difficult to distinguish from normal overeating when a “grazing” pattern is present in which an individual snacks over a more extended period of time—such as all afternoon or evening—yet the overall amount consumed is objectively large and the person feels loss of control. The caloric value reported by Bartholome, Raymong, Lee, Peterson, and Warren (2006) for an objective binge episode averaged 1900 calories (about 8 cups in volume), and for a subjective binge episode about 700 calories (about 4 cups). Binge eating is most typically a long-term, chronic concern but the frequency is very reactive to situational factors and stressors. Thus, the frequency of binge eating within a given person often varies considerably over time. For these reasons, the frequency criteria for BED are slightly different from BN; binge eating must occur on average at least two days a week (rather than two episodes) for six (instead of three) months. The eating patterns of individuals with BED who are not objectively overweight more closely resemble the nonpurging subtype of BN—in that their eating is generally fairly restrictive but their compensatory behaviors (e.g., dieting and exercise) are not clearly inappropriate. These individuals may be characterized as chronic, excessively concerned/distressed dieters.

**Medical Problems Associated with BED**

Immediate medical problems associated with the binge eating specifically are relatively uncommon except for reports of gastrointestinal (GI) distress. More commonly, the medical concerns relate to the comorbid obesity. The incidence of diagnosable BED increases with an increase of weight over normal. Thus, individuals with BED are at risk for the development of obesity and its complications even if they are not currently overweight. The co-occurrence of BED with obesity raises significant treatment issues. For obese binge eaters, it is their weight that is most distressing and they are primarily motivated to seek treatment for weight loss. Many overweight individuals do not endorse loss of control so they are not diagnosable with BED, even though they may have significant health issues exacerbated by their weight status and they may eat large amounts and fail to lose weight. There are some characteristics that differentiate the nonbinge eating obese from obese binge eaters, but this distinction seems to be less
useful the more overweight the individual. Some obese individuals do not endorse loss of control while eating but acknowledge that overall they have lost control over their eating or indicate they have given up trying to control their eating. These individuals have been described as “burnt out” dieters, and they are similar to those meeting criteria for BED (who endorse loss of control during eating).

As long as the amount of food eaten is clearly large (whether this is feeling very full or grazing to excess) and the person is obese, the degree to which loss of control is reported appears to be less important—at least in terms of the treatment that is needed. In an attempt to distinguish BED from more normative types of overeating, current DSM criteria require that the binge eating in BED not only be characterized as out of control but it must also be associated with three (or more) of the following: (a) eating more rapidly than normal, (b) eating until uncomfortably full, (c) eating large amounts when not physically hungry, (d) eating alone due to embarrassment about amounts eaten, and (e) feeling disgusted, depressed, or very guilty after overeating. An individual’s experience of an OBE varies widely. Although many binges in BN are triggered by restriction—that is, waiting until quite hungry to eat—this is less common in BED. In BED, binges are more often experienced as psychologically motivated—the person may recognize that they are not hungry and that they are eating for emotional reasons. Binges may have an obvious trigger—such as a breakup or work stress—but they are often triggered by less intense feelings such as feeling generally deprived, feeling bored or lonely, or feeling unsatisfied with life. Many people downplay the significance of (or even deny) these less intense feelings and report little insight about why they binge.

In some cases binges are clearly a non-normative experience in which an individual dissociates, may eat unusual types or combinations of food, may steal or otherwise take food inappropriately, and may report no enjoyment associated with the eating. One example, identified early on by Stunkard (1959), is night eating where individuals wake from sleep but report an almost trance-like state and extreme difficulty preventing or limiting eating when food is available—yet they rarely leave the house to seek more food. They often have difficulty recalling the episode in the morning. More research is needed to understand these episodes and to determine if they require different intervention than the more normative variations of binge eating.

### Eating Disorder Not Otherwise Specified (EDNOS)

In addition to BED, the EDNOS category includes any pattern of disordered eating that is distressing and impairing but does not meet full criteria for AN or BN. Some of the examples given in DSM-IV describe what are typically labeled subclinical cases of AN or BN. However, these presentations do not differ significantly on many indices of distress and impairment so the utility of designating certain levels as subclinical has been challenged. In particular, current work (Binford & Le Grange, 2005) describing BN-like patterns among adolescents points out the importance of establishing criteria that are more appropriate for clinical presentations at that age. Intervention early in the course of eating disorders is generally more successful so it is helpful to note developmental stage when making diagnoses. The onset of subclinical BN in a 15-year-old needs to be treated urgently and will likely benefit from a different approach than a pattern of chronic, subclinical BN in a 25-year-old which reflects partial recovery from an earlier period of full BN symptoms.
Diagnostic Controversies

Wonderlich, Joiner, Keel, Williamson, and Crosby (2007) reviewed the evidence supporting the current (DSM) classification scheme and concluded that while there is substantial evidence supporting the utility of broad distinctions between AN and BN, there is little evidence to support the specific criteria that are currently in use. Furthermore, the overall scheme is limited because the EDNOS category is so broad. These authors describe current sophisticated statistical approaches that are paving the way for the development of a more empirically based classification scheme. Latent class analysis, a multivariate method that identifies subtypes of related cases, has been used to help identify different patterns as well as to determine the optimal number of categories that should be considered distinct. Their summary suggests there are at least six patterns that will be useful to study. The data support the utility of making a distinction between the two subtypes of AN and support consideration of BED as a distinct pattern, but the data provide no support for the current distinction between the subtypes of BN. Two other distinct patterns emerged: purging disorder (purging after normal to small amounts of food) and subjective binge-eating disorder (loss of control or distress associated with eating normal to small amounts of food).

Taxometric analysis is the other tool that was used to determine if a pattern represented a single underlying condition or if it represented more distinct entities. The studies cited in their review suggest that the behavior of binge eating (large amounts) appeared to be qualitatively distinct from normal (nonbinge) eating, regardless of actual weight status. Evidence also suggested that binge-purge behavior represented a qualitatively different class of behaviors than binge eating by itself. These results suggest it may be more appropriate to group the AN binge-purge subtype with the BN purging subtype, and group BN nonpurging subtype with BED. On the other hand, the evidence suggested that restriction (eating very little) was not qualitatively different from normal eating and simply represented the extreme end of a continuum that included normal eating. These new approaches to classification are significantly altering current conceptualizations about eating disorders, so we expect significant changes in the next revision of the DSM. Two alternative models for classification have already been proposed.

Three Dimensional Model. Williamson, Gleaves, and Stewart (2005) described a three-dimensional model in which binge eating is considered a categorical dimension (high versus low) while fear of fatness/compensatory behaviors and extreme drive for thinness are viewed as continuous variables. Wonderlich and his colleagues discuss two implications of this model that are particularly interesting. First, if binge eating is best viewed as a taxon—that is a separate dimension—it may be that a specific genetic vulnerability, perhaps a phenotype related to low impulse control, could be identified. Such a vulnerability could be unique to eating, but it might also show up as problematic in areas other than eating behavior. If impulsivity is implicated, psychological and pharmacological interventions might be targeted more specifically to address that mechanism. Second, if restricting behavior is best viewed as on a continuum with normal eating, problems with restriction may be better conceptualized as similar to personality disorders (since those are also thought to be extreme ends of a normal continuum of psychological traits). If that view is correct, one would expect longer term treatment might be required, and one would predict that restrictive behavior would be less likely to respond to pharmacological intervention. Within this viewpoint, the binge-purge subtype of AN might represent an individual with both difficulties—which could explain...
why this pattern seems different from BN and why it is generally less responsive to current treatments than BN. While much remains to be done, it seems likely that identifying and studying this wider range of very specific, behaviorally anchored patterns of disordered eating will promote a better understanding of the etiology and psychopathology of disordered eating. Weight status may well have treatment implications, but may have limited utility as a criterion for classification.

