# The Validity and Clinical Utility of Binge Eating Disorder

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**Objective:** This review attempted to examine the validity and clinical utility of the DSM-IV binge eating disorder (BED) diagnosis across a wide range of validating strategies. **Method:** Various electronic databases (Pub Med, Psych Info) were searched for terms relevant to the diagnosis of BED (e.g., binge eating disorder, binge eating) in order to identify papers. Additionally, published papers were reviewed in order to locate additional manuscripts and papers that were presented at meetings. **Results:** The validity and utility of BED varied substantially according to the validator chosen. There is reasonable evidence that BED can be differentiated from other existing eating disorders and is associated with significant impairment and clinical levels of eating disorder psychopathology. The relationship of BED to obesity is complex, and in spite of some positive findings, further research examining the predictive power of BED, beyond the simple presence of obesity and associated psychopathology, in relationship to clinically relevant outcomes is needed. **Discussion:** Binge eating disorder is being considered for inclusion in the DSM-V and various options regarding this decision are reviewed based upon the empirical findings in the paper. 

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## Introduction

Binge eating disorder (BED) was included in the *Diagnostic and Statistical Manual of Mental Disorders, 4th edition, text revision* (*DSM-IV*[1]) as a provisional eating disorder diagnosis. To inform the decision-making process for BED's status in *DSM-V*, the present review examines existing empirical evidence on the validity and clinical utility of BED. Clinical utility, which may be a broader concept than validity, is a standard by which diagnoses for the *DSM-V* should be evaluated.[2] The current review focuses primarily on the ability of the *DSM-IV* BED diagnostic criteria to predict a broad range of external validating variables (e.g., family history, biological parameters, quality of life) and, most importantly, validators that reflect high degrees of clinical utility (e.g., clinical course, response to treatment). In a practical sense, this review will examine evidence that BED discriminates itself from other eating disorders and obesity (without BED) on a variety of validators. Although early research focused on the relationship of BED to eating disorder diagnoses such as anorexia nervosa (AN) and bulimia nervosa (BN), more recent research has tended to emphasize the relationship of BED to obesity, and this recent shift will be recognized in this review.

Another recent review of BED research, by Striegel-Moore and Franko[3] addressed several concepts associated with both validity and clinical utility. Their conclusions regarding the inclusion of BED in the *DSM-V* are detailed below. It is first important to note that Striegel-Moore and Franko[3] applied criteria established by Blashfield et al.[4] to evaluate whether BED should be considered as a diagnosis in the *DSM-V*. The criteria outlined by Blashfield et al.[4] are as follows:

1. There should be ample literature about the proposed syndrome.
2. The diagnostic criteria should be articulated clearly, and assessment instruments should exist that may be used for determining whether an individual meets the criteria.
3. The proposed syndrome should be diagnosable with a high degree of reliability between two or more assessors.
4. Evidence should be available that the proposed syndrome can be differentiated from other (similar) syndromes.

5. Evidence should be provided regarding the coherence and validity of the syndrome.

Applying these criteria, Striegel-Moore and Franko[3] concluded the following: First, given that there were more than a thousand titles including the term BED in a PubMed database search in 2007, there is an ample literature on this diagnosis. Second, the BED diagnostic criteria outlined in *DSM-IV* have been used quite consistently and several psychometrically sound instruments have been developed for the assessment of BED. The authors acknowledged continued debate about the specific criteria of BED, and highlighted the need for further exploration in this area. Third, there is ample evidence that the *DSM-IV* criteria for BED can be reliably applied across diagnosticians. Fourth, the authors concluded that BED can be discriminated from BN and obesity, and is therefore a distinct clinical entity. Fifth, with regards to the validity of BED, the authors concluded that empirical approaches to classification (i.e., latent class analysis) repeatedly identify a class of eating disordered individuals that is consistent with BED. Also, the authors reviewed several indicators of clinical significance including the high prevalence rates of BED, considerable evidence of impairment and suffering in BED patients, along with elevated rates of health service utilization among BED patients as indications of the clinical significance of BED. In spite of some inconsistency in the literature, the authors concluded that BED appears to be a relatively stable and protracted disorder with longitudinal evidence of clinical severity comparable to that of BN.

The authors' overall conclusion was that a case can be made for recognizing BED as a formal eating disorder diagnosis in the *DSM-V*. However, they also suggested that further classification studies were needed, using wider sets of external validators, to examine the clinical utility of newly derived criteria sets in the prediction of course and outcome. Next, we will outline the methods and results of the present review.

## METHOD

The present review follows the recommendations of Striegel-Moore and Franko[3] and examines a wide array of validators. Below, we will review empirical evidence across a variety of validating strategies, which are generally consistent with Kendell[5] and Kendler's[6] recommended validating strategies and include findings in the following areas:

1. Empirically based studies of classification (e.g., latent class analysis and taxometrics)
2. Laboratory-based studies of eating
3. Ecological momentary assessment-based studies of eating in the natural environment
4. Epidemiologic studies
5. Studies of psychiatric comorbidity
6. Studies of medical outcomes
7. Studies of health-related quality of life, life satisfaction, and functional impairment
8. Longitudinal studies of diagnostic status
9. Studies of the effect of BED on treatment outcome
10. Family history studies
11. Studies of genetic polymorphisms
12. Studies of brain function in BED
13. Studies of peptides and hormones in BED
14. Studies of the importance of shape and weight evaluation in BED.

To examine the validity and clinical utility of BED, we conducted a comprehensive literature review on BED and the validating variables. We searched major computer databases (e.g., MedLine, PsychInfo) and also reviewed reference lists from published literature. In addition, we were able to identify recent paper presentations at meetings. Search terms for the computer-based searches included "binge eating disorder" and "binge eating," as well as the key validating variables.

## RESULTS

### EMPIRICAL CLASSIFICATION STUDIES

Supporting Information Table S1 provides a summary of studies,[7–13] which have used statistical methodologies that attempt to identify a class of eating disordered individuals consistent with the *DSM-IV* BED diagnostic criteria. Typically, such studies employ latent structure models (i.e., latent class analysis, latent profile analysis) to examine empirical data to identify naturally occurring groups of eating disordered people based on similarities in symptom status, but these approaches do not help to determine if the distinctions between these groups represent qualitative or quantitative differences. Rarely, researchers have employed taxometric analyses following latent structure models to assess the nature of the boundary between the naturally occurring groups. (Keel et al., submitted for publication).

It should also be noted that not all of the empirical studies of eating disorder classification are relevant to BED. For example, Gleaves et al.[14] conducted a taxometric analysis that had significant implications for a variety of eating disorder classes, but did not include individuals in the sample who displayed prototypic BED behavior. Similarly, Keel et al.[15] conducted latent class analyses on a large sample of eating disordered individuals, but individuals with BED were excluded from the sample,

thus limiting the inferences relevant to the BED diagnostic category.

Several studies have included a variety of eating disordered participants, including individuals displaying BED-type behavior. A BED-like class was reliably discriminated from BN in an epidemiological sample of twins,[7] a sample of 238 binge eating or purging women,[11] and a sample of 687 treatment-seeking eating disorder patients.[13] In contrast, Wade et al.[12] failed to find evidence that BED could be reliably differentiated from other eating disorder profiles in a sample of 1,002 community-based twins.

