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CME Review Article

Binge eating disorder revisited: what's new, what's different, what's next

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- Implement evidence-based treatment in the management of patients with eating disorders
- Incorporate a multidisciplinary approach in the management of patients with eating disorders

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REVIEW ARTICLE

Binge eating disorder revisited: what's new, what's different, what's next

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Binge eating disorder (BED) is the most common type of eating disorder. According to the most recent data available, the estimated lifetime prevalence of BED among US adults in the general population is 0.85% (men 0.42% and women 1.25%). Among psychiatric treatment populations, prevalence is several-fold higher. Although many people with BED are obese (BMI ≥ 30 kg/m^2), roughly half are not. In the DSM-5, BED is defined by recurrent episodes of binge eating (eating in a discrete period of time, an amount of food larger than most people would eat in a similar amount of time under similar circumstances *and* a sense of lack of control over eating during the episode), occurring on average at least once a week for 3 months, and associated with marked distress. BED often goes unrecognized and thus untreated; in one study, 344 of 22,387 (1.5%) survey respondents met DSM-5 criteria for BED, but only 11 out of the 344 had ever been diagnosed with BED by a health-care provider. Psychiatric comorbidities are very common, with most adults with BED also experiencing anxiety disorders, mood disorders, impulse control disorders, or substance use disorders, suggesting that clinicians have patients in their practice with unrecognized BED. Multiple neurobiological explanations have been suggested for BED, including dysregulation in reward center and impulse control circuitry. Additionally, there is interplay between genetic influences and environmental stressors. Psychological treatments such as cognitive behavioral interventions have been recommended as first line and are supported by meta-analytic reviews; however, access to such treatments may be limited because of local availability and/or cost, and these treatments generally lead to little to no weight loss, although successfully eliminating binge eating can protect against future weight gain. Routine medication treatments for anxiety and depression do not necessarily ameliorate the symptoms of BED, but there are approved and emerging medication options, lisdexamfetamine and dasotraline, respectively, that specifically address the core drivers behind binge eating, namely obsessive thoughts and compulsive behaviors regarding food, resulting in marked decreases in binge eating behaviors as well as weight loss.

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Key words: Binge eating disorder, cognitive-behavioral therapy, dasotraline, inter-personal therapy, lisdexamfetamine.

Overview

Since the inclusion of binge eating disorder (BED) as a diagnosis in the fifth edition of Diagnostic and Statistical Manual of Mental Disorders (DSM-5) in 2013,[1] and the approval of lisdexamfetamine to treat moderate to severe BED in 2015,[2,3] interest in the disorder has continued at a rapid pace. A bibliometric search of the US National Library of Medicine's PubMed.gov resource using the text word query "binge eating disorder" revealed 2732 publications, of which 848 have appeared since the publication of the previous version of this review in December 2015.[4]

New information is available on the epidemiology of BED in the USA and provides somewhat lower prevalence estimates than prior reports,[5,6] but the data continue to follow the same patterns when comparing the prevalence and course of BED with other eating disorders. This new analysis is from a nationally representative sample of US adults using data from the 2012–2013 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC-III) comprising of over 36,000 respondents assessed with lay-administered diagnostic interviews.[7] Lifetime and 12-month prevalence of DSM-5-defined BED were 0.85% and 0.44%, respectively. Compared to other eating disorders, these rates are substantially higher than the estimate for lifetime and 12-month prevalence rates for anorexia nervosa (0.80% and 0.05%) or bulimia

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nervosa (0.28% and 0.14%). Women are more likely than men to be diagnosed with BED, with lifetime prevalence rates of 1.25% for women and 0.42% for men. Lifetime BED prevalence rates were 0.94% for non-Hispanic whites, 0.62% for non-Hispanic blacks, 0.75% for Hispanics, and 0.59% for "other" race/ethnicity. Compared with lifetime anorexia nervosa or bulimia nervosa, those with lifetime BED had a later age of onset of their eating disorder and a longer duration of their episodes. Although about 56% of people with lifetime BED in the NESARC-III analysis were obese (body mass index (BMI) ≥ 30 kg/m^2), 20% were normal weight (BMI 18.5–24.9 kg/m^2) and 23% overweight (25–29.9 kg/m^2).