Transdiagnostic Model. Taking a different approach, Fairburn and colleagues (Fairburn & Bohn, 2005; Fairburn, Cooper, & Shafran, 2003) have proposed reducing current diagnostic categories to a single diagnosis, a solution that seems useful in terms of simplifying clinical diagnosis and treatment recommendations. This transdiagnostic approach is based on the argument that a similar core psychopathology—the overevaluation of eating, shape, and weight (or their control)—characterizes the entire range of disordered eating patterns. This explains the significant overlap in symptoms, the fact that many patients migrate across diagnostic categories over time and the fact that a chronic course of residual symptoms is a common outcome. Consistent with this view, Eddy et al. (2002) suggests that the pure restricting type of AN might be better viewed as a phase that typically would occur early in the course of the disorder. Effective treatment might be able to prevent its progression, but if this theory is correct patients who did not respond to intervention would be likely to progress to some type of bingeing or compensating as they fail to be able to maintain their excessive restriction over long periods of time. Consistent with this view, the average age of onset of AN is clearly younger than for BN. On the other hand, there is a subset of patients with AN who are able to maintain chronic restriction and low weight without progressing to binge eating who do not appear to fit that model. Further research is needed to determine what allows certain individuals to maintain chronic restriction long term while most cannot. If poor treatment responders could be identified early on in the course of the disorder, more targeted or more intense interventions might be developed to prevent the restriction pattern from becoming so well entrenched, and/or prevent progression to bingeing/purging.

Within the transdiagnostic view, overconcern is viewed as the core pathology that drives the restriction/dieting which leads to, and perpetuates disordered eating patterns. This pattern is quite evident in BN as overconcern drives the dieting that often triggers binge eating, as well as the compensatory behaviors when the threat of weight gain can not be tolerated. Overconcern also provides the best explanation for the patterns labeled purging disorder and subjective binge eating disorder. When normal to small amounts of food are experienced as loss of control or as requiring purging, overconcern with weight and shape would appear to be the driving force. This conceptualization is less obviously applicable to BED—as many individuals report the onset of binge eating before the onset of either weight concerns or dieting (Mussell et al., 1995). However, it is not clear that early “overeating/binge eating” would escalate to the point of diagnosable BED if it did not occur within the context of overconcern about weight and shape. Thus, overconcern may be the important factor differentiating those who simply overeat and/or become overweight from those who develop BED. The transdiagnostic approach provides a useful way to conceptualize the varied presentations of disordered eating, provides a unifying framework for treatment, and identifies overconcern as the most important target both for prevention efforts and for relapse prevention once unhealthy eating behaviors are eliminated.
Historically less responsive to treatment, it seems likely that identically anchored patterns of diet and psychopathology in anorexia nervosa. This transdiagnostic approach integrates the entire range of symptoms, the fact and time and the fact that a binge episode characterized by the overeating of the entire range of symptoms, the fact and time and the fact that a binge episode characterized by the overeating of food. Likewise, the average age at the onset of anorexia nervosa is between 16 and 18 years old. Further research is needed to identify and understand the underlying mechanisms and their role in the development of the disorder. The core pathology that underlies eating disorders is the pattern of weight gain or loss, and the related phenomena of overconcern with body image, utilization of the diagnosis of binge eating itself. However, it is important to note that there is no evidence that food is a trigger for binge eating in all cases. In some cases, it might be related to a need to relieve abdominal pain or the fear of weight gain. Patients often report guilt, remorse, and self-contempt related to their own behavior. The work broadened thinking about overeating; overeating might be due to decreased sensations of satiety (failure to stop eating) instead of (or in addition to) being due primarily to increased hunger (drive to eat).

Boskind-Lodahl and White (1973) modified this more biological view of binge eating when they published a feminist formulation of “bulimarexia,” describing a binge-purge syndrome among normal weight women. It was conceptualized as a culture-bound syndrome resulting from modern Western culture’s obsession with female thinness—which was also thought to be part of a larger picture of restricted options for women. A few years later, Russell (1979) described similar cases, which he labeled as “bulimia nervosa” and viewed as a variant of AN that was particularly difficult to treat.

As a result of this attention to difficulties related to binge eating, bulimia was added to the 3rd edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III) as a disorder distinct from AN. It was not until DSM-III-R (APA, 1987) that the term bulimia nervosa was introduced. At that point the criteria required some type of inappropriate compensatory behavior, thereby redefining large numbers of individuals with binge eating to the EDNOS category. To address this difficulty, BN was retained as a specific diagnosis in DSM-IV, but Binge Eating Disorder (BED) was added as a
provisional diagnosis. The incidence of bulimia significantly increased in the years following its identification, reaching a peak in 1996 (Curran, Schmidt, Treasure, & Jick, 2005; Keel & Klump, 2003). Interestingly, in one sample of college students the rate of BN decreased from 1982 to 2002. In this sample the prevalence of binge eating decreased over time, but the prevalence of purging remained consistent (Keel, Heatherton, Dorer, Joiner, & Zalta, 2006).

As discussed earlier, empirical support has been found for at least six patterns of disordered eating. Stunkard (1993) suggests that binge eating (i.e., BED) is the pattern that has the clearest historical roots, being observed even in early cultures. This observation is consistent with the current notion that vulnerability to binge eating might reflect a distinct taxon, probably with some genetic basis. Binge eating without compensation is still the most common presentation of disordered eating and furthermore there is less evidence of sex differences than in other presentations. Empirical studies have failed to support the differentiation of nonpurging BN from BED, also suggesting that regular eating of large amounts is likely a distinct syndrome. The degree to which a person adopts compensatory behaviors may be a separate issue, possibly more culturally determined.

The second oldest pattern (which has always had a very low base rate), Stunkard called “uninterrupted dieting,” which refers to the restricting type of AN. It is notable that the binge-purge subtype of AN appears to have emerged somewhat later in history and that recent analyses suggest it might be better grouped with BN. Patterns of regular compensation as a method of weight control—especially the currently most typical ones (vomiting and laxatives)—were not reported that often before 1960. Thus, binge-purge patterns may well constitute more culturally bound syndromes, reflecting the degree of subjectively experienced pressure to be thin. It will be instructive to see what is discovered about the two new patterns that have emerged in recent research. Will subjective binge eating disorder turn out to be a less severe (or early) form of restricting AN? What keeps some women from escalating their pursuit to more unhealthy weights? Will purging disorder turn out to be a particularly serious form of restriction since such an individual is motivated to compensate yet does not seem to be vulnerable to binge eating?

**Epidemiology**

**Prevalence**

Prevalence estimates for BN range from 1 to 3% of the population (APA, 2000; Hoek, 2006). The prevalence of partial syndrome BN (5.4%) is substantially higher than full criteria manifestations (Whitehouse, Cooper, Vize, Hill, & Vogel, 1992). Women in late adolescence or early adulthood (ages 18–24) appear to be at the highest risk for onset of BN (Currin et al., 2005; Fairburn & Harrison, 2003; Hoek & van Hoeken, 2003). Prevalence rates for BED are higher (15 to 30%) than for BN.

The vast majority (90%) of those affected by bulimia nervosa are women (APA, 2000), and the majority of patients presenting for treatment with BED are women as well. However, in community samples the rates of BED are similar for males and females, which provides some support for the hypothesis that binge eating (large amounts) may be associated with certain biological vulnerabilities while patterns involving compensatory behaviors may be more culture bound.

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**Comorbidities**

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Although eating disorders were initially thought to be more prevalent among white females from higher income families, a recent review found that binge eating and purging were equally common among white (Caucasian) females compared to minority females. However, dieting behaviors were reported to be more common in the white sample (Crago & Shisslak, 2003). This report reflects a growing body of literature suggesting that disordered eating and diagnosable eating disorders has increased in recent years among non-white races and ethnicities and among young women in all social classes and within all religions (Walcott, Pratt, & Patel, 2003). Evidence does suggest that living in (or moving to) a Western culture puts one at increased risk for the development of BN (Fairburn & Harrison, 2003; Keel & Klump, 2003).

Course

Typical age of onset for BN is late adolescence or early adulthood (APA, 2000). Studies suggest that a minority of individuals with BN—only about 6%—receive mental health treatment (Fairburn, Welch, Norman, O’Connor, & Doll, 1996; Hoek, 2006), and when treatment is sought the average length of history at presentation is about 5 years (Mitchell, Hatsukami, Eckert, & Pyle, 1985). It is not uncommon for individuals with BN to have had an earlier episode of AN: in community samples, 10 to 14% of those with BN had an earlier episode of AN (Favaro et al., 2003; Kendall, Maclean, Neale, & Kessler, 1991), whereas in clinical samples, the percentage of those with past AN increases to 25 to 37% (Braun, Sunday, & Halmi, 1994; Sullivan, Bulik, & Kendall, 1998). Herzog et al. (1999) found that over a 7 year period, 16% of women with restricting AN developed BN but only 7% of women with BN developed AN—providing some support for the argument that restricting AN may be an early phase that evolves to include binge eating or compensating.