Importantly, very few of these studies have examined the boundary between obesity and the related construct of BED. In other words, among obese individuals can a subgroup be reliably differentiated based on the presence of binge eating? Williamson et al.[10] reported on samples of eating disordered, obese, and normal weight individuals using factor analysis and taxometric analysis. Although these data supported a distinction between BED and other eating disorders, the small sample size of obese individuals without BED reduced the strength of inference regarding the BED–obesity relationship. In another study, Mitchell et al.[9] completed a latent profile analysis of individuals seeking treatment and receiving a diagnosis of eating disorder not otherwise specified (EDNOS). This study produced results in which two classes of obese individuals were identified, one with high levels of eating disorder psychopathology and the other with low levels of eating disorder psychopathology. These results may be interpreted as consistent with a differentiation of individuals with BED from other obese individuals.

**Summary.** Empirical approaches to classification have provided some support for the validity of BED especially in terms of separation from the eating disorders. However, limitations in the number of BED or obese non-BED cases[10,12] in these studies leave the relationship of BED to obesity unclear. Also, in some studies limitations in the measurement of symptoms[7,9,13] reduce the strength of inferences that can be drawn from these data. In large part, these studies tend to be conducted with existing data sets that were not originally designed to conduct empirical studies of classification. Like all eating disorder diagnoses, the validity of BED would be more effectively tested with an empirical approach to classification study if it consisted of an assessment of eating disorder symptoms in a sample carefully selected to provide a rigorous test of discriminant validity.

## LABORATORY-BASED STUDIES OF BED

Supporting Information Table S2 provides a summary of a variety of laboratory-based studies[16–24] examining the eating behavior of individuals with significant levels of binge eating, commonly meeting diagnostic criteria for BED. Early studies of this type indicated that individuals with BED consumed more calories than weight-matched individuals without BED in several different stimulus conditions in the lab (e.g., instructions for binge eating, instructions for non-binge meal consumption).[16,17] Using a variety of different paradigms, the fundamental finding that individuals diagnosed with BED consume more calories than individuals without BED was supported in a number of early reports[18–20] and again in more recent studies.[22–24] Only rarely have researchers failed to find a BED versus obese non-BED difference in caloric consumption in a laboratory paradigm.[19]

Several of these studies have attempted to examine the possible mechanisms associated with the finding that individuals with BED tend to consume more calories. For example, Telch and Agras[18] reported that the level of clinical depression was associated with a propensity to label an eating episode as an eating binge, regardless of caloric intake, thus potentially implicating negative mood in binge eating. However, Geliebter et al.[20] failed to find that depression was correlated with caloric intake. Similarly, Sysko et al.[22] reported that BED was associated with less fullness following food intake, potentially implying a disturbance in a satiety mechanism. However, Geliebter et al.[20] failed to find differences between BED and non-BED participants in terms of postmeal satiety ratings. Guss et al.[21] were intrigued by the fact that BED participants in their study increased their caloric consumption only in response to a binge eating instruction and not in a typical meal instruction. These authors speculated on the potential disinhibitory mechanism that may be stimulated by an instruction to binge eat. Consistent with this idea, Galanti et al.[24] report that test meal intake for binge eating ranges widely, and is highly correlated with impulsivity.

**Summary.** Laboratory studies offer some of the strongest validity data regarding BED and particularly its discrimination from obesity. Like all laboratory studies, questions remain about the ecological validity of such findings. In other words, are BED/non-BED differences in eating specific to the laboratory, or do these findings generalize to the natural environment? Studies addressing this concern are reviewed next.

## ECOLOGICAL MOMENTARY ASSESSMENT AND BED

Supporting Information Table S3 outlines three studies[25,26] and another study has recently been completed (Engel et al., submitted for publication)

that utilize ecological momentary assessment (EMA) to examine the BED diagnostic construct in terms of eating behavior in the natural environment. This methodology is an interesting complement to laboratory-based studies in that criticisms regarding ecological validity are presumably reduced. Two of these studies[25,26] had obese-BED participants and obese non-BED participants carry personal data assistants for the assessment of eating disorder behavior, stress, and mood over a period of 1 or 2 weeks. Thus, these studies allowed a comparison of reports of binge eating by both BED and non-BED obese individuals. Of note, in these reports, participants themselves determined whether or not an actual eating binge had occurred (i.e., self-identified reports). Both studies found similar results: there were no significant differences between BED and non-BED participants in binge eating episode frequency. Furthermore, the Greeno et al.'s[25] study included a dietary recall and these data similarly revealed essentially no difference between the two groups in the caloric intake during a binge eating episode. However, these studies revealed that BED individuals experienced more emotional distress and perceived loss of control preceding binge eating episodes. To the extent that these studies imply that BED and non-BED obese participants do not differ in the size or frequency of binge eating in the natural environment, they pose serious threats to the validity of the BED diagnosis.

Recently, Engel et al. (submitted for publication) completed a similar study with obese individuals who varied in terms of BED status, except that this paradigm used a more objective means of determining whether or not an eating binge had occurred. Rather than relying on participants' determination of a binge eating episode, these researchers used the Nutritional Data System for Research to calculate dietary recalls, along with momentary assessments of loss of control with handheld computers, and only considered an eating episode a binge eating episode if it involved more than a 1,000 kcal and a self-reported perception of a loss of control. Using these criteria, this study found that individuals diagnosed with BED were more likely than obese non-BED individuals to display binge eating on a daily basis in the natural environment, thus providing support for the BED diagnosis. This study, in spite of having a relatively small sample size, provides a more objective assessment of the validity of the BED construct with EMA than previous studies.

**Summary.** This small set of studies provides mixed support for the validity of BED.

## EPIDEMIOLOGICAL DATA AND BED

Supporting Information Table S4 provides a summary of epidemiologically relevant studies of BED,[27–29] which provide data relevant to its construct validity. Hudson et al.[29] studied 2,980 respondents from the National Comorbidity Study-Revised. This sample was weighted for the possibility that respondents may have been more likely to display another psychiatric disorder because of the nature of the sample. The data are somewhat limited in value by the fact that the study did not apply the *DSM-IV* BED diagnostic criteria precisely. Nonetheless, as Supporting Information Table S4 indicates, BED was more common than other eating disorders and had a later age of onset than either AN or BN. Furthermore, BED displayed a longer duration than the other eating disorders and was more likely to be associated with obesity. This is an important study that demonstrates evidence of valid distinctions between BED and other eating disorders in terms of various clinical characteristics, but is limited by the absence of a rigorous comparison of a group of obese individuals who do not meet diagnostic criteria for BED. Furthermore, as with any large epidemiological sample of adults, measurement may be limited by retrospective recall-related errors and biases.

Two other studies have used large community-based samples to examine epidemiologic and clinical characteristics associated with BED. Striegel-Moore et al.[28] studied a sample of 2,046 women in an effort to examine race differences (i.e., Black versus White) on eating disorder diagnoses. Although this study focused on racial factors, some of the findings are relevant to the current review. For example, although not explicitly tested in a statistical sense, Black participants did not display any cases of AN, though both BN and BED were well represented in the Black participants. Overall, White women were more likely than Black women to meet diagnostic criteria for any of the three eating disorders. Also, there was some evidence that BED was associated with a later age of onset than other eating disorders. In another study, Pike et al.[27] examined 150 women with BED and 150 healthy comparison participants. Women with BED had higher body mass index (BMI) scores and were also more likely to be obese than non-BED subjects. Furthermore, among BED subjects, Black participants were more likely to be obese than White participants. Additionally, BED subjects displayed more evidence of eating disorder psychopathology, but this was more characteristic of White BED participants than Black participants. Paralleling this finding, BED was associated with elevated rates of psychiatric comorbidity, but this was more characteristic of White women than Black women.