BED is associated with distress and problems in functioning. Consequences of BED include problems adapting to social roles, poor quality of life and less life satisfaction due to health issues, increased overall medical morbidity and mortality related to weight gain and obesity, and increased use of health-care resources.[1] In one survey, impaired role functioning in work/school, social, and family life, as measured by the Sheehan Disability Scale, was observed in 63% of adults with BED, with 18.5% of adults with BED reporting severe functional impairment.[5] In the analysis of the NESARC-III data,[7] for lifetime diagnoses, rates of any impairment in social function were significantly greater for bulimia nervosa (61.4%) and BED (53.7%) than for anorexia nervosa (30.7%). Moreover, rates of reporting of interference with normal daily activities were significantly greater for BED (52.5%) and bulimia nervosa (49.5%) than for anorexia nervosa (23.5%).

Psychiatric comorbidities are very common, with one study noting that 79% of adults with BED also experience anxiety disorders (65%), mood disorders (46%), impulse control disorders (43%), or substance use disorders (23%).[5] Multiple psychiatric comorbidities are also common and almost 50% of persons with BED have ≥ 3 psychiatric comorbidities.[5] The psychiatric comorbidity is linked to the severity of binge eating and not to the degree of obesity.[1] In an additional analysis of the NESARC-III data,[8] the percentage reported for meeting criteria for a comorbid psychiatric disorder was even higher, at 94%, with BED significantly associated with any mood disorder, major depressive disorder, persistent depression, any anxiety disorder, all individual anxiety disorders (except for panic disorder), posttraumatic stress disorder, alcohol use disorder, any personality or conduct disorder, and all individual personality and conduct disorders. Moreover, after adjusting for sociodemographic variables, BED was significantly associated with increased odds of diabetes, hypertension, high cholesterol, high triglycerides, minor heart conditions, stomach ulcer, arthritis, sleep problems, anemia, fibromyalgia, bowel problems, osteoporosis, lung problems, liver diseases, and nerve problems. After additionally adjusting for other psychiatric disorders, odds ratios for diabetes, hypertension, high cholesterol, and high triglycerides remained significant.

It must be emphasized that BED is distinct from obesity; most obese individuals do not engage in recurrent binge eating. Moreover, compared with weight-matched obese individuals without BED, those with BED consume more calories in laboratory studies and have greater functional impairment, lower quality of life, more subjective distress, and greater psychiatric comorbidity.[1] In general, individuals with BED report being insufficiently physically active, more so than people who are overweight/obese or healthy weight individuals.[9]

Psychological treatments including cognitive behavioral therapy (CBT) and interpersonal therapy (IPT) have been recommended as first line[10,11] and are supported by several different meta-analytic reviews.[12–18] Although CBT and IPT can reduce binge eating behavior, access to such treatments may be limited because of local availability and/or cost. Moreover, 33–50% of patients with BED do not appear to benefit completely or sufficiently from psychological and behavioral treatment.[15] Also, psychological interventions for BED have evidenced little to no weight loss, although successfully eliminating binge eating can protect against future weight gain. Routine medication treatments for anxiety and depression do not necessarily ameliorate the symptoms of BED, but there are approved and emerging medication options, lisdexamfetamine and dasotraline, respectively, that specifically address the core drivers behind binge eating, namely obsessive thoughts and compulsive behaviors regarding food, resulting in marked decreases in binge eating behaviors as well as weight loss.[3]