While the age of onset of objective binge eating varies widely and is often quite young (“I can’t even remember when I didn’t binge”), the age at which an individual would meet full criteria for BED and/or would present for treatment is somewhat later than other EDs, commonly over 25. Once established, BN and BED both tend to run a chronic course—even though this may comprise a number of discrete episodes, often initiated by life transitions or stressors (APA, 2000; Fairburn, Cooper, Doll, Norman, & O’Connor, 2000; Fairburn, Stice, Cooper, Doll, Norman, & O’Connor, 2003; Quadflieg & Fichter, 2003). The chronic course of BN and high relapse rates are consistent across both clinical and community samples. In a five-year prospective study, Fairburn et al. (2000), 15% of the sample originally diagnosed with BN still met criteria at the last assessment, 32% had residual symptoms (EDNOS), and 7% met criteria for BED. Of those originally diagnosed with BED, 9% still met criteria, 12% had residual symptoms (EDNOS), and 3% met criteria for BN.

Even among those who fully recover from BN, relapse rates are high: approximately one-third relapse during follow-up periods (Herzog et al., 1999; Keel & Mitchell, 1997). There is also great flux in clinical status over time. Herzog et al. (1999) found that each year during the 5-year follow-up period, one-third remitted and an additional one-third relapsed.

Comorbidity

BN and BED both overlap with several other forms of psychopathology, including mood, anxiety, substance use, and personality disorders (O’Brien & Vincent, 2003). Mood disorders, especially major depression and dystymia, are common with
estimates ranging from 36% to 50% (O’Brien & Vincent, 2003; Williamson, Zucker, Martin, & Smeets, 2001). A recent review suggests that lifetime prevalence of anxiety disorders among those with bulimia ranges from 41% to 75% (Godart, Flamant, Perdereau, & Jeammet, 2002). Posttraumatic stress disorder was the only anxiety disorder that occurred significantly (three times) more often in BN than AN (Kaye et al., 2005). There is also a strong relationship—in the range of 30% overlap—between substance abuse and binge or purge behaviors, regardless of whether the behaviors occur within the context of AN or BN (O’Brien & Vincent, 2003) or in the context of BED (Eldridge & Agras, 1996).

In terms of Axis II disorders, borderline personality disorder is diagnosed most frequently with BN, with a prevalence rate ranging from 20–28%. However, there is overlap in the criteria as eating disturbance is considered evidence of impulsivity in diagnosing the latter. Avoidant, Dependent, Histrionic, and Paranoid Personality Disorders are also fairly common—with prevalence rates ranging from 9 to 20% each (Cassin & von Ranson, 2005; Sansone, Levitt, & Sansone, 2005). Sansone et al. (2005) reported that one-third of the cases of BED had comorbid Axis II disorders; however, Obsessive-Compulsive Personality Disorder was the most common type.

**Biological Aspects of Bulimia Nervosa and Binge Eating Disorder**

In their recent review of risk factors for eating disorders, Striegel-Moore and Bulik (2007) summarized current work on genetic factors. First, they noted that virtually all studies had been done with European populations so conclusions were correspondingly limited. Nonetheless, there is substantial evidence suggesting high heritability estimates (in the range of 50% to 80%) in twin studies in which BN has been defined in a variety of ways. Heritability effects were strong for the specific symptoms of self-induced vomiting and binge eating, but shared and unique environmental effects better accounted for the cognitive symptom of overconcern with weight and shape. Work by Keski-Rahkonen et al. (2005) showed sex differences in heritability patterns for the symptoms of drive for thinness and body-dissatisfaction. Only one study specifically investigated a syndrome approximating BED (i.e., binge eating without purging) but this study reported a fairly similar heritability estimate (41%) as found for BN. Thus a definite level of familial transmission for both these disorders has been established, but little is known about the specific genes or the mechanisms through which they could influence relevant eating behaviors. Keel, Klump, Miller, McGue, and Iacono (2005) demonstrated shared transmission between EDs and anxiety disorders, but again the basis for this similarity is not known.

In short, little is conclusive at this point in time, but simple answers—that is, main effects for genes—seem unlikely. Genetic variation may largely account for certain core symptoms (possibly binge eating or the ability to maintain low weight without binge eating). When the environment shifts (as in the emergence of the ultra thin ideal or the increased availability of high fat foods) these genetic vulnerabilities are differentially elicited, which can make the shift appear to be culturally mediated. Thus, gene by environment interaction models are most likely to be useful explanations. Current thinking also proposes that it will be more useful to investigate specific endo- and subphenotypes that have narrow effects on certain core behaviors (e.g., impulsivity) rather than attempting to identify genetic influences for specific diagnoses.

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Several specific physiological mechanisms that are believed to be involved in the regulation of eating are currently being explored. It is not clear if differences in the processes of digesting food encourage overeating because it is more difficult for certain individuals to sense satiety or if regular eating of large amounts alters digestive processes. Geliebter and Hashim (2001) found that a larger stomach capacity was specifically related to binge eating. Women with BN and binge-eating obese women had similar increased capacity compared to either normal weight or nonbinge eating obese women. Larger stomach capacity is hypothesized to contribute to lower satiety both directly and by delaying release of CCK in the duodenum. Evidence suggests that women with BN have blunted postprandial release of CCK (Pirke, Kellner, Friess, Drieg, & Fichter, 1994), and that women with BED have similar disturbances in satiety (Sysko, Devlin, Walsh, Zimmerli, & Kessel, 2007). As suggested by the significant overlap between mood disorders and eating disorders, neurotransmitters are also likely to be involved. Recent work utilizing brain imaging (Kaye et al., 2005) identified disturbances in individuals with BN and AN in specific brain areas that persisted after recovery. Dysregulation in serotonin pathways in the brain is hypothesized to influence the hedonic aspects of feeding behavior as well as its well documented effects on mood and impulse control.

Psychosocial Aspects of BN and BED

PERSONALITY TRAITS

Several personality traits have been associated with eating disorders. Some of these seem to be predisposing factors whereas others are thought to be more secondary to the eating disturbance. Methodologically speaking, research in this area varies in terms of the samples that are selected, with some studies collapsing across ED diagnostic groups, some examining one or more distinct ED groups, and others creating groups based on behavioral patterns (e.g., purging vs. nonpurging behaviors). Each type of study provides unique information and, taken together, results across multiple samples leads to a more comprehensive picture of the relation of personality traits to eating pathology. The following sections describe several personality traits with consistent and robust empirical support: impulsivity, compulsivity/obsessionality, perfectionism, sensation seeking, harm avoidance, and self-directedness.

Impulsivity

High impulsivity is commonly reported by individuals with BN, and levels of impulsivity are higher in this group compared to individual with AN or psychiatric controls (Cassin & von Ranson, 2005; Engel et al., 2005). Evidence suggests that impulsive behavior is related to purging—be it in the context of AN or BN—and is associated with higher levels of psychological and eating disordered symptomatology (Favaro et al., 2005). Importantly, evidence suggests that degree of impulsivity abates with recovery, suggesting that the high levels of impulsivity during an episode of BN might be due, in part, to the uncontrolled eating behaviors and associated emotional instability (Cassin & von Ranson, 2005).
Compulsivity and Obsessionality

Obsessive/compulsive features tend to be common in women with eating disorders, both in university and clinical samples (Cassin & von Ranson, 2005). These traits tend to be present even after recovery from the eating disorder (Cassin & von Ranson, 2005; Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006), suggesting that they are not simply a concomitant of the eating pathology. A recent study (Engel et al., 2005) examined the prevalence and effects of both impulsivity and compulsivity in women with BN. Women with high levels of both impulsivity and compulsivity exhibited the highest levels of impairment and psychopathology (in terms of comorbid personality disorders, substance use, eating pathology, and depression), while those women with low levels of both impulsivity and compulsivity demonstrated the lowest levels of functional impairment and comorbid psychopathology. Women who scored high on measures of impulsivity and low on measures of compulsivity had the highest levels of drug and alcohol related problems, while the reverse pattern (low impulsivity and high compulsivity) was associated with higher levels of eating disordered and depressive symptoms.

