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Binge Eating Disorder: Recognition, Diagnosis, and Treatment

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A new diagnostic classification within the eating disorders group called "binge eating disorder" (BED) has been proposed in the DSM-IV. BED identifies a group of patients who regularly engage in binge eating without the regular use of compensatory purging. These patients appear to manifest a primary disturbance in eating behavior, although in some cases the binge eating may be a secondary symptom of depression and/or anxiety. The recurrent and chronic binge eating associated with BED clearly predisposes patients to the morbidity and mortality associated with obesity. Like bulimia nervosa, BED is associated with significant but generally less severe psychiatric comorbidity, including affective, anxiety, and personality disorders. The diagnosis, history, epidemiology, psychiatric comorbidity, and treatment of this proposed disorder are reviewed in this article.

Binge eating disorder (BED) has been proposed as a diagnostic entity and is now listed in the appendix of the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition (DSM-IV).[1-6] BED is defined by recurrent episodes of binge eating at least 2 days a week for at least 6 months. In addition, there is a subjective sense of a loss of control over binge eating, which is indicated by the presence of 3 of 5 specific criteria. These include eating rapidly, eating when not physically hungry, eating when alone, eating until uncomfortably full, and feeling self-disgust about bingeing.

Albert Stunkard[7,8] first described binge eating in a subset of obese patients and coined the term "night eating syndrome" (NES), which is similar to but distinct from BED. The newer, evolved concept of BED does not have the nocturnal component as a requirement. In NES, binge eating occurs nocturnally and is followed by morning anorexia and food restriction, which is thought to contribute to the next cycle of overeating. Other unofficial but related terms have appeared in the literature to describe individuals with binge eating not complicated by purging, such as "obese binge eaters" or "compulsive overeaters."[9-11] Kornhaber[12] described the "stuffing syndrome" in 1970. Since the publication of the DSM-III in 1980, these individuals have been officially, yet nonspecifically, classified as having an "eating disorder not otherwise specified (EDNOS).[13]

The first acknowledgment of binge eating in American psychiatry's diagnostic classification system occurred in the DSM-III; designated "bulimia," it encompassed not only bingeing but purging and preoccupation with body shape and weight as well. The revised edition of the DSM-III (DSM-III-R), published in 1987, adopted the term "bulimia nervosa,"[14] which was coined by Gerald Russell in 1979. Russell conceptualized this syndrome as "an ominous variant of anorexia nervosa."[15] Binge eating per se, without counteractive weight-reducing behaviors, was not identified as a major psychiatric disorder or problem until the recent inclusion of BED in the DSM-IV appendix.[1]

As our knowledge base about psychiatric disorders in general has increased over the years, our diagnostic classification system has evolved to describe them more accurately. Within this overall process, the eating disorders have only recently received serious research interest. The inclusion of nonpurging binge eating as an illness is a natural extension of this evolving process. Like bulimia first, and then bulimia nervosa, the diagnostic classification of BED will allow this group of patients to be further studied from a clinical research perspective and also to receive more accessible and appropriate treatment. In my view, BED depicts a serious psychologic problem that has been heretofore underrecognized and undertreated. However, the exact boundaries of BED remain to be further clarified, and it is likely that the criteria will continue to evolve as our knowledge base increases.

One of the major controversies regarding the diagnosis of BED includes its differentiation from nonpurging bulimia nervosa as currently defined in DSM-IV.[16] Nonpurging bulimia nervosa involves fasting and excessive exercise as compensatory behaviors, as well as preoccupation with body shape and weight.[1] However, the similarities between these 2 conditions appear to outweigh their relatively minor behavioral differences. In clinical practice, these disorders tend not to be distinct entities but exist on a continuum. Patients also go in and out of the criteria over time. It is very difficult clinically to distinguish between what are appropriate weight loss measures to combat obesity versus the excessive amount of counteractive exercise that characterizes nonpurging bulimia

nervosa. In addition, both obese bingers[17,18] and BED patients have been reported to have similar attitudes about body weight and shape, as compared with both nonpurging[19] and purging bulimia nervosa patients.[20] Regardless of the appellation, it is clear from epidemiologic studies that a meaningful number of patients have clinically significant binge eating and related psychopathology, not complicated by purging, that warrants treatment.