Review of the DSM-5 diagnostic criteria for BED

In the DSM-5, BED is defined by recurrent episodes of binge eating (eating in a discrete period of time an amount of food larger than most people would eat in a similar amount of time under similar circumstances *and* a sense of lack of control over eating during the episode), occurring on average at least once a week for 3 months, and associated with marked distress.[1] Binge episodes are also associated with ≥ 3 of the following: eating rapidly, eating until feeling uncomfortably full, eating large amounts of food when not feeling physically hungry, eating alone because of feeling embarrassed by how much one is eating, and feeling disgusted with oneself, depressed, or guilty afterwards. It is not unusual for all these associated features to be present. Although overvaluation of shape or weight is often seen (40%), it is presently not part of the DSM-5 criteria for BED.[19–21] BED is distinguished from bulimia nervosa, in that BED is not associated with regular compensatory behaviors such as purging or excessive exercise, or with dietary restriction, although frequent dieting may be reported.

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Since binge eating is often a secretive behavior, and commonly associated with a high degree of embarrassment or shame, [22,23] it is not ordinarily revealed unless the clinician makes a direct inquiry regarding eating patterns. A screening instrument, based on the DSM-5 criteria, is available.[24,25]

Although physical comorbidities including obesity and metabolic syndrome are common, and may prompt a discussion of eating behaviors, not all persons with BED are obese. Because BED is commonly associated with other psychiatric disorders that bring them to treatment, such as anxiety, depression, impulse control disorders, and substance use, routine assessment of eating behaviors is advisable because persons with prominent coexisting psychiatric conditions, and as yet undiagnosed BED, may not bring up eating issues on their own.[26]

DSM-5 also provides for the specification of partial remission where after full criteria for BED were previously met, binge eating occurs at an average frequency of less than one episode per week for a sustained period of time. For full remission, after full criteria for BED were previously met, none of the criteria have been met for a sustained period of time.

Current severity can also be specified. The minimum level of severity is based on the number of weekly binge eating episodes (mild, 1–3; moderate, 4–7; severe, 8–13; extreme, ≥ 14); however, severity level can be increased to reflect other symptoms and functional disability. For example, someone binging two or three times a week and experiencing substantial distress could be assigned a severity level of moderate instead of mild.

The text in DSM-5 provides additional guidance that is helpful when assessing eating behaviors.[1] Context is important, for example, a quantity of food that might be regarded as excessive for a typical meal might be considered normal during a celebration or holiday meal, such as Thanksgiving. The "discrete period of time" refers to a limited period, usually less than 2 h but a single episode of binge eating need not be restricted to one setting. The text in DSM-5 gives the example of an individual starting a binge in a restaurant and then this continues upon returning home. However, when evaluating a patient for BED, continual snacking on small amounts of food throughout the day would not be considered an eating binge. The food consumption must be accompanied by a sense of lack of control to be considered an episode of binge eating. This means that the person with BED is unable to refrain from eating or to stop eating once started. It is not unusual for an individual to continue binge eating if the phone rings; however, if a roommate or spouse unexpectedly enters the room, the behavior may immediately cease, and can be followed by significant embarrassment. Patients may also describe the eating as a dissociative experience. Some patients with BED may be unable to clearly articulate an acute feeling of loss of control but instead describe a generalized pattern of uncontrolled eating. The text in DSM-5 adds that if individuals report that they have abandoned efforts to control their eating, loss of control may still be considered as present.

In addition to spontaneous episodes of binge eating when opportunities present themselves, binge eating can also be planned in some instances by the hoarding of foods in secret places. The actual types of foods consumed are not necessarily sugary or salty snacks, or carbohydrates, but can also include fruit, yogurt, or foods ordinarily considered as "healthy." The types of food consumed during binge eating episodes vary both across individuals and for a given individual, and the binge eating episode is characterized more by an abnormality in the amount of food consumed than by a craving for a specific food.