Perfectionism

Important facets of perfectionism include high expectations of one’s own performance, self-criticism, belief that others critically evaluate one, fear of failure, and excessive concern over mistakes (Cassin & von Ranson, 2005; Fairburn et al., 2003; Franco-Paredes, Mancilla-Diaz, Vazquez-Arevalo, Lopez-Aguilar, & Alvarez-Rayon, 2005). Perfectionism has been noted as a risk factor for bulimic pathology and a maintenance factor for more general eating pathology (Stice, 2002). Based on a multidimensional assessment of perfectionism, individuals with BN have elevated levels of perfectionism in both the acute and recovery phases of the disorder (Cassin & von Ranson, 2005; Franco-Paredes et al., 2005). Recent studies suggest that perfectionism combines with low self-efficacy and perceived overweight to predict subsequent onset of eating disordered symptoms (Bardone-Cone, Abramson, Vohs, Heatherton, & Joiner, 2006; Lilenfeld et al., 2006). Interestingly, associations with perfectionism were strongest for fasting and purging and the relationship between perfectionism and binge eating was mediated by fasting (Forbush, Heatherton, & Keel, 2007). As a testament to its important role in eating pathology, perfectionism is one of four key maintaining factors described in the transdiagnostic cognitive-behavioral model of eating disorders (Fairburn, Cooper, et al., 2003).

Other Traits

Three additional personality characteristics have shown some promise and warrant further investigation: sensation or novelty seeking, harm avoidance, and self-directedness. Individuals with binge-purge variants of eating disorders report higher levels of sensation seeking or novelty seeking when compared to either individuals with restricting eating disorder (Cassin & von Ranson, 2005; Favaro et al., 2005; Vervaeet, van Heerlingen, & Audenaert, 2004) or controls (Cassin & von Ranson, 2005). Individuals with BN also report high levels of harm avoidance (Berg, Crosby, Wonderlich, & Hawley, 2000; Cassin & von Ranson, 2005), and when compared to the restrictive subtype, individuals who binge and purge tend to score low in self-directedness (Cassin & von Ranson, 2005; Vervaeet et al., 2004).

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Emotion Disregulation

Women with eating disorders typically report intense mood states and increased mood lability. Several studies have examined the association between negative affect and eating disorder symptoms, as well as the interaction between negative affect and other known risk factors for eating pathology. There is strong support for a relationship between negative affect and bulimic symptoms, such that increased levels of neuroticism or negative affect prospectively predict onset of eating pathology (Leon, Fulkerson, Perry, Keel, & Klump, 1999; Lilenfeld et al., 2006; Stice, Akutagawa, Gagger, & Agras, 2000). In addition to increasing the risk of eating disorders, negative affect maintains binge eating in individuals who struggle with disordered eating (Stice et al., 2000) and the elevated levels of negative affect seem to persist even after recovery from the eating disorder (Lilenfeld et al., 2006). Negative affect is also related to other eating disorder risk factors, suggesting that negative affect might impact eating pathology through multiple pathways. Research suggests that negative affect mediates the relationship between body dissatisfaction and bulimic symptoms (Sim & Zeman, 2005; Stice, Nemeroff, & Shaw, 1996) and that it moderates the relationship between dieting and binge eating (Stice et al., 2000). In addition, increases in negative affect lead to increased body dissatisfaction (Stice, 2002).

Binge eating and purging behaviors (as well as other ED behaviors) have been conceptualized as strategies to regulate emotions. Women with eating disorders have a hard time identifying their feelings, are unsure about how to cope with emotions, and have difficulty tolerating adverse mood states (Fairburn, Cooper, & Shafran, 2003; Sim & Zeman, 2005). To manage these difficulties in emotion regulation they turn to bingeing and/or purging as a way to distract from or modulate negative feelings. Conversely, negative affect has also been found to exacerbate ED symptoms (Polivy & Herman, 2002), often leading to a downward spiral of increased negative affect and increased eating pathology. From a behavioral perspective, the binge-purge episodes become highly reinforcing. In the moment, binge eating decreases negative affect, which serves to negatively reinforce the behavior (Deaver et al., 2003). However, this momentary relief is soon followed by feelings of guilt, shame, and anxiety about having binged (Corstorphine, Waller, Ohanian, & Baker, 2006). The original negative emotions are then replaced by negative feelings centered on eating behavior and body shape and weight concerns, presumably an area with more perceived control. In the context of BN, compensatory behaviors arise to reduce the negative feelings about having binged. In this manner, compensatory behaviors are negatively reinforced since they help to mitigate unpleasant emotions and decrease sensations of fullness, which often feel distressing (Corstorphine et al., 2006; Williamson, White, York-Crowe, & Stewart, 2004). The purging behavior is also positively reinforced since it increases levels of happiness and relief (Corstorphine et al., 2006). The entire binge-purge episode is reinforced because it serves to control emotions that otherwise feel overwhelming.

Cognitive Dysfunction

Cognitive manifestations of BN can be organized into four main areas: (a) appearance overvaluation, (b) internalization of the thin ideal, (c) cognitive biases, and (d) rigid and obsessive thinking patterns. As mentioned previously, appearance overvaluation is one...
of the diagnostic criteria for BN, occurring when an individual’s self-worth is disproportionately affected by physical attributes such as body shape and weight (Williamson et al., 2004; Fairburn, Cooper, et al., 2003; Polivy & Herman, 2002). Focus becomes narrowed on eating and control over eating as a means to achieve the desired physical outcome. Appearance overvaluation prospectively predicts later onset of eating disordered symptoms (Lilenfeld et al., 2006; Stice, Presnell, & Spangler, 2002), and overvaluation of eating, weight, and shape has been implicated as the central maintaining feature in eating pathology (Fairburn, Cooper, et al., 2003).

Current Western culture endorses an ultra-thin ideal for women’s body shape and size. Thin-ideal internalization is the degree to which an individual adopts and subscribes to this cultural standard. Internalization of the thin ideal is hypothesized to increase body dissatisfaction, since women who highly value a thin ideal are more likely to be dissatisfied with their weight and shape and are more likely to engage in behaviors to help them achieve the thin ideal. Research confirms that a high level of thin ideal internalization is a risk factor for body dissatisfaction, dieting, and negative affect (Stice, 2002). Internalization of the thin ideal also prospectively predicts later onset of binge eating and bulimic pathology, as well as maintaining bulimic pathology (Stice, 2002).

Women with eating disorders exhibit cognitive biases when processing information related to food and to weight and shape; and evidence suggests that these biases affect both attention and memory processes. Women with eating disorders tend to pay increased attention to stimuli that are eating, shape, or weight related. In addition, such individuals are better able to encode and recall eating, weight, and shape related information in comparison to other categories of information. Another manifestation of cognitive bias is overestimation of body shape and size, which leads to increased body dissatisfaction and increased desire to lose weight (Polivy & Herman, 2002; Williamson et al., 2004). These biased ways of thinking are thought to increase negative affect; conversely, increases in negative affect are thought to increase the biases in information processing (Williamson, White, York-Crowe, & Stewart, 2005). Importantly, some evidence suggests that the biased thinking might be more a result of—rather than a cause of—the eating disorder (Polivy & Herman, 2007).

Women with BN demonstrate a thinking pattern that tends to be characterized by obsessive thought patterns and rigid, dichotomous thinking. The obsessive thought patterns manifest as high levels of preoccupation with eating, weight, and shape (Cooper, 2005; Polivy & Herman, 2002). Rigid and dichotomous thinking leads to categorization of food as good or bad, as well as leading to very inflexible expectations for eating behaviors (and often for life in general). Dichotomous thinking is hypothesized to give rise to the Abstinence Violation Effect. Individuals have rigid dietary rules and when they break one of these rules even to a small extent, they feel like they have blown it completely so there is no reason to try further. Thus, small slips become full binges.

Cooper, Todd, and Wells (2004) described four categories of automatic thoughts that result in and maintain bulimic pathology: (a) thoughts of no control (e.g., I will never be able to stop eating if I have that piece of candy), (b) permissive thoughts (e.g., buying a whole bag of bagels really is not that big a deal; it won’t hurt), (c) positive thoughts (e.g., eating the pizza will make me feel much better), and (d) negative thoughts (e.g., I am going to gain a ton of weight). The positive thoughts are hypothesized to promote binge eating, while the negative thoughts encourage compensatory behaviors.