In the laboratory, BED patients have been shown to eat significantly more calories during a binge meal than non-BED obese patients.[21,22] (Simple obesity is defined as a BMI>=30). Dietary restraint and/or disinhibition appear to play major roles in triggering binge episodes.[23-25]

As discussed above, the occurrence of binge eating in a subset of obese individuals has been noted by clinical investigators for some time. As a logical outgrowth of this work, the prevalence rate of BED was first reported in cohorts of obese patients attending weight loss clinics or programs.[3-5] In these samples, 20% to 46% of subjects were reported to meet BED criteria using self-report measures.[3,5,10,11] However, it is important to observe that patients tend to overestimate the presence of binge eating on self-report questionnaires, as opposed to the prevalence rates gained from structured interviews using standardized criteria, such as the Questionnaire of Eating and Weight Patterns.[8,26,27] Spitzer and colleagues[3] reported that the prevalence of BED in weight control samples as assessed by questionnaire was approximately 30%, with the rate being slightly higher in females than males. In 2 field studies of nonpatient community samples,[3] these authors reported BED prevalences of 3.3% and 4.6%, with the rates being comparable in females and males (5.3% vs 3.1%). In a college student sample,[3] the rate was 2.6%, and there was no significant difference in the rates between females and males, a striking difference between BED and bulimia nervosa patients. The validity of BED was supported by associations with impaired work and social functioning, overconcern with body shape and weight, general psychopathology, and amount of time on diets. No significant racial differences were found in BED prevalence rates in these studies.

A study of a representative sample of 3006 adult women in the US was carried out by our group using a structured telephone interview based on DSM-III-R and proposed DSM-IV criteria.[28-30] Target households were identified by random digit dialing and were taken from four stratified regions of the US. We found that 1.0% of adult women met lifetime BED criteria, with about two thirds of these women meeting current criteria (6- and 12-month prevalence). BED respondents were distinct from another 2.4% of women who met lifetime criteria for bulimia nervosa. Surprisingly, there were no significant differences in age, weight, or race between respondents with BED and respondents with bulimia nervosa, although both groups were significantly younger and heavier than non-eating-disordered respondents. Because these results were obtained from a carefully, controlled representative sample of US women, they confirm that a substantial number of American adult women have clinically significant problems with binge eating not complicated by purging. When the binge duration criteria were relaxed from 6 to 3 months, the rate of BED increased from 1.0% to 1.6%.[28]

In a community study from California using a structured telephone interview, 1.8% of 455 adult women met DSM-IV BED criteria.[31] Another 3.8% of women met all but the frequency criteria for BED.

In a questionnaire-based community study from Norway involving 1849 adult women, the lifetime prevalence of BED was 3.2%.[32] And a similar study from France[33] based on a self-report questionnaire found a 9% to 15% BED rate in weight control samples and a 0.7% rate in a community sample of 447 women who were not patients. Although these studies have major methodologic differences, the results suggest that the prevalence of BED, like that of bulimia nervosa, may vary by culture and country.

The medical comorbidity associated with BED is essentially the same as that associated with obesity, including increased morbidity and mortality from cardiovascular disease, hyperlipidemia, adult-onset diabetes mellitus, and certain cancers, such as endometrial and breast cancers. This risk increases linearly as weight or body mass index (BMI; weight divided by height squared, or kg/m^2) increases. Because of the increasingly recognized overlap between obesity and psychiatric disorders,[34] and society's continued stigmatization of both the obese and the mentally ill, psychiatric input is going to be increasingly required for the optimal treatment of these patients. This relationship is further complicated by the fact that many psychotropic medications, as well as some nonpsychotropic drugs, are associated with weight gain and other possible medical complications. Obese patients with BED have been reported to have greater degrees of eating and weight-related pathology, as well as body image distortion and preoccupation, when compared with non-BED obese patients.[11,35,36]

Obese patients with BED who attend weight loss clinics have been reported to have a harder time remaining in weight loss programs and losing weight.[10,37] However, in one controlled study comparing BED obese and non-BED obese patients, the presence of BED did not affect weight-loss outcome or dropout rate.[38] In a community study, Ferguson and Spitzer[39] reported that unsuccessful dieters were more likely to meet BED criteria than successful dieters. No differences in resting metabolic rate, thyroid hormone levels, or serum lipid levels between obese bingers and obese nonbingers has been reported.[40,41] Obese bingers were reported to have a higher degree of weight cycling in one study,[42] but not in another.[41]

The relationship between BED and other psychiatric comorbidity has been of major clinical and research interest. A number of investigators have reported that a subset of obese patients engage in overeating or bingeing in response to emotional stress, so-called "emotional eating."[43,44] BED patients have a greater tendency to overeat in response to negative mood states than other patients.[19] Systematic studies of obese patients meeting BED criteria indicate higher-than-expected rates of affective, anxiety, and personality disorders, in addition to emotional problems in general.[40,42,44-49] In one study of 107 obese women with BED, a significant positive relationship was found between severity of binge eating and degree of psychiatric symptomatology, as measured by several psychometric instruments (Binge Eating Scale [BES] SCL-90, Beck Depression Inventory, Inventory of Interpersonal Problems, Rosenberg Self-Esteem Scale).[44] DeZwaan and colleagues[42] also found an association between binge eating and a number of measures of psychopathology (HAM-A, HAM-D, Three-Factor Questionnaire, Binge Eating Scale, Eating Disorder Inventory, Beck Depression Inventory, New York State Self-Esteem Scale.)