**Summary.** These studies, relying on large community-based samples, imply that BED differs from AN and BN in a host of epidemiologic variables including age of onset, racial composition, association with obesity, and psychiatric comorbidity.

## STUDIES OF PSYCHIATRIC COMORBIDITY AND BED

Supporting Information Table S5 displays BED studies on psychiatric comorbidity.[30–38] Several empirical studies suggest that obese individuals who binge eat have higher rates of Axis I and Axis II mental disorders than overweight or obese individuals who do not binge eat.[30–33] Moreover, Telch and Agras[34] and Yanovski et al.[33] provide evidence that the presence of comorbid mental disorders is specifically related to whether or not people engage in binge eating, rather than their level of obesity. These findings suggest that binge eating's association with comorbid psychopathology is not merely due to its correlation with obesity. In addition, compared to BN, BED has been associated with lower levels of comorbid psychopathology across a number of studies[30,36,37] and with equivalent rates of anxiety and mood disorders in at least one study.[35]

Two recent population-based studies have been published on comorbidity and BED. The first, by Grucza et al.[38] consisted of administering a Patient Health Questionnaire (PHQ) to a population-based sample that was recruited by random digit dialing. Of the 884 participants, 67 were categorized as BED, based upon their answers to the PHQ. They found that individuals with BED (with or without obesity) had significantly higher rates of major depression, generalized anxiety disorder, and panic attacks than individuals without BED. Obesity without BED was not associated with higher rates of psychopathology. A major strength of the study is that it is population-based, and therefore may reduce the likelihood of inflated levels of comorbid mental disorders that could exist in clinical samples (i.e., Berkson's bias). A notable weakness of the study is the method by which the psychiatric diagnoses were made. Individuals self-rated binge eating episodes without being provided with a clear definition of "objectively large" binge eating. This may have led to "false positives" in the BED group. In addition, BED criteria in the study did not match the *DSM-IV*'s criteria exactly. The PHQ asks if the person has engaged in binge eating twice per week for 3 months as opposed to the *DSM-IV*'s 6-month time period.

Finally, in a large, nationally representative, population-based study, Hudson et al.[29] compared individuals with AN, BN, and BED in terms of comorbid mental disorders. They found that, of the participants with eating disorders, individuals with BN were at the highest risk for comorbid disorders [94.5%; odds ratio: 4.7 (4.3−7.5)], followed by individuals with BED [(78.9%; odds ratio: 2.3 (2.6−3.7)], and that individuals with AN were at the lowest risk (relatively) for comorbid disorders [56.2%; 2.1 (1.2−2.9)].

**Summary.** These studies provide evidence that BED patients display significant psychiatric comorbidity (roughly comparable to other eating disorders) that cannot be simply explained by the presence of obesity.

## STUDIES OF MEDICAL OUTCOME AND BED

Supporting Information Table S6 lists the few studies[39–45] that have examined medical outcomes associated with BED. Fairburn et al.[39] compared 5-year course and outcome between community-based cohorts with BED ($n = 48$) or BN ($n = 102$). The BED and BN cohorts did not differ significantly in terms of 5-year weight gain, although the BED group (4.2 ± 9.8 kg) did gain more weight than the BN (3.3 ± 10.1 kg) group. A greater proportion of BED subjects had a BMI > 30 at follow-up (39% vs. 20%, $p = 0.06$). However, the proportion of BED subjects with a BMI > 30 was also greater at baseline (22% vs. 12%).

In a large cross-sectional epidemiological study of 4,651 female primary care and obstetric-gynecology patients, 245 BED and 44 BN cases were diagnosed based upon self-report information.[40] In comparison to patients with no psychiatric diagnoses, BED was associated with greater health impairments, more severe physical symptoms, and higher rates of diabetes. These differences remained after controlling for co-occurring psychiatric diagnoses. However, these analyses did not control for differences in BMI. Few differences in medical outcomes were found between BED and BN patients.

Bulik et al.[41] examined 5-year medical outcomes among 59 obese women with binge eating as compared to 107 obese women without binge eating in a population-based longitudinal study of White twins. Binge eating at Wave 1 was determined based on interview-based response to a lifetime binge eating question. The obese binge eating group reported significantly greater health dissatisfaction than the obese group at 5-year assessment. Although the rates of all major medical disorders (hypertension, visual impairment, asthma/respiratory illness, diabetes, cardiac problems, osteoarthritis) were higher for the obese binge eating group, none of these differences reached statistical significance. The authors note, however, that the probability of the binge eating group having a higher prevalence for all six medical conditions by chance alone would be $p = 0.015$,

which is interpreted as support for a BED-negative health relationship.

Reichborn-Kjennerud et al.[42] present cross-sectional questionnaire data from 8,045 Norwegian twins on the relationship between medical symptoms and binge eating in the absence of compensatory behaviors. Eating disorder diagnoses and eating-related behaviors were assessed through self-report responses to nine specific diagnostic questions. BMI, lifetime occurrence of physical symptoms, and disorders were established based upon self-report. After controlling for BMI, binge eating was associated with increased risk of insomnia and early menarche in women, and increased risk of neck–shoulder, lower back, and chronic muscular pain, as well as greater impairment due to physical health in men.

At the recent Eating Disorder and Classification Conference in Washington D.C., Hudson[43] presented a 2.5 year follow-up study comparing medical outcomes in 137 individuals with BED, 139 BMI-matched controls without BED, and 51 controls without BED not matched for BMI. There were no differences between BED and non-BED groups in weight change, blood pressure, or individual metabolic syndrome components (dyslipidemia, hypertension, IGF/IGT). However, BED was associated with greater risk than the non-BED groups in the development of *any* (i.e., at least one) metabolic syndrome component. The BED group also reported more frequent health care visits than the non-BED groups, a finding that has been reported previously.[45] The analysis of the 5-year outcomes is currently underway.

Hasler et al.[44] conducted a prospective community-based study of 591 young adults followed over a 20-year period from age 19. They found that binge eating four or more times in the last year as assessed with a structured diagnostic interview was positively associated with both increased weight gain and being overweight. Binge eating remained positively associated with being overweight even after controlling for a variety of sociodemographic (e.g., gender, income, education, family history of weight problems), psychopathological (e.g., SCL-90R scores, atypical depression, generalized anxiety disorder), and behavioral (e.g., physical activity) factors.

Finally, although not equivalent to a BED diagnosis, there is evidence that binge eating and loss of control eating in children and adolescents confer an increased risk for negative medical outcomes in terms of increased body fat, worsening insulin resistance, and increased triglicerides,[46,47,48] as well as increases in psychological symptoms[49,50] and reduced efficacy of weight loss interventions.[48] These findings are important because they may help to ultimately clarify that aspects of a BED diagnosis are most detrimental to physical and psychological health.

**Summary.** Overall, support for the idea that BED or binge eating predicts weight gain or negative medical outcomes is both limited and mixed. Many of the studies are limited by small sample sizes. However, the largest prospective study of adults[44] shows an association between binge eating and weight gain even after controlling for a variety of potential confounds. Finally, there is good evidence to suggest that BED appears to be associated with increased health care utilization.