BED prior to DSM-5

BED is not a new entity. Walter Hamburger in 1951 described "a compulsive craving for food . . . This craving is frequently uncontrollable and must be satisfied."[27] Albert Stunkard in 1959 described "enormous amounts of food may be consumed in relatively short periods . . . is regularly followed by severe discomfort and self-condemnation."[28] In 1980, binge behavior was included as a component of the DSM-III diagnostic criteria for bulimia.[29] Research on binge eating in the 1980s was commonly focused on obese persons, and in general, the observed differences between obese binge and obese non-binge eaters included greater levels of psychopathology among the binge-eaters, as well as a greater likelihood to drop out of weight-loss treatment and being more likely to regain lost weight more rapidly.[30] The concept of "purging" and "non-purging" bulimia was further refined, with persons with either disorder exhibiting lower self-esteem and higher anxiety than non-bulimic controls.[31] In 1987, the diagnostic entity of "Bulimia" in DSM-III was replaced by "Bulimia Nervosa" in DSM-III-R, [32] and criteria were made more specific, requiring both binge eating and compensatory behaviors, with a minimum average of two binge eating episodes a week for at least 3 months, and with persistent overconcern with body shape and weight. Consequently, binge eaters without compensatory behaviors could no longer be diagnosed using DSM-III-R. The diagnostic entity of BED was offered as a solution to this problem,[33–35] but ultimately BED was not adopted for the DSM-IV when released in 1994,[36] and the use of the diagnostic entity "Eating Disorder Not Otherwise Specified" (EDNOS) was the option provided to diagnose BED patients. Although specific research criteria for BED were outlined in Appendix B (Criteria Sets and Axes Provided for Further Study) of the DSM-IV, these were not intended for clinical use outside research settings. This situation remained

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unchanged in 2000 with the release of DSM-IV-TR.[37] In clinical settings, EDNOS was frequently diagnosed and became the most commonly documented eating disorder diagnosis.[38] Overreliance on "Not Otherwise Specified" or miscellaneous categories is not desirable in any diagnostic schema, and ultimately BED was recognized as a distinct eating disorder upon the publication of DSM-5 in 2013.[1]

Neurobiology

Essential to the understanding of BED is that there is a loss of control over eating. This is related to obsessive thoughts about food and associated compulsive eating behaviors. When BED is successfully treated, decreases in binge eating frequency are highly correlated with reductions in scores on scales that measure food-related obsessive thoughts and compulsive behaviors.[39] Multiple neurobiological explanations have been suggested for BED, including dysregulation in reward center and impulse control circuitry,[40–42] with potentially related disturbances in dopamine neurotransmission that regulates "wanting food"[43–45] and endogenous μ-opioid signaling that regulates "liking food".[45–47]

Additionally, there is likely an interplay between genetic influences such as functional polymorphisms of the dopamine D2 receptor gene and of the μ-opioid gene[47–49] and environmental stressors.[50,51] Antecedents to binge eating include negative affect; interpersonal stressors; dietary restraint; negative feelings related to body weight, body shape, and food; and boredom.[1]

Recognizing and Diagnosing Binge Eating Disorder

BED can be invisible. Feelings of shame and embarrassment about eating behaviors are very common among persons with BED and thus symptom concealment is often encountered, with families usually unaware of the extent of the binge eating behaviors. Persons with BED may not know that BED is an actual disorder and that there are treatments for it, and thus it may not be spontaneously brought up for discussion. Typically, it is commonly a person's psychiatric and somatic comorbidities that are the focus of the health-care provider's attention, and the BED consequently goes unrecognized. In an Internet survey conducted in 22,397 US adults, 344 participants (1.5%) met the DSM-5 criteria for BED in the past 12 months, but of these 344 respondents with BED, only 11 (3.2%) had ever been diagnosed with BED by a health-care provider.[52]

As noted, a screening instrument is available.[24,25] Called the Binge Eating Disorder Screener-7 (BEDS-7), it was developed based in part on the DSM-5 diagnostic criteria and is intended to be completed by the patient. The first question is "During the last 3 months, did you have any episodes of excessive overeating (i.e., eating significantly more than what most people would eat in a similar period of time)?" If the answer is no, the remaining six questions do not apply. Otherwise, the reminder of the screener is completed, and depending on the answers provided, a more thorough clinical evaluation based upon the complete DSM-5 criteria for BED should be done. A simpler approach would be to ask patients routinely about loss of control over eating.[53] Clinicians already routinely ask about appetite and change in weight when conducting health examinations. It is not unduly burdensome to then ask, "Have you ever eaten more than you intended?" followed by "Did you ever feel like it wasn't easy to stop?" If these questions are endorsed, a more complete evaluation will be required.