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SELF-WORTH IS DISPROPORTIONATELY COMPLICATED (Wilkinson 2002). Focus becomes distorted by the desired physical state of eating disorders, and overcompensation can lead to increased body dissatisfaction and restrictive behaviors to help maintain a thinner ideal internalization (Stice, 2002). Thus, increased sociocultural pressure to be thin and elevated thin ideal internalization lead to increases in body dissatisfaction (Stice, 2002) which directly increases eating pathology.

**Body Dissatisfaction**

Body dissatisfaction is a consistent and robust risk factor for eating pathology, as well as a maintenance factor (Lilenfeld et al., 2006; Stice, 2002; Young, Clopton, & Bleckley, 2004). In addition to its direct impact on eating pathology, body dissatisfaction leads to dieting and negative affect, which in turn further impact eating disorder symptoms (Stice, 2002; Stice & Shaw, 2002). It is also hypothesized that other causal factors impact eating pathology through body dissatisfaction (Polivy & Herman, 2002). For example, increased sociocultural pressure to be thin and elevated thin ideal internalization lead to increases in body dissatisfaction (Stice, 2002) which directly increases eating pathology.

**Dieting**

Elevated dieting and dietary restraint prospectively predict binge eating onset, level of bulimic pathology, and negative affect (Jacobi, Hayward, de Zwann, Kraemer, & Agras, 2004; Stice, 2002; Stice et al., 2002). In addition, self-reported dieting is a maintenance factor for bulimic pathology (Stice, 2002), where binge eating arises in response to rigid, stringent efforts at dietary control (Fairburn, Cooper, et al., 2003). In fact, many individuals with BN cycle between restrictive eating and binge-purge behaviors. Evidence suggests that dieting may result, in part, from body dissatisfaction (Polivy & Herman, 2002; Stice, 2002), where it serves as an attempt to fix the weight and body problem. More recent studies demonstrate that successful caloric deprivation (dieting) does not lead to increased eating disorder symptoms (Stice, 2002), suggesting that unsuccessful dieting or struggles with dieting may be more problematic than actual dieting, per se. However, excessively restrictive dieting increases the likelihood of struggle and can trigger binge eating, so extreme dieting is problematic in its own right. However, the level of restriction that is experienced as problematic (difficult) appears to vary widely among individuals making it hard to identify a particular point at which dieting becomes more harmful than helpful.

**Interoceptive Awareness**

Interoceptive awareness refers to the ability to identify internal sensations, with regard to both physiological and emotional states. Women with eating disorders report poor interoceptive awareness (Jacobi, Hayward, de Zwann, Kraemer, & Agras, 2004), and this inability to identify internal sensations prospectively predicts onset of eating disorder symptoms (Kilgen et al., 1996; Leon, Fulkerson, Perry, & Early-Zaid, 1995). Some research suggests that even after recovery these women continue to struggle in identifying their internal states (Lilenfeld et al., 2006). However, specific intervention to train appetite awareness has been shown to improve awareness of internal hunger and satiety cues.

**Body Mass**

Individuals with higher premorbid body mass were found to be at increased risk for perceived pressure to be thin, body dissatisfaction, and dieting (Stice, 2002). However, higher initial body mass did not predict either negative affect or eating pathology (Stice, 2002). Hence, it seems that body mass influences other risk factors to eating pathology, rather than directly being a risk factor itself.
Eating Disorders: Bulimia Nervosa and Binge Eating

Childhood Sexual Abuse/Stress

Physical and sexual abuse have been hypothesized to be significant risk factors for disordered eating. However, recent evidence suggests that childhood abuse is a risk factor for general psychopathology, as opposed to a risk factor specific to eating disorders (Hund & Espelage, 2005; Katenaohl, Burge, & Kellogg, 2005; Moyer, DiPietro, Merkowizt, & Stunkard, 1997; Schmidt, 2003). In a similar vein, Striegel-Moore et al. (2007) reported that elevated levels of perceived stress prior to the age of 14 constituted one pathway to binge eating (BN and BED), but this pathway only accounted for 13% of the cases, and no specific stressors were detected. Thus, the etiology of BN as well as BED likely involves multiple pathways, some of which include childhood abuse and other early stressors.

Environmental Factors

Sociocultural Pressure to be Thin

With the obvious deluge of images of overly thin women in the mass media, one misconception is that eating disorders are largely due to misguided efforts (excessive dieting) to attain our culture’s thin ideal. As just noted, it has been difficult to establish the degree to which dieting is a direct risk factor. Similarly, efforts to document a clear causal relationship between the level of media exposure and eating disorders have not shown consistent, strong effects. A more pervasive or indirect transmission of the thin ideal may be occurring because research does more clearly indicate that individuals who immigrate to a Western culture and those living in relatively more urban than rural areas are at increased risk for eating disorders (Becker, Keel, Anderson-Fry, & Thomas, 2004). Two points are worth noting here. First, individuals clearly differ in the extent to which they personally adopt the thin ideal. The degree of internalization of the thin ideal and the degree to which a person personally feels pressured by others to be thin appear to be more important than simply degree of media exposure (Stice, Presnell, & Spangler, 2002). Furthermore, while media exposure appears to increase risk of body dissatisfaction and subsequent dieting as well as binge eating, media exposure appears to play less of a role in maintaining already established eating disorder symptoms (Stice, 2002).

Family

The family may serve as one of the important ways that the thin-ideal is transmitted and internalized. The attitudes and behaviors of family members and peers (e.g., feeding patterns, critical comments, weight-related teasing, modeling of restrictive eating or other disordered eating behaviors)—seem to be stronger influences than the less personal media exposure. It is also useful to note that parental obesity is a risk factor for eating disorders (Jacobi et al., 2004). While this could be a result of shared genetic vulnerability to obesity (or impulsivity), it may also reflect more subtle environmental influences. Individuals with overweight family members are more likely to have been exposed to negative societal attitudes towards obesity (stigma), and thus may impose excessive pressure on themselves. Similarly, parents who are overweight may exert excessive and unhelpful pressure on children to be thin as they hope to prevent them from developing environment skills to build with BN he positive effects to maintain an national eating habits.

Assessment

Currently, a variety of diagnostic instruments are available to use in the assessment of eating disorders. The Eating Disorder Examination (EDE) (Fairburn & Beglin, 1994) is a commonly used tool to assess eating disorder symptoms. It is a 36-item scale that measures past four weeks' eating behavior and includes questions about attitudes, dieting, and binge eating. The EDE is widely used in research and clinical settings and is considered to be a valid and reliable measure.

A number of factors can influence the development of eating disorders, including sociocultural pressures, family dynamics, and psychological factors. Understanding these factors can help in the assessment and treatment of eating disorders. Developing effective interventions that address these underlying issues is crucial for improving outcomes for individuals with eating disorders.
risk factors for dism-buse is a risk factor to eating disorders; Moyer, DiPietro, Striegel-Moore et al. of 14 constituted accounted for 13% of BN as well as childhood abuse and developing weight problems. Beyond transmitting the pressure of the thin ideal, family environment may also play a role by failing to teach adaptive eating behavior or coping skills to build self-esteem (which would reduce risk for EDs). Families of individuals with BN have been described as more chaotic, conflicted, and critical, and as low in positive expressiveness, cohesion, and caring. A negative family environment would increase anxiety and depression which would make individuals more vulnerable to emotional eating and therefore possible development of obesity or EDs.

Assessment

Currently, the most widely used assessment for disordered eating behaviors (across all diagnoses) is the semistructured interviewer-administered Eating Disorder Examination (EDE) (Fairburn & Cooper, 1993). It primarily assesses attitudes and behaviors over the past four weeks and takes about an hour to administer. Such an assessment is important to use in the research context where highly reliable diagnoses are required, but it can be useful in a clinical context because the interview format allows for greater clarification of complex eating disorder concepts that use terms (e.g., binge eating and overconcern about weight) about which there is less agreement. Furthermore, the interview insures that many important constructs are assessed which might not be elicited in an unstructured format due to the secrecy and shame associated with many of the behaviors. However, the training required to achieve reliability and the burden for staff and patients is sufficiently high that briefer methods are more typically used in clinical work.