## STUDIES OF HEALTH-RELATED QUALITY OF LIFE (HRQOL), LIFE SATISFACTION, AND FUNCTIONAL IMPAIRMENT

In the following section several studies investigating health-related quality of life, life satisfaction, or functional impairment in BED patients are reviewed (see Supporting Information Table S7). Although these constructs are considered by most to be distinct from each other, we view them as related constructs and at least partially overlapping. Given the large number of empirical reports addressing these constructs, studies were chosen based on the following criteria: (1) number of BED patients included in the study, (2) variety in the chosen comparison groups, (3) inclusion of a variety of generic and disease-specific outcome measures, and (4) scientific rigor.

A number of studies have shown that obese BED participants report greater impairments in generic HRQOL, life satisfaction, or functional impairment.[51–55] Masheb and Grilo[54] reported that BED participants have considerably impaired scores on the SF-36 Health Survey compared to those seen in U.S. norms. Additionally, obese BED patients report lower HRQOL than obese non-BED patients.[51,53] Further, women with BED generally report functional impairment at similar levels to groups of eating disorder patients.[40] Finally, disease-specific HRQOL instruments have shown a similar pattern of results: obese BED patients report greater impairments than obese non-BED patients in overall HRQOL, as well in a variety of specific domains of HRQOL.[55]

One interesting study attempted to partial out the unique contribution of BED on HRQOL after accounting for possible confounding differences between obese BED and non-BED groups. Kolotkin et al.[52] compared 95 BED patients to 435 obese non-BED patients and found that the BED group was comprised of a higher percentage of females, was younger, more likely to be White, had a higher BMI, and was more psychologically distressed. Importantly, after controlling for these group differences,

BED status was unrelated to obesity-specific HRQOL, suggesting that the diagnosis of BED did not uniquely predict HRQOL in obese individuals.

**Summary.** BED patients report more HRQOL and functional impairment than a variety of comparison groups, although the unique association of HRQOL and binge eating is unclear.

## LONGITUDINAL STUDIES OF BED DIAGNOSTIC STATUS

Supporting Information Table S8 depicts a series of longitudinal studies[39,56–58] (Agras et al., submitted for publication) that have examined either the diagnostic stability of BED in terms of category crossover, or its propensity for recovery or remission relative to other eating disorders. Each of these longitudinal validators provides meaningful clinical information. In terms of diagnostic stability, the evidence is relatively unclear. Agras et al. (submitted for publication) reported that ~50–60% of individuals with subclinical or full syndrome BED are best classified as presenting with a BED-like disorder 12 months following the initial assessment. Fairburn et al.[39] found much less evidence that individuals with a BED diagnosis retained that diagnosis at 5-year follow-up. Only 9% of the 48 individuals diagnosed with BED initially were found to still meet diagnostic criteria for the syndrome. Cachelin et al.[56] completed a small follow-up study of individuals with BED, and also found considerable variability in maintenance of the BED diagnosis at 6-month follow-up. Finally, Fichter and Quadflieg[58] longitudinally studied a large group of various eating disordered patients, including 60 BED patients. They found little evidence of diagnostic crossover between BED and AN. However, there was considerable transition from BED to other forms of EDNOS or BN. Only 1.7% of BED patients retained their diagnosis at 2-year follow-up. Thus, these longitudinal data suggest that BED is highly distinct from AN, and there is little evidence to suggest that BED represents a partially remitted form of BN.

In terms of recovery rates, another longitudinal validator with considerable clinical relevance, Fairburn et al.[39] reported that 85% of their BED cases recovered within 5 years, and that individuals with BED were more likely to recover than individuals with BN. In a 4-year follow-up study, Agras et al. (submitted for publication) found that ~50% of the previously diagnosed cases recovered from BED. Similarly, individuals with the subclinical form of BED recovered in ~48% of the cases. Using a very different strategy, Pope et al.[57] reported an average duration of BED of 14.4 years in their family study of BED probands and their relatives. They concluded that BED is a highly stable disorder that displays more chronicity than AN or BN. However, this study is significantly limited, because number of years with BED was determined based upon the retrospective recall of participants. Finally, Fichter and Quadflieg[58] reported the recovery rates at 2, 6, and 12 years to be 65, 78, and 67%, respectively.

**Summary.** Although there is variability in the data, it does appear that BED differs from other eating disorders in terms of a greater tendency toward recovery and fluctuation, although this may be embedded in a chronic pattern of remission and relapse. It also appears that those with BED are less likely than AN or BN to crossover to another active eating disorder.

## TREATMENT STUDIES AND THE BED DIAGNOSIS

Three types of treatment-related studies are examined below. The first type of study examines the moderating influence of the BED diagnosis on weight loss interventions in obese or overweight samples. The second type of study compares the efficacy of multiple treatments delivered to BED participants and a comparison group (e.g., obese, non-BED) in what may be called a BED-nonBED treatment specificity design. A third design compares different treatments (e.g., cognitive-behavioral therapy [CBT] versus behavioral weight loss) applied to subjects all meeting criteria for BED in what may be termed a BED multiple-treatment design. Each of these designs will be reviewed and discussed below.

## STUDIES EXAMINING THE BED DIAGNOSIS AS A MODERATOR OF BEHAVIORAL WEIGHT LOSS TREATMENTS

Supporting Information Table S9 provides a summary of studies[59–74] examining the moderating effect of a BED diagnosis on behavioral and surgical treatments for obesity. Evidence that the presence of BED has a significant influence on weight loss interventions would be an important factor to consider for clinical utility.

**Behavioral Weight Loss.** A series of behavioral weight loss or very low calorie diet (VLCD) studies were conducted before the official *DSM-IV* BED criteria were established. Consequently, these studies do not provide a stringent test of the current BED diagnostic criteria, but are worthy of brief mention. All three of these studies[59–61] categorized obese individuals into those with binge eating or those without binge eating, and examined the influence of this classification on behavioral weight loss or VLCD interventions. All three studies[59–61] failed to detect any classification-related effect on response to

these treatments. As noted, the measures of binge eating used in these studies probably would overlap with the *DSM* BED diagnosis, but are clearly not isomorphic with that construct. Yanovski et al.[62] did find that BED impacted several outcomes in VLCD interventions. For example, BED patients were more likely to drop out, display "extreme" deviations from the weight loss protocol, and also showed less weight loss in the refeeding phase of the VLCD. However compared to non-BED obese patients, BED individuals did not distinguish themselves in *overall* adherence to the protocol, weight loss at the end of protocol, or weight loss at 1-year follow-up. BED subjects were more inclined to show substantial weight regain at 3-month follow-up, which was perceived as a negative outcome. However, Yanovski et al.'s[62] findings were not supported by two other studies, which found that BED was associated with *decreased* likelihood of dropout from behavioral weight loss programs,[63,65] *greater* weight loss at the end of treatment, and to a lesser degree at the end of 1-year follow-ups. In a similar study, Raymond et al.[71] compared individuals with BED to individuals with subclinical BED or no binge eating in a VLCD weight loss program. They found no differences in weight loss at end of treatment, 1-year follow-up, or in the number of sessions completed by either group. This study is limited by the fact that subclinical binge eaters were included in the obese non-binge eater group, which may have obscured potential differences.