In the National Women's Study, Dansky and colleagues[28] found that the lifetime prevalence of major depression was 31% in BED respondents and 36% in bulimia nervosa respondents. Both of these rates were significantly higher than the 15% rate of major depression in the nonbingeing comparative group. It is notable that major depression was not present in the majority of respondents, given that some BED opponents argue that binge eating is merely a symptom, albeit atypical, of depression, but these results do not support this assertion in most people with BED. A recent study of 30 BED patients vs. 30 non-BED patients confirms the finding that dysphoric emotional states often trigger binge eating episodes and a sense of loss of control.[50] However, these patients are not necessarily clinically depressed at the time of bingeing. In one study of the chronological relationship between the times of onset of bingeing, dieting, and depression, it was found that BED subjects tended to begin bingeing during adolescence and prior to the onset of depression, dieting, or obesity.[51] Nevertheless, the higher rates of depression and anxiety associated with BED support an affect-regulation hypothesis for binge eating.

Dansky and associates[28] also found that the lifetime prevalence of posttraumatic stress disorder (PTSD) was 21% in BED respondents compared with 9% in nonbingeing respondents. Unlike bulimia nervosa, rates of criminal-victimization experiences (including rape, molestation, attempted sexual assault, and aggravated assault) were comparable to the non-BED/non-bulimia nervosa group. However, given the higher rate of lifetime PTSD, the subjects with BED may have been exposed more often to other types of traumatic experiences or stressors than were subjects without BED. In a clinical sample, Yanovski and coworkers[47] also failed to find a difference in reported rates of sexual abuse in BED versus non-BED obese subjects. However, BED patients did have significantly higher rates of panic disorder and personality disorder in this study.

Clinical experience dictates that BED patients often report histories of significant family dysfunction, if not overt childhood physical and emotional abuse and/or neglect. Hodges and colleagues[52] studied the perceived family environments of 131 eating disorder patients presenting for evaluation and treatment, including 43 patients with BED.[52] Scores on the Family Environment Scale (FES) indicated less cohesion in the families of BED patients compared with the families of anorexia nervosa, but not bulimia nervosa, patients. In addition, lower scores were found on the activity-recreation subscales for the BED group compared with all other eating disorder subtypes (anorexia nervosa, bulimia nervosa, and anorexia nervosa plus bulimia nervosa). The BED group also had higher conflict and control subscale scores and lower cohesiveness, expressiveness, independence, intellectual-cultural, and activity-recreation subscale scores compared with 2 normal control samples.

Higher rates of impulsive behaviors, such as kleptomania and compulsive buying, have been reported in patients with BED.[53] Likewise, higher rates of cluster B and C personality disorders have been reported in patients with BED.[46,34] Although rates of substance abuse disorders were not significantly higher in obese BED patients compared with obese non-BED patients, the rate of

alcoholism in family members of BED patients was significantly higher.[47] Given these relationships, patients with BED have been hypothesized to fall within the continuum of compulsive-impulsive disorders[53] and affective spectrum disorders.[54]

Behavioral treatments for obesity have been shown to work repeatedly, but only in the short term for the vast majority of patients.[37] Patients with BED appear to be more resistant to these commonly employed strategies and are more likely to relapse in the long-term, even if initially successful. In fact, it may be that dietary restraint (ie, dieting) has a disinhibiting effect on "binge eating," thereby contributing to the marked weight fluctuations that these patients often manifest. In addition, emotional issues and psychiatric comorbidity are not typically addressed in purely behavioral forms of treatment. These patients may have a variety of needs that are best approached from the standpoint of a biopsychosocial model. Therefore, a multidisciplinary approach is often required, including working with the patient's internist or family practitioner, dietitian, psychotherapist, and physical therapist. A common philosophy of treatment is to put the goal of weight loss on the "back-burner" initially. Decreasing binge eating by normalizing eating behavior and addressing associated emotional symptoms and/or psychiatric disorders must take precedence for successful treatment to occur.