The DSM-5 criteria for BED, when discussed with the patient, can be a powerful psychoeducational tool that can help patients feel validated that their symptoms are real and that they have a well-defined disorder. However, attention will need to be paid to the words used to ask about eating as evidenced in a study where language preferences for discussing obesity and binge eating were examined in over 800 people in an online community sample.[54,55] The preferred obesity-related terms were "weight" and "BMI." Binge-related terms were generally ranked neutrally, and preferred descriptions were "kept eating even though not physically hungry" and "loss of control." Specific words to avoid include "fatness" and "excess fat," as well as "large size," "heaviness," and "obesity." Also to be avoided are terms such as "willpower." It was noted that discussions about weight and binge eating are very sensitive and that "conversations about weight and binge eating might create such distress that individuals would prefer not to discuss the topics at all."[54] Gender differences included women's stronger aversions to some terms, possibly related to more frequent experiences of weight stigmatization than men or to greater sensitivity to potentially pejorative terms. The authors concluded that "healthcare providers are encouraged to practice sensitivity and display empathy during discussions of weight and weight loss counseling with female patients in particular."[54] Additional obstacles to a comprehensive evaluation, as identified in a study of physician–patient conversations about BED,[56] included the observations that psychiatrists focused on weight-related issues, whereas patients focused on the emotional impact and triggers of binge eating episodes, and that there is often miscommunication about the severity of binge eating episodes, as well as judgment, bias, and shame surrounding BED.

The text in DSM-5 provides useful information on differential diagnosis.[1] When contrasting BED with bulimia nervosa, the recurrent inappropriate compensatory behavior (e.g., purging, excessive exercise, diuretic abuse, laxative abuse) seen in bulimia nervosa is absent

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in BED, and although persons with BED may report frequent attempts at dieting, they do not display marked or sustained dietary restriction. When contrasting BED with simple obesity, the levels of overvaluation of body weight and shape, and rates of psychiatric comorbidity are higher in obese individuals with BED than in those without BED. Regarding BED and mood disorders, increases in appetite and weight gain are included in the criteria for major depressive episode and in the atypical features specifiers for depressive and bipolar disorders, however, increased eating in the context of a mood episode may or may not be associated with loss of control; if the full criteria for both disorders are met, both diagnoses can be given. When considering BED and borderline personality disorder, binge eating is included in the impulsive behavior criterion that is part of the definition of borderline personality disorder; if the full criteria for both disorders are met, both diagnoses should be given.

Treatments for Binge Eating Disorder

Overview of psychological treatments for BED

Psychological treatments such as CBT and IPT can reduce binge eating behavior.[10-18, 57] In one 16-week study, the effectiveness of group CBT and group IPT for binge eating was assessed in 56 women with "nonpurging bulimia" using a randomized design with a wait-list control.[58] At study end, both group CBT and group IPT treatment conditions showed significant improvement in reducing binge eating, whereas the wait-list condition did not. Moreover, binge eating remained significantly below baseline levels for both treatment conditions at 6-month and 1-year follow-ups. A similar randomized study of group CBT versus group IPT, this time larger (N = 162) but without a control condition, found that both treatments showed initial and long-term efficacy for the core and related symptoms of BED.[59] Unfortunately, access to CBT or IPT may be limited because of local availability and/or cost. CBT and IPT also require at least a moderate amount of insight and motivation on the part of the participant. Moreover, psychological treatment approaches have generally not resulted in weight loss, although successfully eliminating binge eating might protect against future weight gain.[10] Alternatives to CBT and IPT are also necessary when success is not achieved; about 50% of patients with BED do not fully respond to psychological or behavioral treatments.[15]