A 36 item self-report version (EDE-Q) (Fairburn & Beglin, 1994) provides the basis for a possible diagnosis as well as the same four subscales (continuous scores for restraint, eating concern, shape concern, and weight concern) as the EDE. However, a recent factor analysis suggested that a three-factor solution is more appropriate for the EDE-Q (Peterson et al., 2007). Correlations between the interview and self-report versions and between change on the two measures were reasonable (Sysko et al., 2007) even though within individuals discrepancies were common, particularly in terms of frequency of types of binge episodes. It is not clear if clients are more willing to disclose information in a self-report or if an interviewer is more likely to elicit accurate information. Regardless, more objective (interviewer) ratings and more subjective/self reports of constructs such as overconcern and distress each convey useful information.

A number of other self-reports are widely used. The more global, continuous measures provide indices of degree of improvement that can be useful to provide a broader picture than simple frequency counts of specific behaviors. Many of these measures contain multiple subscales that attempt to capture the wide range of important constructs associated with disordered eating. The Eating Disorder Inventory (EDI-3) (Garner, 2005) and the Bulimia Test Revised (Thelen, Farmer, Wonderlich and Smith, 1991) are most commonly used in assessing outcome. In addition, very specific self-reports are available to assess narrower constructs that may be of interest—such as specific cognitions, body dissatisfaction, restraint, dietary intent, or food avoidance (see review by Anderson & Paulosky, 2004). Body checking is a specific construct that has only recently been identified as an important aspect of disordered eating that needs to be assessed. The Body Checking Questionnaire (Reas, Whisenhunt & Netemeyer, 2002) is a 23 item self-report of the frequency and nature of those behaviors.
Assessment of eating disorders in children and adolescents is not as well developed, but several inventories and interviews are available. The Children’s Eating Attitudes Test (Ch-EAT) (Garner and Garfinkle, 1979) has been used and the use of a shorter, 26-item version was reported by Wallin, Kronovall and Majewski (2000). Shapiro et al.’s (2007) report on the Children’s Binge Eating Disorder Scale, a brief structured interview, suggests that this type of assessment would be more appropriate if one’s purpose was just to assess the degree to which overeating had become problematic for a child.

Treatment outcomes in BN and BED trials are reported in a number of ways. Sometimes successful outcome is defined as percent reduction of the core behaviors or as no longer meeting criteria for the disorder, which allows for some residual level of symptoms. Often, outcome is reported in terms of abstinence from the core symptoms, generally defined as no binges or purges in the past four weeks as assessed by the EDE. This time frame is needed to determine abstinence because eating behaviors tend to fluctuate over time and a week or two is not necessarily representative. If used to evaluate treatment outcome, the four-week time frame should be the four weeks after the end of treatment rather than the last four weeks of treatment. Abstinence may be a bit too strict a criteria, as many individuals improve dramatically but do not become abstinent. However, there is support for abstinence being an important outcome. Individuals who still have residual symptoms at the end of treatment generally fare more poorly during follow-up than those who become abstinent by the end of treatment.

Abstinence from purging is relatively easy to assess, but abstinence from binge eating can be more problematic and thus may not be as useful as an outcome measure. Elimination of clear OBES is usually obvious, but the line between OBES and SBEs typically becomes even more blurred after treatment as the size of OBES is often greatly diminished. This issue is sometimes addressed in BN studies by simply reporting binges (i.e., combining OBES and SBEs) but this can also be misleading as SBEs don’t count as diagnostic criteria. Eliminating SBEs that lead to purging is clearly important in BN. However, abstinence from SBEs in the absence of purging may be too strict a criterion to be useful. Clients may not clearly endorse loss of control, especially after treatment. Some clients describe having “bingey” feelings—episodes that do not quite feel normal but do not involve really large amounts and not clear loss of control. Clinically, it is useful to have clients continue to monitor and report such feelings even though the obvious OBES (and purges) may have been eliminated. Residual “urges to binge and/or urges to purge,” are useful to monitor as they can indicate the need to continue with treatment until the urges become infrequent.

In addition to self-reports, daily self-monitoring records (of food, binges and purges) are frequently an important aspect of assessment as well as treatment. Self-monitoring avoids the difficulty of retrospective recall, and has greater validity in terms of specific behaviors. However, monitoring does not provide as useful an index of the more global attitudes of the person. Thus, both monitoring and self-reports continue to be important. Monitoring of appetite rather than food intake (Craighead, 2006) is a relatively recent development that can provide useful indices of clinically relevant variables. For example, episodes can be identified as hunger violations (waited until very hungry to start eating), satiety violations (continued to eat past moderate sensations of stomach fullness), or both. This kind of specific assessment of behavioral patterns may be particularly useful in treatment planning and evaluating response to treatment.

Treatment

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INTERVENTIONS FOR BULIMIA NERVOSA

Psychosocial and pharmacological treatments are available that substantially reduce the core problem behaviors associated with BN and BED. However, only about half of the individuals treated for BN achieve (and maintain) full remission of their primary symptoms (binge eating and unhealthy compensatory behavior). Though much improved, many patients show a chronic course of residual symptoms—that is, restrictive eating patterns, preoccupation with eating and food, and impaired social functioning due to those issues. Negative body image tends to persist as most individuals fail to achieve a weight that is psychologically satisfactory to them or they remain dissatisfied with aspects of their shape. While a higher percentage of individuals with BED (70–80%) achieve remission from their primary symptom (objectively large binge episodes), very few are able to lose substantial weight. Since most of these individuals are objectively overweight and have health as well as body image concerns, most are not totally satisfied with the outcome of treatment. Thus, more effective ways to address issues related to residual restrictive eating, weight goals, and negative body image remains a high priority.

A substantial number of reasonably powered clinical trials for BN have been reported (see review by Wilson, Grilo, & Vitousek, 2007). These have clearly established cognitive behavior therapy (CBT) as the first line treatment, either alone or in combination with medications. Interpersonal psychotherapy (IPT) is a second established option. Initial outcome is not as positive, but individuals receiving IPT continue to show improvement over the course of follow-up so that by one year follow-up those individuals are not significantly different from those who received CBT.

Several antidepressants have demonstrated overall effectiveness equivalent to CBT, but an even smaller proportion of individuals (20%) achieve abstinence by the end of treatment and relapse following medication withdrawal is a significant problem (see Mitchell, Agras, & Wonderlich, 2007). Medication is generally equally effective whether or not the individual has diagnosable comorbid depression, therefore the mechanism through which it works remains unclear. Evidence suggests that medication enhances restraint while CBT clearly works through reducing restraint—which may account for some of the difficulties with relapse following medication withdrawal. Thus, medication alone is not currently recommended for BN, but medication combined with CBT is recommended when comorbid depression is present, and medication may be useful to add if an individual does not respond adequately to CBT alone.

Clinical trials have been almost exclusively done with young adult women, but what is known about treatment for males or older women suggests that the CBT treatment effect is quite robust and does not require significant modifications for other adult populations. Recent work by Lock (2005) and others (Schapman, Lock, & Couturier, 2006) demonstrates that CBT can be successfully adapted for adolescents. Similarly, Le Grange, Crosby, Rathouz, & Levanthal (2007) have reported positive results for their adaptation of family-based therapy for AN to address BN in adolescents.

In clinical trials, CBT has been provided as individual (or group) outpatient therapy, most typically 12 to 24 sessions. In clinical practice, the format and time frame vary. Three elements form the core of CBT. First, clients engage in daily self-monitoring based on what they are eating and when, to encourage careful examination of their eating patterns. Second, clients are strongly encouraged to adopt a structured plan of three
meals and two snacks a day to replace the chaotic and unhelpful restraint strategies they have developed in their struggle to restrict intake. Third, clients learn to utilize behavioral analysis and problem-solving to develop effective alternatives for bingeing and purging and to do cognitive challenging to address dysfunctional thought patterns and their underlying overconcern with weight and shape. For patients who respond to CBT, rapid change in symptoms over the first six to eight weeks is reported. Then, therapy may address more individualized concerns and plans to prevent relapse are put into place.