Guided by the successes in the treatment of bulimia nervosa,[55,56] depression, and anxiety disorders, recent studies using sophisticated, manual-driven, cognitive-behavioral therapy (CBT) have shown promise in the treatment of BED. This form of psychotherapy pays particular attention to the patient's behavior and thinking rather than the underlying feelings or psychodynamics. There have been only a few controlled trials of CBT in BED so far. In a 10-week study of CBT versus waiting-list controls in 44 women with nonpurging bulimia, Telch and colleagues[57] found a 94% decrease in the frequency of binge eating episodes, while the waiting-list controls showed a decrease of only 9%. Seventy-nine percent of the CBT group became completely abstinent from bingeing.

In a similar study comparing 10 weeks of group CBT versus group interpersonal psychotherapy (IPP) versus waiting-list controls in 46 nonpurging bulimic patients, Wilfrey and associates[58] found that the number of binge days per week decreased by 48% during group CBT, 71% during group IPP, and 10% during the wait-list period. However, Agras and coworkers[59] reported that IPP offered no added benefit to BED patients unresponsive to CBT. In a study of obese binge eaters, Smith and others[60] reported an 81% decrease in the frequency of binge eating episodes following 16 weeks of CBT, but there was no control group in this study.

Given the available data, treatment should initially focus on the reduction of binge eating per se as well as on eating regular meals with little or no snacking, particularly before bedtime. In addition, treatment should identify and challenge cognitive distortions. If binge eating and the associated lack of restraint and disinhibition are successfully controlled, then some degree of weight loss may become an automatic secondary effect. Patients may have more energy to embark on a mild-to-moderate exercise regimen and may also be generally less depressed and anxious. However, in patients unresponsive to behavioral and/or psychotherapeutic treatments, psychopharmacologic approaches should be considered.

Because of its strong links to affective illness and other disorders linked to serotonin dysregulation,[61] clinical investigators have hypothesized that the selective serotonin reuptake inhibitors (SSRIs) would be a good treatment for both obesity and BED (Table I). A double-blind placebo-controlled study (N=45) of fluoxetine in the treatment of obesity showed an early weight loss response, but this effect completely disappeared by the end of 1 year on the drug.[62] In another study of fluoxetine in 45 obese patients (with and without binge eating), Marcus and colleagues[63] reported that patients who received fluoxetine plus behavior modification therapy lost significantly more weight than those on placebo and behavior modification.[63] This difference between fluoxetine and placebo persisted regardless of the presence of binge eating; however, the sample sizes of these subsets were too small for definitive conclusions regarding the similarities and differences between bingeing and nonbingeing obese patients.

Preliminary open-label studies of fluvoxamine and paroxetine in BED patients have indicated significant reductions in binge frequency.[54] More controlled studies of BED with new-generation antidepressants are clearly needed, but this class of drugs holds promise. In particular, sertraline, venlafaxine, and nefazodone are also likely to significantly impact binge eating favorably given their serotonin reuptake inhibition.

Tricyclic antidepressants. Some data exist regarding the possible role of tricyclic antidepressants (TCAs) in the treatment of BED.

In a 12-week study of 23 women with nonpurging bulimia, McCann and Agras[64] reported that desipramine reduced binge eating by 63% compared with a 6% increase reported with placebo. In another study of 33 bingeing obese patients and 22 patients with bulimia nervosa, Alger and associates[65] reported no significant difference in binge frequency following treatment with imipramine or naltrexone versus placebo. However, imipramine significantly reduced binge duration in bingeing obese patients. Tricyclic antidepressants, especially desipramine, may therefore play a role in the treatment of BED, as they do in bulimia nervosa. However, in clinical practice, it is generally recommended that the first-line psychopharmacologic treatment strategy involve an SSRI.[54]

TCAs are often associated with weight gain, probably resulting from a combination of hyperphagia induction via stimulation of noradrenergic pathways in the hypothalamus and a decrease in metabolic rate. The use of MAOIs is of questionable value in a population that loses control over eating and thus would have difficulty maintaining the necessary food restrictions. However, MAOIs could be a consideration in clear-cut cases of atypical depression that are unresponsive to SSRIs, venlafaxine, nefazodone, desipramine, and/or dexfenfluramine.

There has been more recent interest in dexfenfluramine (dextrorotatory fenfluramine) since its US release in 1996 for the treatment of obesity. In a large double-blind placebo-controlled study in 4 European countries, dexfenfluramine has been shown to significantly reduce weight loss by an average of 10% over the course of 1 year.[66] This amount of weight loss, although modest, is known to significantly reduce medical comorbidity in obese patients (eg, improving hypertension, hyperlipidemia, and glycemic control). Notably, depression, headache, asthenia, and diarrhea were the major reasons given by subjects for study discontinuation. Dexfenfluramine appears to have little or no potential for abuse, given that animal studies indicate no hedonic-reinforcing properties (similar to saline).