In a study of the impact of BED on weight loss in an effectiveness design, Pagoto et al.[68] examined the impact of a clinically derived BED diagnosis on weight loss in a 16-visit outpatient weight loss program. All patients ($n = 131$) who enrolled in a weight loss program were included in the analysis. The sample was on average morbidly obese (average BMI = 43.08), and the obesity program was designed to prevent diabetes through lifestyle interventions. There was a significant effect of diagnosis on weight loss, with individuals exhibiting baseline BED diagnoses losing substantially less weight (weight change = −3.10%) when compared to obese individuals without BED (weight change = −5.35%). Furthermore, only 16% of the individuals with BED met the program goal of a 7% weight loss, which is substantially less than the total sample percentage of 31%. It is also worth noting that these researchers examined the effect of a diagnosis of major depression on weight loss, and found that individuals with major depression lost significantly less weight ($M = -5.28\%$) than individuals without major depression ($M = -3.29\%$ weight loss). Also, this study reports that BED was not associated with dropout, whereas major depression was. In spite of the value of this study in terms of evidence that BED moderates weight loss, it failed to examine the predictive power of either BED or major depression when controlling for the other diagnosis. Therefore, the unique predictive power of BED, above and beyond major depression, was not tested.

As part of the Look Ahead trial, Gorin et al.[70] studied 5,145 individuals with Type 2 diabetes. All individuals had BMIs greater than 25 $kg/m^2$, and were randomized to either an intensive lifestyle intervention or a diabetes and support education treatment condition. The researchers used the Questionnaire of Eating and Weight Patterns to assess binge eating and classified 123 subjects as a BED group, whereas 4,222 subjects remained in the comparison group. In terms of weight loss, the intensive lifestyle group had a more favorable outcome than the diabetes support and education condition, regardless of BED status. Across treatment conditions, baseline BED diagnosis did not moderate outcome for either weight loss or reduction of risk factors for cardiovascular disease. The authors conducted more intensive analyses of binge eating behavior of subjects, and found that individuals who retained their BED diagnosis at the end of treatment lost considerably less weight than individuals who met criteria for baseline BED diagnosis but failed to meet diagnostic criteria at the end of treatment. They concluded that individuals with BED should not be discouraged from entering behavioral weight loss programs and, in fact, they noted the possibility that weight loss programs can improve binge eating. In other words, this study conveyed no data to support the role of BED as a moderator of treatment; however, the assessment study for BED represents a weakness, especially for diagnostic inferences.

**Bariatric Surgery.** Supporting Information Table S9 provides a summary of studies[72–74] examining the moderating effect of the BED diagnosis on surgical interventions for weight loss. Burgmer et al.[72] examined a variety of forms of binge eating in a sample of bariatric surgery candidates. These researchers failed to find evidence that preoperative binge eating predicted weight loss at 12-month follow-up, although the number of *DSM-IV* BED patients was insufficient for an optimal test of the diagnostic construct. Similarly, Busetto et al.[73] in a more rigorously assessed patient group, failed to find evidence that BED was associated with differential weight loss following surgery and at 5-year follow-up, but found that BED was associated with a greater likelihood of surgical complications. In the only study that provided evidence of BED as a significant moderator of weight loss following bariatric surgery, Sallet et al.[74] found that 2 years after Roux-en-y surgery, individuals with BED or subthreshold BED lost less weight than individuals

not meeting these diagnostic criteria. Thus, the moderating significance of BED on weight loss in surgical patients is mixed.

Although not directly related to BED, there is recent evidence suggesting that postoperative binge eating or so called "loss of control eating" predicts negative outcomes in bariatric surgery such as less weight loss or more weight regain.[75–78] Although again not a precise model for BED, these findings may help to clarify those aspects of BED that are most predictive of weight gain.

**Meta-Analysis of the Moderating Effect of BED on Weight Loss Treatment.** In a unique meta-analysis, Blaine and Rodman[69] identified samples of obese individuals with BED who had received some form of weight loss treatment (i.e., psychotherapeutic, drug, surgery). In a creative strategy, they identified samples of obese non-BED individuals in the literature who had received the same fundamental treatment and matched them to the BED sample on variables including sample size, percentage of females in the sample, mean age, treatment type, and mean sample pretreatment BMI. This meta-analysis is unique in that the investigators were able to "create" comparisons between BED and non-BED samples of obese individuals, which were not technically conducted in the same study. Thirteen pairs of matched samples were submitted to meta-analytic analysis that examined the impact of BED on weight loss and decreases in depression. Obese BED samples lost significantly less weight (average weight loss = 1.3 kg) compared to obese non-BED samples (average weight loss = 10.5 kg), but there were no differences between the groups in the extent to which depression scores were reduced during weight loss treatment, although both groups showed significant reductions in depression.

The Blaine and Rodman meta-analysis[69] is unique and implies that BED has clinical utility in terms of its impact on weight loss. However, two key limitations should be noted. First, although the actual treatments that were matched between samples may be a rough equivalent, they are clearly not comparable in a highly detailed comparison and should not be considered equal. Although treatments may be roughly comparable in length and frequency of sessions across studies, the details of the procedures cannot be equated. Second, the assessment of BED in this meta-analysis was based largely on self report measures such as the Binge Eating Scale and the Questionnaire of Eating and Weight Patterns-Revised. Although these measures may be correlated with a *DSM-IV* BED diagnosis, they are not isomorphic with such a diagnosis.

Thus, the idea that BED will moderate the effectiveness of weight loss treatments, such as behavioral weight loss programs or bariatric surgery, is inconsistently supported in the empirical literature and the overall effect appears small. This is significant because these studies do not indicate that behavioral weight loss experts or bariatric surgeons should modify their treatments for individuals with BED.

**BED-nonBED Treatment Specificity Designs.** Treatment specificity designs may offer a powerful test of the validity and utility of a construct, if the design includes at least two different groups receiving at least two different treatments. This has long been considered a powerful test of the clinical utility for diagnoses.[5] In such a design, one is able to determine if a diagnostic group or classification variable moderates treatment response across all tested treatments and whether each treatment tested shows differential effectiveness across diagnoses. Two studies have included both multiple diagnostic groups and multiple treatments in treatment studies of BED (see Supporting Information Table S9). Porzelius et al.[64] compared obese individuals with differing levels of BED in two behavioral weight loss programs. One of the programs was more behavioral in nature, while the other relied on a more broad-based cognitive-behavioral strategy. There was a trend for more severe binge eaters to lose more weight in the cognitive-behavioral intervention than the strictly behavioral intervention and for more moderate binge eaters to respond more favorably to the behavioral program. There was no significant difference between treatments for those with less severe binge eating. Although this finding reflects some degree of specificity, the assessments of BED were made with a self-report instrument for binge eating and consequently are somewhat limited.

Nauta et al.[66,67] completed a study comparing behavioral weight loss treatment to a cognitive therapy designed to promote weight loss using cognitive and behavioral techniques to reduce binge eating or overeating. BED subjects differed from non-BED subjects in terms of greater decreases in weight and shape concerns over time, increases in self-esteem, and weight regain after treatment. However, there was not evidence that BED participants showed a differential treatment response to one treatment over the other across a broad range of measures.

These two studies suggest that the diagnosis of BED has a small effect on treatment response regarding weight loss, but evidence regarding specificity of the treatment types was contradictory. It is important to note that the number of studies that have actually utilized BED-nonBED treatment specificity designs is extremely limited, in contrast to a growing number of BED, multiple treatment designs that are reviewed below.