To address the problem of partial response, Fairburn and colleagues (Fairburn, Cooper, et al., 2003) developed (and are currently evaluating) an extended CBT model in which four specific mechanisms that may be impeding treatment are directly addressed: severe clinical perfectionism, unconditioned and pervasive low self-esteem, and significant difficulties with either mood regulation or interpersonal relationships. This version of CBT stems from the transdiagnostic model and is being applied to all variants of eating disorders—including BED.

Several other modifications of CBT have been developed and are currently being evaluated. Preliminary data suggest these are viable alternatives, providing therapists with options that may be more acceptable or effective with some clients. Safer, Telch, and Agras (2001) reported on the use of dialectical behavior therapy (DBT)—which directly targets the emotion disregulation hypothesized to trigger many binge-purge episodes. Dicker and Craighead (2004) and Hill (2007) reported on the use of appetite awareness training (Craighead, 2006), an approach providing specific training to focus on internal cues—particularly stomach fullness. In this approach, food monitoring is replaced with self-monitoring of hunger and fullness to shift client attention away from their typical overfocus on food type. Individuals with a range of disordered eating patterns have rated appetite monitoring as significantly more helpful and acceptable than monitoring food.

To address issues of accessibility and affordability, several CBT-based versions of self-help have been developed and evaluated (Mitchell, Agras, & Wonderlich, 2007). Generally, results from these studies indicate that self-help is somewhat (but not significantly) less effective than traditional therapy—abstinence is lower and drop-out higher. However, self-help is clearly more cost effective. Guided self-help fares somewhat better than pure self-help, and guidance provided by a mental health professional is generally more effective than guidance provided by other professionals. Thus, guided self-help appears to be a positive and cost effective first step within a stepped care model of treatment, but it does not appear to be adequate to achieve remission for the majority of individuals with BN.

Perhaps the most important outcome from the many studies of CBT for BN is the observation that change early in treatment is the best predictor of ultimate outcome. Current treatments are quite effective for about half of the clients, so the field must now turn its attention to predicting the poor and partial responders. Currently, the only reliable predictor of poor response is initial severity. Dicker and Craighead (2004) reported that baseline purging once a day or more best differentiated their partial and poor responders from those who attained abstinence by the end of treatment. Other studies show that extending weekly outpatient treatment beyond 24 weeks is of limited benefit in getting partial responders to remission. Similarly, individuals who fail to respond to their initially assigned therapy are not generally helped significantly by switching to different approaches. Thus, more intensive and comprehensive treatments are being developed. Longer term help those...
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Interventions for Binge Eating Disorder

Most of the treatments that have been used to treat BN have also been applied to BED (Brownley, Berkman, Sedway, Lohr, & Bulik, 2007). Interestingly, CBT does not have the short-term advantage over interpersonal psychosocial therapy as it does for BN. Various types of cognitive behavioral therapy such as appetite awareness training, and dialectical behavior therapy all produce similar, quite large reductions in frequency of binge eating when applied to BED, but none have led to significant weight loss (on average). It is not clear why this is the case. If the calories previously consumed as binges are just distributed more appropriately throughout the day in meals and snacks, eating may not feel out of control, but the overall intake may not be reduced adequately to trigger weight loss. Across treatments, remission rates—defined as abstinence from binge eating (generally no OBEs in the past month)—range from about 70 to 85%. It is not clear if this general effectiveness across interventions is more of a placebo response, or structured CBT has less of an advantage because its primary mechanism is reduction of restraint. Since individuals with BED already have lower levels of restraint than those with BN perhaps the more critical aspect of treating BED is reducing emotional triggers for binge eating. All of the interventions may accomplish this in different ways.

Since a variety of interventions appear to be viable for treating BED, client preference and therapist training/availability may dictate the preferred treatment approach. Many clients with BED respond positively to the structure of CBT but a substantial subgroup, particularly the burned out dieters, appear to respond more positively to alternatives that do not require food monitoring. Craighead, Elder, Niemeier, and Pung (2002) found that about a third of their participants with BED assigned to appetite monitoring indicated they would not be willing to monitor food. Similarly, it may turn out that certain clients respond better to the explicit focus on emotions and interpersonal issues that form the core of interventions such as DBT and IPT. An internet-based self-help program developed by Shapiro et al. (2007) has shown promise as a highly accessible self-help option. Such a program has great potential to provide cost effective interactive support.
Eating Disorders: Bulimia Nervosa and Binge Eating

For BED with comorbid obesity, behavioral weight loss (BWL) is another viable option, but the most recent direct comparison to CBT found the modest initial weight loss shown in BWL did not continue. By one year the effects of the two treatments were not different on either binges or weight (Munsch et al., 2007). However, as found in earlier studies, individuals who had achieved abstinence from binge eating lost more weight than those who continued some binge eating. The concern in using BWL was that it might increase bingeing as it is designed to increase restriction (in the sense of increasing awareness of, and accountability for, limiting overall caloric intake). However, both BWL and CBT promote the same structure (a regular three meal and two snack pattern). Thus, it appears that restriction/dieting in this way (eating smaller amounts but eating often) is not problematic and in fact may be helpful. Notably, BWL has typically been provided in a group format, both for reasons of cost effectiveness and to provide social support for lifestyle change. Fairburn and colleagues (Cooper, Fairburn, & Hawker, 2004) hypothesize that behavioral treatment provided in individual sessions is likely to be more effective for weight loss, regardless of the presence or absence of binge eating. If their current study evaluating individual therapy for weight loss supports the greater effectiveness of that approach, individual BWL may turn out to be the first line treatment for BED with comorbid obesity.

It is useful to note that, if not treated, BED is characterized by a chronic but fluctuating course (often linked to life stressors). Thus, although all interventions seem to be equally effective in the short run, it will be particularly important for future research to examine extended follow-up to determine if some interventions fare better over the long run. However, obesity is now being conceptualized as a chronic medical problem that is likely to need extended or intermittent intervention over a long period of time. Therefore, BED with obesity is likely to need the same. Flexible, long term access to treatment may be needed to help individuals maintain treatment gains (both binge abstinence and weight loss), especially through periods of crisis or high stress.

As with BN, several classes of antidepressant medications have shown similar effectiveness as psychotherapy in the treatment of BED. Again, there is little evidence regarding mechanisms of action, but anti-depressants appear to increase restraint in BN so that is likely to be the mechanism. Recent work investigating the effects of topiramate (a mood stabilizer) suggests a promising new approach. This medication is hypothesized to reduce binge eating by moderating general tendencies toward impulsivity. Initial studies on BED with comorbid obesity reported topiramate was associated with modest weight loss in addition to significant reduction of binge eating (about 58% abstinence) but rates of medication discontinuation were 30% (McElroy et al., 2007). This percent abstinence from binge eating is not quite as high as reported for most psychosocial interventions. Given the genetic vulnerabilities hypothesized to affect binge eating as well as weight, it is highly likely that treatments combining biological and psychosocial interventions will ultimately be most effective for BED.

Prevention of Eating Disorders

There has been an explosion of eating disorder prevention research over the past decade, with the publication of over 50 distinct investigations (Neumark-Sztainer et al., 2006). Two recent meta-analyses (Fingeret, Warren, Cepeda-Benito, & Gleaves, 2006; Stice & Shaw, 2004) have synthesized relevant literature and key findings from these reviews which selected programs had and eating sizes for g concluded the follow: (versus educationa authors by single sess time to co tween sess repeated s reported tl genic effect purely psy so that issu

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reviews will be delineated. Approximately two-thirds of the programs targeted a college-aged population, while the remaining targeted high school students. Most of the participants were female. The majority of studies targeting college students were selected programs in that they targeted high-risk individuals. Overall, prevention programs had a positive impact on knowledge, established risk factors for eating pathology, and eating pathology itself. The effect size for knowledge was large, while the effects sizes for general eating pathology and dieting were in the small range. Both reviews concluded that targeted and selected prevention programs had a larger impact than universal prevention programs. Stice and Shaw (2004) reasoned that high-risk individuals (who would be targeted by selected prevention programs) and older individuals might have more subjective distress and might thereby have been more motivated to engage in the preventive interventions.