Studies of d,l-fenfluramine (racemic fenfluramine) have shown that acute administration significantly reduces binge eating in bulimic subjects.[67] Drug trials in patients with bulimia nervosa have been equivocal, but suggest a possible beneficial effect of fenfluramine in certain cases.[68,69] Its use must be weighed carefully against the rare life-threatening adverse effect of primary pulmonary hypertension, which is estimated to occur in 23 to 46 cases per million annually, compared with 1 to 2 cases per million annually in the general population.[70] The use of dexfenfluramine is also limited by the manufacturer's relative contraindication for concomitant SSRI use. Concomitant use of SSRIs and dexfenfluramine runs the risk of inducing the serotonin syndrome, which is characterized by mental status change (delirium, hypomania), hypertonus, myoclonus, restlessness, tremor, diaphoresis, shivering, and hyperreflexia. But even more importantly, an SSRI or TCA is likely to inhibit the uptake of dexfenfluramine into the presynaptic neuron, which is required for its therapeutic action, thereby negating its effect on serotonin release. There have been reports of adverse effect from racemic fenfluramine withdrawal (eg, depression), so this agent and dexfenfluramine should be tapered slowly, never abruptly, when discontinued.[71,72]

Results from a recently published double-blind controlled study of dexfenfluramine in BED patients indicates a significant 3 times reduction in binge eating as compared with that for placebo.[73] After controlling for the effects of baseline weight and depression scores, the magnitude of dexfenfluramine's effect over placebo was increased. However, it is important to note that binge eating frequency returned to pretreatment levels after the drug was discontinued, much like the weight gain that usually occurs upon discontinuation of all anorexiants. Although related, it may be that the pharmacologic mechanisms underlying reduction in binge eating are significantly different from those underlying weight reduction.

There are no published reports on the use of psychostimulants in the treatment of BED. Even though acutely administered stimulants suppress binge eating,[74] the risks of addiction and the possible induction of affective and psychotic symptomatology make this agent class undesirable as a therapeutic tool.

The opiate antagonists show some possible therapeutic potential in the pharmacologic treatment of BED. In an acute challenge study, Marrazzi and colleagues[75] studied naltrexone in double-blind placebo-controlled fashion in 1 BED subject and found that naltrexone significantly reduced binge frequency and urges to binge. However, this finding contrasts with the study by Alger and associates[65] in which naltrexone was no different from placebo in obese bingers (but naltrexone did reduce bingeing in bulimic subjects). The opiate antagonist naloxone has also been reported to significantly reduce binge eating in BED patients.[76] Naloxone significantly suppressed energy intake relative to saline in binge eaters but not in nonbinge eaters, and butorphanol had no

significant effect on food intake.[76] These studies may be relevant to the finding that obese BED subjects have significantly higher pain detection thresholds compared with non-BED obese patients and normal controls.[77]

In the only study so far that assessed the combination of psychotherapy with medication, the addition of desipramine did not increase the anti-binge eating effect of CBT. However, weight loss was facilitated by the combination.

BED is a well-validated diagnostic entity proposed in DSM-IV that is characterized by recurrent binge eating without purging of any kind. It is distinguished from bulimia nervosa, nonpurging type, by the absence of fasting or excessive exercise as a way of "undoing" the weight-promoting effects of bingeing. However, in the clinical setting, these conditions overlap considerably, and it is difficult to distinguish them from each other. BED occurs in approximately 1% of women in the US and in a sizable proportion of those seeking weight loss in bariatric programs. It is important that the diagnosis is not based on self-report alone but also on clinical interview. BED carries specific comorbidities, especially obesity, major depression, and anxiety disorders (particularly panic disorder and PTSD). Treatment approaches show promise in both the psychotherapeutic and psychopharmacologic realms. It is prudent to start with CBT and to aggressively treat associated psychiatric comorbidity, perhaps with an SSRI initially. If this fails, a trial of desipramine, dexfenfluramine, nefazodone, venlafaxine, naltrexone or naloxone is a consideration. As in other psychiatric and medical disorders, the benefits versus the risks (of both treatment and nontreatment) must be weighed carefully.

- Binge Eating Disorder. An overview of the new diagnostic classification known as binge eating disorder (BED). Topics addressed include epidemiologic antecedents, physiologic/psychiatric comorbidities, and proposed strategies of psychopharmacologic management. Provided by Healthwatch Online.
- Position of The American Dietetic Association: Nutrition Intervention in the Treatment of Anorexia Nervosa, Bulimia Nervosa, and Binge Eating. A commentary on the onset and psychopathology of anorexia and bulimia nervosa. Issues addressed include the validity of the proposed diagnostic entity, binge eating disorder, to classify individuals presenting with binge eating without concomitant compensatory behaviors.
- Symptoms of Binge Eating Disordered. A brief outline detailing the behavioral presentations associated with binge eating disorder. Provided by The Center for Eating Disorders, St. Joseph Medical Center.
- Treatment of Anorexia Nervosa, Bulimia Nervosa and Binge Eating. A report on the origin and psychophysiology of eating disorders. Specific focus is placed upon binge eating disorder as a distinct diagnostic classification, as well as on the characteristic behaviors associated with recurrent and chronic episodes. Provided by Addictions & More.