**BED Multiple-Treatment Designs.** In the eating disorder literature, there have recently been

a growing number of treatment specificity studies that compared the relative efficacy of different treatments for BED in samples comprised solely of BED subjects.[79–82] (see Supporting Information Table S9). These studies converge to indicate that individuals meeting criteria for a BED diagnosis show greater reduction in binge eating and associated psychopathology when receiving CBT or IPT than when being treated with treatments that may be viewed as nonspecific, such as fluoxetine or behavioral weight loss programs and provide useful information about differential treatment approaches for BED. These findings suggest that BED is *not* a nonspecific class that responds equally to virtually all treatments (including behavioral weight loss) and that specialty treatments that target eating disorder psychopathology are preferred when the desired outcome is reduction of binge eating. Thus, BED multiple treatment designs provide data supporting the clinical utility of BED in that the presence of the diagnosis predicts a superior reduction in binge eating when provided with specific treatments (e.g., CBT or IPT). Such information is valuable to a practicing eating disorder clinician who is presented with a BED case and needs to make a choice about the most effective treatment.

However, it is important to note that this is a different question than that addressed in the previous sections in studies examining the moderating influence of a BED diagnosis on weight loss treatments. Studies that include at least two diagnostic groups answer a different question than treatment designs that include only one diagnostic group. This issue may be clarified with an example. Kendell[5] makes the point that therapeutic trials can be useful for testing clinical validity of psychiatric diagnoses and highlights the power of multiple diagnostic groups in treatment studies for the purpose of diagnostic validation. He offers the example of differentiating between two diagnoses (i.e., hypochromic anemia and pernicious anemia) through the demonstration that pernicious anemia responds only to vitamin $B^{12}$ therapy while the more common hypochromic anemia responds to oral iron therapy, thus providing differential treatment inferences while simultaneously validating the diagnostic distinction between the two conditions. In this example, not only are two treatments compared, but they are also compared across two classes or diagnostic groups. The inclusion of the diagnostic comparison group enhances the strength of inference about the validity of the diagnostic distinction by accounting for treatment outcome variance that can be attributed to diagnostic variability (i.e., two types of anemia). On the other hand, consider a similar study using only one diagnostic group, similar to the BED, multiple treatment design. If we conducted a study in which only one class of patients (i.e., pernicious anemia) was randomized to either $B^{12}$ or oral iron treatments, we may find that patients receiving $B^{12}$ respond in a superior fashion. With such results, could we infer that pernicious anemia is diagnostically distinct from hypochromic anemia? In the absence of a diagnostic comparison group, such an inference is not justified. Paralleling this example, assigning subjects with BED to either CBT or behavioral weight loss, and demonstrating a differential treatment response, does not support the discrimination of BED from an alternative class (e.g., obesity).

The fundamental point is that these two research designs answer different questions. Designs that include only one diagnostic group, compared across different treatments provide a specific type of information. If one treatment is superior to another for a given diagnostic class, it is evidence of clinical utility because the diagnosis informs a clinician to select one treatment over another that has been shown to be more efficacious. Obviously, this has substantial relevance to the practicing clinician who makes diagnoses and then applies what is believed to be the most effective treatment for that diagnosis.

On the other hand, multiple diagnostic group designs help to determine if two putatively different diagnostic groups respond differently to one or more treatments and test the validity of the diagnoses. It is plausible to imagine such a design in the eating disorders in which individuals with BN and individuals with BED are randomized to either interpersonal psychotherapy or cognitive behavioral therapy-enhanced. This hypothetical study would help us to determine if the BN-BED distinction is valid in terms of treatment response to these particular treatments. If there were differences between BN and BED in their response to either treatment, it would support the validity of these diagnoses. If the interaction of diagnosis and treatment was significant, it would not only support the distinction between BED and BN, but would also help us to determine which treatment is most appropriate for each diagnosis, conferring clinical utility. Thus, BED-multiple treatment designs offer valuable data regarding clinical utility, while BED-nonBED treatment specificity designs provide useful information about validity.

**Summary.** The influence of BED on weight loss treatments is unclear and limited by a paucity of relevant studies. A unique meta-analysis provides some support for the validity of BED on this issue. Empirical evidence supports the idea that several specialty treatments are superior to behavioral weight loss for reducing binge eating and associated psychopathology in BED, which offers clinical utility.

## FAMILY HISTORY AND BED

Supporting Information Table S10 provides a brief overview of evidence that BED is a diagnosis that tends to run in families,[83,84] which may have implications for its etiology. Hudson et al.[83] studied 300 probands, half of whom met criteria for BED. Subjects were age- and sex-matched, and 888 first degree relatives of the probands were blindly interviewed for family history information. The data indicated that relatives of BED probands were significantly more likely to carry a BED diagnosis than non-BED probands' relatives. Furthermore, relatives of BED subjects displayed higher BMIs and prevalence of obesity even after controlling for proband obesity. In a follow-up study, Javaras et al.[84] utilized the same data along with a Norwegian twin registry that included heritability estimates for binge eating. These researchers estimated that the heritability of BED in the family history study was ~0.57 and the heritability for binge eating in the twin registry was ~0.39. These data imply that the propensity for BED to run in families may be largely accounted by the additive effects of genes.

**Summary.** Early evidence suggests that BED appears to demonstrate familiality that may reflect genetic influences.

## GENETIC AND PSYCHOBIOLOGICAL VALIDATION OF BED

Supporting Information Table S11 provides selective review of information related to genetic and psychobiological variables in BED subjects. Supporting Information Table S11 provides a synopsis of six different candidate gene studies in BED, which examined several different polymorphisms.[85–90] Two studies examined *DSM-IV* BED diagnoses in terms of the MC4R polymorphism and found no evidence that individuals with BED were any more likely to carry this polymorphism than non-BED subjects.[85,86] Similarly, Monteleone et al.[87] examined the cDNA 385C polymorphism and failed to find evidence that this genetic abnormality is associated with BED, although it did appear to be associated with obesity. Monteleone et al.[88] also failed to find any evidence of the validity of BED in terms of the CLOCK polymorphism, but did find evidence that BED was associated with the LL genotype or the l allele distribution on the 5HTTLPR.[89] However, because control subjects were not obese in this study, it is unclear if the polymorphism is related to binge eating or obesity. Finally, Davis et al.[90] report that BED subjects with a 9 repeat allele of the dopamine transporter gene display more appetite suppression in response to methylphenidate than non-BED individuals with the same allelic expression, suggesting that this genetic variable may have some influence on BED.

A few studies have begun to examine BED in terms of brain functioning (see Supporting Information Table S11).[91,92] Karhunen et al.[91] reported that BED subjects showed greater left hemisphere activation using SPECT than non-BED control subjects, but only when presented with food-related stimuli. In this study, BED subjects showed increased hunger that correlated with cerebral blood flow in the frontal and prefrontal areas.

Geliebter et al.[92] used fMRI to study obese and lean women in terms of food-related activation of specific brain regions. Obese subjects with binge eating showed more frontal activation in response to binge food cues than other groups, but this was apparently not true of lean subjects with binge eating. Therefore, it is not clear if this brain activation finding is related to binge eating or obesity. Furthermore, the BED designation was not completely consistent with *DSM-IV* criteria, which limits the degree of diagnostic validity that can be inferred.