Overview of pharmacotherapies for BED

Pharmacotherapies for BED have been actively researched.[12,14,16,18,60-63] Medications have included antidepressants (selective serotonin reuptake inhibitors, serotonin–norepinephrine reuptake inhibitors, and bupropion), anticonvulsants (topiramate), anti-obesity/anorectic agents targeting appetite and weight (sibutramine), attention-deficit hyperactivity disorder medications (lisdexamfetamine), and medications for addictive disorders (naltrexone). Until the studies of lisdexamfetamine, the tested medicines fell short in terms of robustness of effect, tolerability, or both. Each alternative appeared to have a significant shortcoming, for example, although antidepressants can reduce the frequency of binge eating behavior, they are not efficacious regarding weight loss, and although topiramate has efficacy regarding reduction of both binge eating and weight, it has a negative impact on cognitive function.

Lisdexamfetamine

At present, lisdexamfetamine is currently the only FDA-approved agent for the treatment of BED. Lisdexamfetamine is a pro-drug for the stimulant medication dextroamphetamine.[3] As per the product label, [64] lisdexamfetamine is indicated for the treatment of moderate to severe BED and has a limitation of use in that lisdexamfetamine is not indicated for weight loss. As with all CNS stimulants, the presence of cardiac disease and risk of abuse must be assessed when prescribing. The recommended starting dose is 30 mg/day to be titrated in increments of 20 mg at approximately weekly intervals to achieve the recommended target dose of 50–70 mg/day. Lisdexamfetamine is taken once daily in the morning, with or without food; afternoon doses are to be avoided because of the potential for insomnia. Approval of lisdexamfetamine for the treatment of BED was based on a clinical development program that included an 11-week Phase II proof-of-concept, placebo-controlled study, testing fixed doses of lisdexamfetamine 30, 50, and 70 mg/day, [65] and two 12-week Phase III placebo-controlled studies examining lisdexamfetamine 50–70 mg/day.[66] Statistically significant reductions in binge eating days/week, the primary outcome measure, were observed for lisdexamfetamine doses of 50–70 mg/day, with large effect sizes.[66] Additionally, large effects were observed on reductions in the Yale-Brown Obsessive-Compulsive Scale Modified for Binge Eating.[66,67] Improvement on efficacy measures was noted as early as 1 week and was maintained throughout the 12-week studies.[68] Across gender and age, participants exhibited comparable clinical responses to lisdexamfetamine compared with placebo.[69] Improvements were also noted regarding functional impairment as measured by the Sheehan Disability Scale.[70]

Number needed to treat (NNT) and number needed to harm (NNH) can be used to quantify the effect sizes for desired and undesired outcomes, respectively.[71-73] Optimal NNT values representing medium to large effect sizes range from 2 to 5, the lower the better, and optimal NNH values are generally ≥ 10, the higher the better.[73]

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Response rates across all trials (as defined by a Clinical Global Impressions-Improvement score of "very much improved" or "much improved") were 86% for lisdexamfetamine versus 48% for placebo, resulting in a NNT of 3.[2] Remission rates (as defined by 4-week cessation of binge eating) were 40% for lisdexamfetamine versus 15% for placebo, resulting in a NNT of 4.[2] Reductions in weight ranged between 5.2% and 6.25% for lisdexamfetamine 50 or 70 mg/day versus no relevant changes observed for placebo. Discontinuation rates because of adverse events (AEs) were low, 4.6% for lisdexamfetamine versus 2.3% for placebo, yielding a NNH of 44.[2] The most common AEs (incidence $\geq$ 10% and $\geq$ 2-times placebo) in the Phase III trials as summarized in the product label[64] were dry mouth (36% and 7%, for lisdexamfetamine and placebo, respectively, NNH 4) and insomnia (20% vs. 8%, NNH 9).[2] A limitation inherent to registration trials in general, and to the above trials as well, is the exclusion of patients with clinically significant psychiatric or somatic comorbidities.