References

1. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, ed 4. American Psychiatric Press, Washington, DC, 1994.
2. Spitzer RL, Devlin MJ, Walsh BT, et al: Binge eating disorder: To be or not to be in DSM-IV. Int J Eat Disord 10:627-629, 1991.
3. Spitzer RL, Yanovski S, Wadden T, et al: Binge eating disorder: Its further validation in a multisite study. Int J Eat Disord 13:137-153, 1993.
4. Spitzer RL, Stunkard A, Yanovski S, et al: Binge eating disorder should be included in DSM-IV: A reply to Fairburn et al's "The Classification of recurrent overeating: The binge eating disorder proposal." Int J Eat Disord 13:161-169, 1993.
5. Fairburn CG, Welch SL, Hay PJ: The classification of recurrent overeating: The "Binge Eating Disorder" proposal. Int J Eat Disord 13:155-159, 1993.
6. Brody ML, Walsh BT, Devlin MJ: Binge eating disorder: Reliability and validity of a new diagnostic category. J Consult Clin

Psychol 62:381-386, 1994.

7. Stunkard AJ: Eating patterns and obesity. Psychiatric Quarterly 33:284-294, 1959.
8. Stunkard AJ, Berkowitz R, Wadden T, et al: Binge eating disorder and the night-eating syndrome. Int J Obes 20:1-6, 1996.
9. Loro AD, Jr, Orleans CS: Binge eating in obesity: Preliminary findings and guidelines for behavioral analysis and treatment. Addict Behav 6:155-166, 1981.
10. Marcus MD, Wing RR: Binge eating among the obese. Ann Behav Med 9:23-27, 1987.
11. de Zwaan MD, Mitchell JE: Binge eating in the obese. Ann Medicine 24:303-308, 1992.
12. Kornhaber A: The stuffing syndrome. Psychosomatics 11:580-584, 1970.
13. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, ed 3 rev, American Psychiatric Press, Washington, DC, 1980.
14. American Psychiatric Association: Diagnostic and Statistical Manual of Mental Disorders, ed 3 rev, American Psychiatric Press, Washington, DC, 1987
15. Russell GFM: Bulimia nervosa, an ominous variant of anorexia nervosa. Psychol Med 9:429-448, 1979.
16. Spitzer RL: Nonpurging bulimia nervosa and binge eating disorder. Am J Psychiatry 148:1097-1098, 1991.
17. Cash TF: Binge-eating and body images among the obese: A further evaluation. J Soc Behav Personality 6:367-376, 1991.
18. Wilson GT, Nonas CA, Rosenblum GD: Assessment of binge eating in obese patients. Int J Eat Disord 13:25-33, 1993.
19. Eldredge KL, Agras WS: Weight and shape overconcern and emotional eating in binge eating disorder. Int J Eat Disord 19:73-82, 1996.
20. Raymond NC, Mussell MP, Mitchell JE, et al: An age-matched comparison of subjects with binge eating disorder and bulimia nervosa. Int J Eat Disord 18:135-143, 1995.
21. Goldfein JA, Walsh BT, LaChaussee JL, et al: Eating behavior in binge eating disorder. Int J Eat Disord 14:427-431, 1993.
22. Yanovski SZ, Leet M, Yanovski JA, et al: Food selection and intake of obese women with binge-eating disorder. Am J Clin Nutr 56:975-980, 1992.
23. Marcus MC, Wing RR, Lamparski DM: Binge eating and dietary restraint in obese patients. Addict Behav 10:163-168, 1985.
24. Yanovski SZ, Sebring NG: Recorded food intake of obese women with binge-eating disorder before and after weight loss. Int J Eat Disord 15:135-150, 1994.
25. Yanovski SZ: The chicken or the egg: Binge eating disorder and dietary restraint. Appetite 24:258, 1995.
26. Greeno CG, Marcus MD, Wing RR: Diagnosis of binge eating disorder: Discrepancies between a questionnaire and clinical interview. Int J Eat Disord 17:153-160, 1995.
27. Nangle DW, Johnson WG, Carr-Nangle REC, et al: Binge eating disorder and the proposed DSM-IV criteria: Psychometric analysis of the Questionnaire of Eating and Weight Patterns. Int J Eat Disord 16:147-157, 1994.