As can be seen in the bottom section of Supporting Information Table S11, a series of studies have examined the validity of BED on the basis of comparisons of various peptides and hormones.[93–99] These studies have generally failed to identify a reliable difference in particular peptides or hormones that is specifically associated with BED. For example, a growing number of studies examined ghrelin.[93–97] Two of these studies failed to find evidence that changes in ghrelin levels were associated with BED, above and beyond the effects of obesity,[93,95] while Geliebter et al.[94,96,97] found that BED was associated with decreased ghrelin, even when subjects were comparable in terms of weight. Furthermore, BED appears associated with lower baseline ghrelin and less ghrelin decrease after meals. Other studies have examined cortisol in BED subjects[98,99] and there was conflicting evidence that BED subjects showed differences in cortisol levels when compared to weight-matched subjects, with one study identifying a difference,[98] and the other failing to find a difference.[99]

**Summary.** There are not yet any genetic polymorphisms or neurotransmitter/peptide findings that are clearly and consistently related to BED. Studies of brain function in BED are interesting and preliminary.

## SHAPE AND WEIGHT CONCERNS IN BED

Several recent studies have examined the idea that weight and shape concerns may help to improve the construct validity of the BED diagnosis.

As Supporting Information Table S12 reveals, BED cases that display high levels of weight and shape concern appear to have a different profile of psychopathology than BED cases without similar concerns.[100–103] Masheb and Grilo[100] found that BED subjects (whether obese or not) tended to display lower restraint, but similar binge eating frequency and concern about shape and weight as BN subjects. They concluded that BED cases display shape and weight concerns on par with BN. Three other studies converge to suggest that within the BED diagnostic construct, the presence of high levels of shape and weight concern help differentiate potential subgroups of BED cases.[101–103] In each of these studies, it appears that high degrees of shape and weight concern are associated with greater levels of psychopathology and impairment in comparison to BED participant/patients with lower levels of shape and weight concerns. Furthermore, Grilo et al.[103] indicate that these differences remain the same even after controlling for depression scores.

In an extension of these previous studies, Grilo[104] presented preliminary new data examining the predictive significance of overvaluation of weight and shape. Reanalyses of the previous studies[105,106] examined the predictive value of overvaluation of shape and weight and suggested that this cognitive feature was a significant predictor of outcome, but not a moderator (interact with specific treatments) in either study. Grilo[104] also reported data examining three different treatments for BED (i.e., CBT, behavioral weight loss, and CBT/behavioral weight loss combination), and again found that regardless of treatment condition, overvaluation significantly predicted post-treatment levels of binge eating frequency. These data suggest that overvaluation of shape and weight in BED is not only a predictor of concurrent clinical variables, but also appears to have predictive validity that is clinically meaningful.

**Summary.** Overvaluation of shape and weight displays evidence of clinical utility across an array of validating strategies and may warrant consideration as a diagnostic criterion or specifier.

## Discussion

To examine the current status of BED as a meaningful diagnosis for the *DSM-V*, we adopted two concepts that have frequently been applied to diagnostic discussions, but have varied in their definitions. The concepts of validity and clinical utility have been considered as essentially synonymous,[107] independent,[108] or as separate but overlapping constructs.[109] Our conclusions rely heavily on the ideas of Kendell and Jablensky[108] in that we consider BED to have validity if there is evidence of a clear boundary between BED and its related or similar conditions. We consider BED to have clinical utility if it provides nontrivial information about the prognosis and likely treatment outcomes, and/ or testable propositions about biological and social correlates.[108] Put differently, utility may depend on two things: (1) the quantity and quality of information in the literature, and (2) whether the implications of that information, particularly about etiology, prognosis, and treatment are substantially different from the implications of analogous information about other related syndromes.

### Is BED valid?

The empirical classification research (i.e., latent structure models, taxometrics) reviewed previously highlights that BED is typically identified as a class separate from AN-like or BN-like classes. Therefore, it seems to display a boundary with other traditional eating disorders in empirical taxonomic studies. However, studies assessing the boundary with obesity are limited. As a result, a key boundary test of validity for BED has not been conducted in a sufficient number of studies to lend strong support to the notion of BED is a diagnosis that is well differentiated from obesity in empirical classification studies. However, research designs that compare at least two putative diagnostic groups on relevant validators may also help to identify possible diagnostic boundaries. These studies are summarized in the next section.

### Does BED have clinical utility?

Again, evidence for a distinction between BED and more traditional eating disorder diagnoses like AN and BN has been summarized previously,[110–112] and is reasonably strong with evidence that BED differs from AN and BN in a number of clinically relevant variables, such as recovery rates, diagnostic stability, age of onset, gender distribution, BMI, dietary restraint, relative age of onset of dieting and binge eating, psychiatric comorbidity, and binge characteristics. Thus, BED appears to distinguish itself from current eating disorders.

However, the comparison to obesity is less clear. This is not to say that all cases of BED necessarily represent a subset of the obese population or that BED must distinguish itself from obesity in order to have clinical utility (comparisons to existing eating disorders on a variety of other validators may demonstrate sufficient clinical utility of the diagnosis). Yet, discrimination from obesity on clinically significant validators would refute the idea that BED cannot be discriminated from obesity with concurrent psychiatric disturbance.[113] To the extent that such a model is true, it does not lend support to including BED as a diagnosis and implies that

reliance on existing diagnoses, such major depression or generalized anxiety, could be used to capture the essential clinical information. Consequently, evidence that BED represents more than obesity with psychiatric and psychological disturbance becomes important in the support for the creation of a new eating disorder diagnosis and is relevant to this review.

When compared to obese individuals without BED, individuals with the BED diagnosis consume more calories in various eating episodes, although this is better documented in lab settings than in the field. There is also evidence that the BED diagnosis is correlated with significant functional impairment, lower quality of life, and psychiatric comorbidity. Additionally, there is preliminary evidence that the BED diagnosis captures a phenomenon that runs in families and seems to be accounted in part by additive genetic variance. However, there is currently little evidence to suggest that BED is correlated with any particular genetic polymorphism, peptide or neurotransmitter abnormality, or particular pattern of dysfunction in brain function.

Most importantly for clinical classification purposes, however, is the question of whether BED contributes nontrivial information about prognosis and treatment outcome. The early studies on the impact of BED on weight loss interventions were mixed and inconclusive. More recent studies continue to be inconclusive, but a controlled meta-analysis provides some greater evidence for the possible moderating influence of BED on weight loss. There is accumulating evidence that individuals carrying a BED diagnosis show greater reduction in binge eating and associated psychopathology with speciality treatments that target such psychopathology than with more generic and nonspecific weight loss treatment. Thus, the diagnosis informs treatment planning that is clinically relevant.

Prospective prediction of negative medical or psychiatric outcomes in individuals with BED is also clinically useful information, but currently rather limited in the empirical literature. Although BED is often correlated with negative medical status, studies are frequently cross-sectional with weak assessment of either BED or medical status. The recent Look Ahead trial provided a modicum of evidence that BED is associated with medical parameters associated with poor health, but the study by Haslam et al.[44] offers more compelling prospective data supporting this relationship.