The results of a 39-week long-term maintenance of efficacy study of lisdexamfetamine for BED are available.[74] The primary endpoint was time to relapse of binge eating symptoms in adults with moderate to severe BED. During the 26-week, double-blind, randomized-withdrawal phase of the study, lisdexamfetamine demonstrated superiority over placebo on time to relapse. Observed relapsed rates for lisdexamfetamine versus placebo were 3.7% versus 32.1%, respectively, resulting in a NNT of 4. Additional long-term data are available from a 12-month extension study to the short-term studies.[75] Of the 604 enrolled participants, 369 completed the study. Discontinuation rate because of an AE was 9.0%. Treatment-emergent AEs reported in $\geq$ 10% of participants were dry mouth (27.2%), headache (13.2%), insomnia (12.4%), and upper respiratory tract infection (11.4%). Among the participants in the extension study, the mean reduction in weight observed at week 52 or at early termination was approximately 7 kg.

Dasotraline

In late stage of clinical development for the treatment of moderate to severe BED is dasotraline, a dopamine and norepinephrine reuptake inhibitor. Dasotraline is absorbed slowly and has a long elimination half-life resulting in stable plasma concentrations over 24 h with once-daily dosing.[76] Dasotraline carries a low potential for abuse.[77] Data are available in poster form from two 12-week randomized, double-blind, placebo-controlled studies that have been completed in adults with moderate to severe BED, one using flexible doses of dasotraline 4–8 mg/day,[78,79] and the second using fixed doses of dasotraline 4 and 6 mg/day.[80] The outcome measures used in both studies were similar to what was used in the lisdexamfetamine BED clinical development program. In the flexible-dose study, dasotraline was dosed at 4 mg/day for the first 2 weeks and increased to 6 mg/day at week 2 per investigator's judgment. The dose could be decreased to 4 mg/day (weeks 2–4) per investigator's discretion for reasons of tolerability. By week 4, subjects previously escalated to 6 mg/day remained on 6 mg/day or were escalated up to 8 mg/day. Additional dose changes were allowed following the week 4 visit, but no changes were permitted during weeks 11–12. Statistically significant reductions in binge eating days/week, the primary outcome measure, as well as reductions in the Yale-Brown Obsessive-Compulsive Scale Modified for Binge Eating were observed for dasotraline, with large effect sizes.[78] In the dasotraline group, 46.5% of subjects achieved at least 4 consecutive weeks cessation from binge eating versus 20.6% in the placebo group, yielding a NNT of 4. In addition, significant improvement was observed in measures of weight and shape concern, and intensity of attempts to restrict food intake.[79] AEs that occurred more frequently with dasotraline versus placebo (incidence $\geq$ 10% and $\geq$ 2-times placebo) included insomnia (44.6% vs. 8.1%, NNH 3), dry mouth (27.4% vs. 5.0%, NNH 5), decreased appetite (19.7% vs. 6.9%, NNH 8), anxiety (17.8% vs. 2.5%, NNH 7), and decreased weight (12.1% vs. 0%, NNH 9). Discontinuation due to AEs occurred in 11.3% of subjects with dasotraline versus 2.5% with placebo, yielding a NNH of 12. Mean body weight in the dasotraline group decreased by 4.78 kg (4.9%) from baseline to endpoint, compared to a small increase of 0.40 kg in the placebo group.