28. Dansky BS, Brewerton TD, Kilpatrick DG, et al: The nature and prevalence of binge eating disorder in a national sample of women, in Widiger TA, Frances AJ, Pincus HA, et al (eds): DSM-IV Sourcebook, Washington, DC, APA Press, Inc. In press.

29. Brewerton TD, Dansky BS, Kilpatrick DG, et al: The prevalence of binge eating disorder in United States women. Abstracts from the proceedings of the 149th Annual Meeting of the American Psychiatric Association, New York, NY, May 5-9, 1996, p 76.

30. Dansky BS, Brewerton TD, O'Neil PM, et al: The National Women's Study: Relationship of crime victimization and PTSD to bulimia nervosa. Int J Eat Disord 21:213-228, 1997.

31. Bruce B, Agras WS: Binge eating in females: A population-based investigation. Int J Eat Disord 12:365-375, 1992.

32. Gotestam KG, Agras WS: General population-based epidemiological study of eating disorders in Norway. Int J Eat Disord 18:119-126, 1995.

33. Basdevant A, Pouillon M, Lahlou N, et al: Prevalence of binge eating disorder in different populations of French women. Int J Eat Disord 18:309-315, 1995.

34. Specker S, de Zwaan M, Raymond N, et al: Psychopathology in subgroups of obese women with and without binge eating disorder. Comprehensive Psychiatry 35:185-190, 1994.

35. Mussell MP, Mitchell JE, de Zwaan M, et al: Clinical characteristics associated with binge eating in obese females: A descriptive study. Int J Obes Relat Disord 20:324-331, 1996.

36. Mussell MP, Peterson CB, Weller CL, et al: Differences in body image and depression among obese women with and without BED. Obes Res 4:431-439, 1996.

37. Keefe PH, Wyshogrod D, Weinberger E, et al: Binge eating and outcome of behavioral treatment of obesity: A preliminary report. Behav Res Ther 22:319-321, 1984.

38. Ho KSI, Nichman MZ, Taylor WC, et al: Binge eating disorder, retention, and dropout in adult obesity program. Int J Eat Disord 18:291-294, 1995.

39. Ferguson DJ, Spitzer RL: Binge eating disorder in a community-based sample of successful and unsuccessful dieters. Int J Eat Disord 18:167-172, 1995.

40. Wadden TA, Foster GD, Letizia KA, et al: Metabolic, anthropometric, and psychological characteristics of obese binge eaters. Int J Eat Disord 14:17-25, 1993.

41. Adami GF, Gandolfo P, Campostano A, et al: Obese binge eaters: Metabolic characteristics, energy expenditure and dieting. Psychol Med 25:195-198, 1995.

42. de Zwaan MD, Mitchell JE, Seim HC, et al: Eating related and general psychopathology in obese females with binge eating disorder. Int J Eat Disord 15:43-52, 1994.

43. Ganley RM: Emotions and eating in obesity: A review of the literature. Int J Eat Disord 8:343-361, 1989.

44. Telch CF, Agras WS: Obesity, binge eating and psychopathology: Are they related? Int J Eat Disord 15:53-61, 1994.

45. Marcus MD, Wing RR, Ewing L, et al: Psychiatric disorders among obese binge eaters. Int J Eat Disord 9:69-77, 1990.

46. Fichter MM, Quadflieg N, Brandl B: Recurrent overeating: An empirical comparison of binge eating disorder, bulimia nervosa,

and obesity. Int J Eat Disord 14:1-16, 1993.

47. Yanovski SZ, Nelson JE, Dubbert BK, et al: Association of binge eating disorder and psychiatric comorbidity in obese subjects. Am J Psychiatry 150:1472-1479, 1993.

48. Antony MM, Johnson WG, Carr-Nangle RE, et al: Psychopathology correlates of binge eating and binge eating disorder. Compr Psychiatry 35:386-392, 1994.

49. Kuehnel RH, Wadden TA: Binge eating disorder, weight cycling, and psychopathology. Int J Eat Disord 15:321-329, 1995.

50. Telch CF, Agras WS: Do emotional states influence binge eating in the obese? Int J Eat Disord 20:271-279, 1996.

51. Mussell MP, Mitchell JE, Weller CL, et al: Onset of binge eating, dieting, obesity, and mood disorders among subjects seeking treatment for binge eating disorder. Int J Eat Disord 17:395-401, 1995.

52. Hodges EL, Cochrane CE, Brewerton TD: Family characteristics of binge eating disorder. Int J Eat Disord, 1997. In press.