## Is BED simply a marker for psychopathology?

A variant of the idea that BED is fundamentally an eating disordered subtype of obesity is that it represents a marker for other psychopathology, rather than an eating disorder. Stunkard and Allison[113] speculated that BED lacks the specific psychopathology and stability to warrant its inclusion as a true diagnosis and that it may be understood as a simple marker for generic, nonspecific psychopathology. Based on the premise that a diagnosis is designed to prescribe the course of an illness and a course of action, Stunkard and Allison reasoned that BED should not be considered a viable diagnosis. They suggested that BED is highly unstable and frequently remits. Moreover, they indicated that BED responds to a wide array of treatments, including those that do not target underlying psychopathology related to BED, such as behavioral weight loss. They reasoned that in the absence of data supporting the predictive value regarding a reliable clinical course and a specific response to treatment, BED should not be considered a distinct eating disorder diagnosis.

Several pieces of data in this review address Stunkard's and Allison's previous analysis and offer new findings in that regard. It remains true that BED is more likely to remit than other eating disorders,[58] (Agras et al., submitted for publication) but in spite of its remitting pattern, patients perceive the disorder as stable and enduring, often lasting over a decade.[57] Furthermore, the findings that certain specialized treatments targeting specific BED related psychopathology perform better in reducing binge eating than do nonspecific behavioral weight loss treatments[79,80] is not consistent with Stunkard's and Allison's hypothesis. These findings are inconsistent with the idea that the diagnosis of BED fails to carry any predictive value regarding course or clinical outcomes.

However, Stunkard and Allison's implications regarding the overlap between BED, depression, anxiety, and possibly personality traits, such as neuroticism, remain generally untested in the literature. Few studies have examined the predictive validity of BED after controlling for both obesity and some type of affective disturbance and remain unclear in terms of their implications. On the one hand, there is evidence that the presence of BED, and particularly BED with overvaluation of shape and weight, predicts eating disorder psychopathology beyond that accounted for simply by depression.[104] Similarly, there is evidence that binge eating predicts long-term medical complications after controlling for levels of affective distress.[44] On the other hand, there is evidence to suggest that after controlling for emotional distress and weight, BED offers little in terms of incremental validity in the prediction of functional impairment.[52] However, there is evidence that inclusion of certain criteria, such as overvaluation of shape and weight may help

to discriminate BED from depression and distress, and produce a diagnostic construct with more specific psychopathology.[104] Although new data challenge the original hypothesis of Stunkard and Allison, studies examining the relationship of BED to other psychopathological constructs are needed to further test this idea.

## Conclusions

The following options may be considered regarding the future of the BED diagnosis:

1. Retain BED as an example of EDNOS and a disorder in need of further study in the DSM-V.

   Arguments in favor of this option would be that some of the critical prospective or taxonomic tests of the validity or clinical utility of BED have not yet been conducted. It could be argued that such a decision would prompt further rigorous research that is needed to more definitively test the diagnostic validity of BED. However, such an option overlooks significant amounts of research that have already been conducted, which may provide enough information to make an informed decision.
2. Include BED as a formal diagnosis in the *DSM-V.*

   This option is supported by the well-documented number of patients who may have a disturbance in binge eating behavior, marked psychiatric comorbidity, functional impairment, and regularly report to psychiatric and eating disorder treatment facilities. Furthermore, there is preliminary evidence that this is a relatively enduring and impairing condition, which may have a currently unspecified familial basis. Additionally, the salient feature of the diagnosis, binge eating, is differentially responsive to treatments. The primary argument against such inclusion is that critical studies testing the predictive value of the diagnosis regarding clinical outcomes and response to treatment are limited in number and rely on research designs that do not rigorously assess discriminant validity from related conditions.
3. Eliminate BED from the eating disorder diagnoses.

   This option would be supported if there was a compelling body of literature, suggesting that BED cannot be distinguished from other eating disorders, obesity, or another psychiatric disorder, such as mood or anxiety disorders. It would also be supported if the diagnosis did not predict other important biological, psychological, or social variables. As noted, the evidence that BED can be discriminated from other eating disorders is substantial. There is also evidence on certain variables (psychiatric comorbidity, functional impairment, eating behavior, and eating disorder psychopathology) that BED can be discriminated from obesity, but further studies on clinical validators are warranted. Additionally, the finding that some specific treatments result in greater symptom relief is clinically useful and important. Finally, the idea that BED could be replaced by the application of mood or anxiety disorder diagnoses, particularly in overweight or obese individuals, with no net loss of information is not sufficiently supported in the literature for serious consideration. Recent treatment specificity findings are not consistent with a nonspecificity theory and studies examining overlap between mood or anxiety disorders and BED are insufficient in number to make any strong conclusions.
4. Include BED in the DSM-V with overvaluation of shape and weight as a specifier. Support for this option comes from a small number of studies, which have consistently suggested that overvaluation of shape and weight has clinical utility and predicts a variety of outcomes above and beyond obesity, depression, and simple BED. Arguments against this option would be based primarily on the limited number of studies that have been conducted on this construct.
5. Include overvaluation of shape and weight as a criterion in the BED criteria set in the DSM-V. Support for this option rests in the limited data suggesting that BED patients with overvaluation regarding shape and weight show the clearest discrimination from obesity. Arguments against this option would be that a significant number of individuals with binge eating and possible distress and impairment would be excluded from the diagnosis if overvaluation of shape and weight were required.

## Summary

The diagnosis of BED outlined by the DSM-IV Task Force has received considerable empirical attention. Many of these studies have addressed research questions that are relevant to assessing the validity and clinical utility of this diagnosis. These studies help to examine two key questions regarding the status of BED. First, is BED distinguishable from other eating disorders and does it confer clinically useful information? There has been relatively consistent and strong support for the identification of

a latent class resembling BED across a significant number of empirically based classification studies, lending support to the notion that BED can be meaningfully discriminated from other eating disorders. Furthermore, there is evidence that individuals with BED display rates of eating disorder psychopathology, subjective distress, impairments in quality of life, and psychiatric comorbidity at a level that is similar to other eating disorder diagnoses, implying that BED is characterized by clinically significant levels of psychopathology. Moreover, BED displays a clinical course that differs from both AN and BN and diagnostic crossover between AN and BED is extremely low. Thus, in relationship to other eating disorders, the diagnosis of BED seems to represent a diagnostically distinct entity that carries clinically useful information.

Second, can BED be discriminated from obesity and does the presence of BED confer clinically useful information beyond that associated with simple obesity or obesity with nonspecific psychopathology? There have been relatively few empirical classification studies that have included both obese BED subjects and obese non-BED subjects in taxometric analyses or latent class analyses. Thus, a definitive number of studies examining the presence of an objective boundary between BED and obesity using these methodologies has not yet been conducted. However, laboratory-based studies suggesting that individuals with BED clearly consume more calories than obese non-BED subjects provide some degree of support for the content validity of BED. The amount of support for the clinical utility of the BED diagnosis in relationship to obesity depends on the validating variable chosen. There is strong evidence that obese subjects with BED experience more psychiatric comorbidity, eating disorder psychopathology, subjective distress, and impairments in quality of life than do obese non-BED subjects. Evidence that BED predicts a clinical course associated with negative medical and psychiatric outcomes is limited and would bolster the clinical utility of the diagnosis. The finding that BED responds to some psychological treatments more completely than to nonspecific treatments provides evidence that the diagnosis has clinical utility for treatment of binge eating behavior, but treatment-related evidence supporting the validity of the diagnosis in relation to other diagnostic groups is rare.

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## NOTES