Although both meta-analyses converged in certain findings, each study also contributed unique perspectives. Stice and Shaw (2004) found that, in addition to risk status, the following characteristics strengthened the impact of preventive programs: interactive (versus didactic), multisession, females only, participants over age 15, no psychoeducational content, and trials that used validated measures (Stice & Shaw, 2004). The authors hypothesized that programs with multiple sessions were more impactful than single session programs because participants in the multisession programs had more time to consolidate new knowledge and skills, and were able to try out new skills between sessions and problem solve at later sessions. The authors also raised the idea that repeated sessions may be helpful due to increased social support. Fingar et al. (2006) reported there was no evidence to suggest that preventive interventions produce iatrogenic effects. Secondly, with regard to type of intervention, those researchers found that purely psychoeducational approaches were just as effective as skills-based approaches so that issue of the most effective content requires further investigation.

Summary and Future Directions

Extensive research is summarized describing the differences in symptoms, course, prevalence, etiology, and response to treatment for the two patterns of disordered eating that involve episodes of objectively large binge eating, Bulimia Nervosa, and Binge Eating Disorder. Recent empirical approaches to classification confirm that objective binge eating and compensatory behaviors each constitute a distinct class of behavior while restrictive eating is better conceptualized as the end of the continuum of normal eating. This approach to classifying eating disorders by focusing on specific behavioral patterns and considering weight as a separate, primarily medical concern is likely to be useful as we seek to better understand the genetic vulnerabilities, and the interactions between those vulnerabilities and the environment that result in the development of multiple pathways to disordered eating. Since weight is influenced by numerous other factors besides eating behavior—such as exercise behavior and possible biological determinants of weight that are not yet fully understood, it is not particularly surprising that attempting to include weight as a diagnostic criteria introduces unhelpful variance if one’s goal is to understand behavioral patterns. Weight status (either under or over) must of course be taken into consideration with treatment recommendations, but identifying specific behavioral patterns may be more helpful in understanding different etiologies—and perhaps genetic vulnerabilities associated with various behavioral patterns.
Since current empirical approaches suggest that binge-purge behaviors constitute a distinct typology regardless of weight status, the current BN and AN binge-purge subtypes may be better treated as one category while the BN nonpurging type may best be grouped with BED (neither includes specific purging behaviors). Current evidence also suggests that the nonbinge-obese are more similar to normal weight individuals than to the binge-eating obese on a number of psychological and biological variables—suggesting it would not be most useful to limit the diagnosis of BED to those who are overweight, but instead to consider obesity as a separate, comorbid condition that may need to be addressed. However, the frequency of binge eating increases with degree of overweight so making a distinction between binge eating and significant "over" (but not binge) eating is less likely to be useful the more overweight the individual is.

Making a distinction between objective and subjective binge eating remains one of the most difficult and controversial aspects of assigning current eating disorder diagnoses. However, empirical approaches to classification tend to uphold the utility of identifying regular eating of clearly large amounts as a problematic and pathological type of eating. In contrast, the utility of identifying "clearly small" amounts seems less clear and likely less useful. In addition to AN, which is based primarily on weight not eating behavior, two behavioral eating patterns have been described as potentially useful subtypes of EDNOS that specifically exclude eating large amounts (OBEs) but do not require "clearly small" amounts. The first, labeled subjective binge eating disorder, is similar to BED but the binges do not involve clearly large amounts, and the second is purging disorder, which involves purging after normal to small amounts of food. Current thinking suggests that a genetic vulnerability—perhaps higher impulsivity—might make it more likely that a person would eat large amounts. If so, then BED and BN would share this vulnerability but subjective binge-eating, purging disorder and restricting type AN would not. Cultural pressures to be thin may be more highly implicated in those patterns as distress is triggered even though the amounts eaten are relatively normal. AN binge-purge type could fall in either category as size of binges varies considerably.

Two alternative classification schemes have been proposed. The three dimensional model (Williamson, Gilaves and Stewart, 2005) suggests at least six behavioral patterns that may be useful to study. A better understanding of specific behavior patterns may encourage the development of novel biological and psychological interventions to target those patterns, particularly during periods of acute symptoms. The alternative approach to classification is the transtheoretical model of Fairburn and colleagues (Fairburn and Bohn, 2005) that proposes collapsing current categories into a single ED diagnosis. Overconcern with weight, shape, or controlling eating is viewed as the core pathology that must be addressed for successful treatment, regardless of the specific problem behaviors. Both of these approaches are likely to make useful contributions to our overall understanding of the development of disordered eating. Since the current culture promotes excessive eating yet endorses the thin ideal, a certain level of concern with weight and shape is expected. Normative discontent and normative dieting may be unavoidable and not necessarily predictive of the emergence of significant eating pathology. What is needed is to find ways to identify critical points when normal concern becomes overconcern, and when normal dieting escalates to the point that binge eating is triggered. In the current environment, even weak genetic vulnerabilities related to regulation of eating behavior (e.g., vulnerabilities that intensify or reduce the experience of hunger, and those that intensify or reduce impulsive or compulsive behaviors) are likely to be e...
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likely to be expressed. Initial success, or lack of success, in managing weight may send
individuls down various different pathways, only some of which end in eating disor-
ders. As far as treatment is concerned, the emphasis at initial presentation is likely to be
on specific maladaptive eating patterns. Somewhat more specific interventions and med-
ications could hopefully be developed to target specific behaviors more effectively.
However, for prevention and for long-term maintenance of recovery from eating disor-
ders, the transdiagnostic model clearly suggests that the underlying overconcern with
weight and shape motivates disordered eating behaviors. This overconcern needs to be
 targeted at some point regardless of specific problematic behavioral patterns.
 Extensive research examining personality traits that are associated with eating disor-
ders was also reviewed. Sample selection in most of these studies is not consistent with
the DSM diagnostic groupings, which means that many of the findings are not specific
to BN or BED. However, there is substantial evidence—at least for BN—that indi-
viduals diagnosed with this disorder experience increased impulsivity, compulsivity and
obessionality, perfectionism, sensation seeking, and harm avoidance, and that they
have comparatively low levels of self-directedness. Thus, regardless of the decisions
that will be made regarding the official diagnostic classification system, further work is
clearly needed to understand how the interaction of personality and biology leads to
different presentations of disordered eating. In addition, ability to lose weight varies
widely and needs to be better understood. Are there important biological differences—
 for example in the experience of hunger and satiety that impact an individual’s ability to
restrict or to maintain a thin weight? Do some people experience more intense hunger or
are some just better able to tune out or tolerate hunger? Does impulsivity or lack of
satiety account for the inability to stop eating after normal amounts? Does overconcern
 drive inappropriate compensatory behaviors? What prevents some individuals from re-
sorting to those methods? Are there differences in prohibitions against compensatory
behaviors or is the difference due to lower levels of overconcern? Future research to
answer these questions will improve our understanding of the complex presentations of
 disordered eating and improve the treatment options currently available.
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Eating Disorders: Bulimia Nervosa and Binge Eating


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**Eating Disorders:**

- Bulimia Nervosa
- Binge Eating

**Description**

Eating disorders are a group of conditions characterized by abnormal eating behaviors and attitudes towards food. This includes conditions such as Anorexia Nervosa, Bulimia Nervosa, and Binge Eating Disorder. These disorders are characterized by a persistent pattern of overeating, followed by attempts to compensate for this behavior. The symptoms can vary widely, but common signs include excessive weight loss or weight gain, frequent episodes of binge eating, and the use of compensatory behaviors such as vomiting or misuse of laxatives.

**Symptoms**

- Distorted body image
- Refusal to maintain a healthy weight
- Use of diuretics, laxatives, or other purging behaviors
- Depression
- Anxiety
- Social isolation
- Preoccupation with food

**Causes**

Eating disorders can be caused by a combination of biological, psychological, and environmental factors. Genetic predisposition, hormonal changes, and family history are some of the biological factors. Psychological factors include low self-esteem, perfectionism, and family dynamics. Environmental factors can include peer pressure, media representations of body image, and cultural influences.

**Treatment**

Treatment for eating disorders typically involves a multidisciplinary approach, which can include therapy, medication, and nutritional counseling. Cognitive-behavioral therapy is often used to help individuals understand and change their thoughts and behaviors related to food and body image. In some cases, medication may be prescribed to help manage symptoms or reduce the risk of complications related to the disorder.

**Prevention**

Prevention strategies can help reduce the risk of developing an eating disorder. This includes promoting healthy eating habits, encouraging positive body image, and addressing underlying psychological issues. Parental support and education can also play a crucial role in preventing eating disorders, especially in adolescents and young adults.