53. McElroy SL, Keck PE, Phillips KA: Kleptomania, compulsive buying, and binge eating disorder. J Clin Psychiatry 56(suppl 4):S14-S26, 1994.

54. Hudson JI, Carter WP, Pope HG: Antidepressant treatment of binge eating disorder: Research findings and clinical guidelines. J Clin Psychiatry 57(suppl 8):S73-S79, 1988.

55. Mitchell JE, Pyle R, Eckert ED, et al: A comparison of antidepressants and structured intensive group psychotherapy in the treatment of bulimia nervosa. Arch Gen Psychiatry 47:149-157, 1990.

56. Fairburn CG, Jones R, Peveler RC, et al: Three psychological treatments for bulimia nervosa. Arch Gen Psychiatry 48:463-469, 1991.

57. Telch CF, Agras WS, Rossiter EM, et al: Group cognitive-behavioral treatment for the nonpurging bulimic: An initial evaluation. J Consult Clin Psychol 58:629-635, 1990.

58. Wilfrey DE, Agras WS, Telch CF, et al: Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: A controlled comparison. J Consult Clin Psychol 61:296-305, 1993.

59. Agras WS, Telch CF, Arnow B, et al: Does interpersonal therapy help patients with binge eating disorder who fail to respond to cognitive-behavioral therapy? J Consult Clin Psychol 63:356-360, 1995.

60. Smith DE, Marcus MD, Kaye W, et al: Cognitive-behavioral treatments of obese binge eaters. Int J Eat Disord 12:257-262, 1992.

61. Brewerton TD: Toward a unified theory of serotonin dysregulation in eating and related disorders. Psychoneuroendocrinology 20:561-590, 1995.

62. Ferguson DM, Feighner JP: Fluoxetine-induced weight loss in overweight non-depressed humans. Int J Obes 11(suppl 3):S163-S170, 1987.

63. Marcus MD, Wing RR, Ewing L, et al: A double-blind, placebo-controlled trial of fluoxetine plus behavior modification in the treatment of obese binge-eaters and non-binge eaters. Am J Psychiatry 147:876-881, 1990.

64. McCann UD, Agras WS: Successful treatment of nonpurging bulimia nervosa with desipramine: A double-blind, placebo-controlled study. Am J Psychiatry 147:1509-1513, 1990.

65. Alger SA, Schwalberg MD, Bigaouette JM, et al: Effect of a tricyclic antidepressant and opiate antagonist on binge-eating behavior in normoweight bulimic and obese, binge-eating subjects. Am J Clin Nutr 53:865-871, 1991.

66. Guy-Grand B, Appelbaum M, Crepaldi G, et al: International trial of long-term dexfenfluramine in obesity. Lancet 2:1142-1144, 1989.

67. Robinson PH, Checkley SA, Russell GFM: Suppression of eating by fenfluramine in patients with bulimia nervosa. Br J Psychiatry 146:169-176, 1985.

68. Russell GFM, Checkley SA, Feldman J, et al: A controlled trial of d-fenfluramine in bulimia nervosa. Clin Neuropharmacol 11(suppl 1):S146-S159, 1988.

69. Blouin AG, Blouin JH, Perez EL, et al: Treatment of bulimia with fenfluramine and desipramine. J Clin Psychopharmacol 8:261-269, 1988.

70. Abenhaim L, Moride Y, Brenot F, et al: Appetite-suppressant drugs and the risk of primary pulmonary hypertension: International Primary Pulmonary Hypertension Study Group. N Engl J Med 335:609-616, 1996.

71. Steel JM, Briggs M: Withdrawal depression in obese patients after fenfluramine treatment. Br Med J 3:26-27, 1972.

72. Harding T: Depression following fenfluramine withdrawal. Br J Psychiatry 121:338-339, 1972.

73. Stunkard AJ, Berkowitz R, Tanrikut C, et al: d-fenfluramine treatment of BED. Am J Psychiatry 153:1455-1459, 1996.

74. Ong YL, Checkley SA, Russell GFM, et al: Suppression of bulimic symptoms with methylamphetamine. Br J Psychiatry 143:288-293, 1983.

75. Marrazzi MA, Markham KM, Kinzie J, et al: Binge eating disorder: Response to naloxone. Int J Obes 19:143-145, 1995.

76. Drewnowski A, Krahn DD, Demitrack MA, et al: Naloxone, an opiate blocker, reduces the consumption of sweet high-fat foods in obese and lean female binge eaters. Am J Clin Nutr 61:1206-1212, 1995.

77. Raymond NC, de Zwaan M, Faris PL, et al: Pain thresholds in obese binge-eating disorder subjects. Biol Psychiatry 37:202-204, 1995.

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