In the fixed-dose study, all patients randomized to dasotraline received 4 mg/day during the first 2 weeks, after which those assigned to the higher dose group received 6 mg/day. At week 12, treatment with dasotraline was associated with significant reduction in number of binge eating days per week in the 6 mg/day group versus placebo but non-significant improvement in the 4 mg/day group versus placebo.[80] Outcomes on secondary measures, including the Yale-Brown Obsessive-Compulsive Scale Modified for Binge Eating, generally also favored dasotraline. The proportions of patients who achieved 4-week cessation of binge eating episodes were 34.0%, 33.5%, and 30.2% for the dasotraline 6 mg/day, dasotraline 4 mg/day, and placebo groups, respectively, thus evidencing little or no difference on this outcome. However, when measuring the outcome of $\geq$ 75% reduction of binge eating episodes at endpoint, this was observed in 75.9%, 69.6%, and 56.2% for the dasotraline 6 mg/day, dasotraline 4 mg/day, and placebo groups, respectively, yielding NNT values of 6 for dasotraline 6 mg/day versus placebo and 8 for dasotraline 4 mg/day versus placebo. The most common AEs (incidence $\geq$ 10% and $\geq$ 2-times placebo) on dasotraline 6 mg/day and 4 mg/day versus placebo were insomnia

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(40.1% and 29.8% vs. 13.5%, NNH 4 and 7), dry mouth (26.5% and 21.1% vs. 6.7%, NNH 5 and 7), decreased appetite (16.0% and 9.3% vs. 6.7%, NNH 11 and 39), nausea (13.0% and 11.8% vs. 5.5%, NNH 14 and 16), and anxiety (13.6% and 8.7% vs. 2.5%, NNH 9 and 17). Discontinuation due to an AE occurred in 14.1% of patients on dasotraline 6 mg/day, 8.6% on dasotraline 4 mg/day, and 1.2% on placebo, yielding NNH values of 8 and 14, respectively. Mean percent change in body weight at endpoint was −4.3% and −3.6% for patients randomized to dasotraline 6 and 4 mg/day, respectively, in contrast to an average percent weight gain of 0.4% for patients receiving placebo. The percentage of patients experiencing a $\geq$7% decrease in weight at endpoint was 26.2%, 24.0%, and 2.3% for dasotraline 6, 4 mg/day, and placebo, respectively, yielding NNH values of 5 for either dose of dasotraline versus placebo.

There is an App for that

Smartphone applications (apps) have become popular. However, evidence supporting many of the health apps is lacking and care must be taken when recommending them to patients.[81] Many eating disorder apps are geared toward making it simpler to log binge eating but can also help provide meal reminders, affirmations, and seamless patient–clinician in-app linkage. "Recovery Record" is perhaps one of the better-known eating disorder smartphone apps and is described in the literature by its authors[82,83] and users.[84] Another "Noom Monitor" is intended to facilitate guided self-help CBT.[85] Reviews of smartphone apps for the treatment of eating disorders are available and should be studied before proceeding with their use.[86,87]

Conclusions

BED is different from overeating and requires the presence of distinguishing features, such as loss of control and strong feelings of shame and guilt, which are not normally associated with overeating. Psychiatric and somatic comorbidities are very common, as are functional impairments; however, BED may go undiagnosed for many years because patients seeking treatment for psychiatric or somatic disorders are not always specifically asked about their eating behaviors. BED occurs in both men and women across racial and ethnic groups, and although BED is frequently associated with obesity, about half of adults with BED are of healthy weight or overweight. The precise etiology of BED is not known; however, research suggests an underlying neurobiological basis for BED, with risk factors that include genetic and environmental influences. Effective treatment modalities include psychotherapy (specifically, CBT and IPT) and pharmacologic approaches. A medication approved by the FDA for the treatment of moderate to severe BED is lisdexamfetamine. In late stage of clinical development for the same indication is dasotraline.

Disclosures

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1. The lifetime prevalence of binge eating disorder (BED) in the United States is _____
   A. 0.25%
   B. 0.90%
   C. 0.85%
   D. 0.13%

2. Psychiatric comorbidities are very common, with one study noting that ___% of adults with BED also experience anxiety disorders.
   A. 45
   B. 79
   C. 23

3. The only FDA-approved medication for BED is?
   A. naltrexone
   B. bupropion
   C. lisdexamfetamine
   D. sibutramine

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