BINGE-EATING DISORDER

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DISCLOSURE STATEMENT

Dr. Hilbert received honoraria as a consultant from Popewoodhead, Globaldata, and Weight Watchers, and published books about cognitive-behavioral therapy with Hogrefe.

KEYWORDS

Eating disorder, binge eating, obesity, diagnosis, treatment

SYNOPSIS

Binge-eating disorder (BED), first included as a diagnostic entity in the Diagnostic and Statistical Manual of Mental Disorders Fifth Edition, is characterized by recurrent episodes of binge eating without regular compensatory behaviors to prevent weight gain. With a complex multifactorial etiology, BED is the most frequent eating disorder co-occuring with significant psychopathology, mental and physical comorbidity, obesity, and life impairment. Despite its significance, BED is not sufficiently recognized, diagnosed, or treated. Evidence-based treatments for BED include psychotherapy and structured self-help treatment, with cognitive-behavioral therapy as most well-established approach, and pharmacotherapy with lisdexamfetamine as FDA approved medication with a limitation of use.

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KEY POINTS

- Binge-eating disorder is a clinical eating disorder characterized by recurrent binge eating in the absence of regular compensatory behaviors to prevent weight gain.
- Being the most frequent eating disorder, binge-eating disorder co-occurs with significant psychopathology, mental and physical comorbidity, obesity, and life impairment.
- Despite its significance, binge-eating disorder is not sufficiently being recognized, diagnosed, and treated.
- Evidence-based treatments for binge-eating disorder mainly include psychotherapy and structured self-help treatment, with cognitive-behavioral therapy being the most well-established approach, as well as pharmacotherapy.
- Lisdexamfetamine is FDA approved for binge-eating disorder with a limitation of use.

Binge-eating disorder (BED) was first included as its own diagnostic entity in the Fifth Edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) within the Feeding and Eating Disorders section.[1] BED's hallmark feature is recurrent binge eating, involving the consumption of an amount of food that is definitively larger than what others would eat under comparable circumstances within a certain time, associated with a feeling of loss of control over eating. Diagnosis of BED according to DSM-5 (307.59) requires this objective binge eating to occur at least once per week over 3 months. In contrast to binge eating in bulimia nervosa, binge eating in BED occurs without regular inappropriate compensatory behaviors aimed at preventing weight gain, such as self-induced vomiting, fasting, or laxative misuse. Binge eating in BED is further characterized by behavioral abnormalities, such as eating rapidly or until feeling uncomfortably full, and results in marked distress.

The DSM-5 offers two specifications for the diagnosis of BED: current severity as indicated by the weekly frequency of binge eating; and partial or full remission status after meeting full criteria of BED. In addition, within the Other Specified Feeding or Eating Disorders, DSM-5 newly defines a lower-threshold form of BED (307.59), also associated with significant distress or impairment in life functioning: BED of low frequency and/or limited duration can be diagnosed if objective binge eating is occurring less than once per week and/or for shorter than 3 months, while all other DSM-5 criteria of BED still need to be met.

The International Classification of Diseases and Related Health Problems Tenth Edition (ICD-10)[2] subsumed BED under the Other Eating Disorders (F50.8), without any further specific diagnostic criteria. For the Eleventh Edition of ICD (ICD-11), scheduled for 2019, BED was designated as its own eating disorder diagnosis. Deviating from the DSM-5, the ICD-11 diagnosis of BED was proposed to be broadened and based on both objective or subjective binge eating, termed "loss of control eating." Loss of control eating is characterized as eating an objectively or subjectively large amount of food, while experiencing a sense of loss of control over eating. While the sense of loss of control is a well-validated feature of binge eating, it has been cautioned that the size of a binge provides only incremental information in the explanation of illness severity and psychopathology in diverse eating disorders,[3] so that the clinical significance of this proposed diagnosis of BED may be decreased.

CLINICAL PRESENTATION

Establishing BED as its own diagnostic entity in DSM-5[1] was based on extant literature demonstrating the clinical significance of this disorder. Individuals with BED suffer from increased eating disorder (e.g., weight and shape concern) and general psychopathology (e.g., depressiveness, anxiety), and high comorbidity with mood disorders, anxiety disorders, and substance use disorders, and personality disorders, notably borderline personality disorder, when compared to individuals without BED, and individuals with or without obesity. Additionally, BED frequently co-occurs with attention-deficit/hyperactivity disorder.[4] Thus, BED displays similar psychological impairments as the other well-defined eating disorders, anorexia nervosa and bulimia nervosa.

In addition to psychological comorbidity, BED frequently co-occurs with obesity (body mass index, BMI $\geq$ 30.0 kg/m^2). Because of this comorbidity, individuals with BED have an increased risk of the obesity-related medical sequelae, for example, type 2 diabetes mellitus, hypertension, and dyslipidemia, and of premature mortality.[5,6] Further health conditions increased in individuals with BED include asthma, gastrointestinal symptoms, sleep and pain disorders, neurological problems, and gynecological conditions.[7,8]

The eating behavior in BED is characterized by a general tendency toward overeating. In laboratory test meal studies, individuals with BED overate during both binge-eating and non-binge-eating meals when compared to weight-matched controls.[9] They described more variable meal patterns including more snacking, nibbling, or double meals.[10] When compared to bulimia nervosa, individuals with BED displayed lower dietary restraint and actual restriction,[11] although they exhibited a pronounced body image disturbance.[12] Further, they indicated greater craving, emotional eating, eating for coping purposes,[13] and greater hedonic hunger than controls, and thus experience a greater motivation to eat for pleasure while not in caloric need.[14]

BED results in significant decreases in health-related quality of life and social function impairment, which is similar to the other defined eating disorders.[15,16] Also underlining the severity of this disorder, BED is associated with increased health care utilization and related costs,[15] often years before being diagnosed with BED.[17]

EPIDEMIOLOGY

BED is the most prevalent eating disorder. A representative community-based investigation of 36,306 adults in the US with a primary focus on alcohol use disorder documented prevalence estimates of lifetime BED according to the DSM-5 of 0.9%, and twelve-month estimates of 0.4%.[16] In a further representative community sample of 6,041 Australian adolescents and

adults, the point prevalence of BED (DSM-5) amounted to 5.6%.[18] In addition, BED of low frequency and/or limited duration had a prevalence rate of 6.9%. Other population-based prevalence estimates of BED tend to fall in-between these rates.[19]

BED presents with a distinct sociodemographic profile, when compared to anorexia nervosa and bulimia nervosa, including a more balanced gender ratio, higher rates of obesity, later age of onset, and longer duration of eating disorder symptoms.[19] For example, Udo and Grilo found a 2-3 times increased odds for women to be affected by BED than men, a more than two-fold increased odds for obesity, a mean age of onset of 24.5 years, and a mean duration of eating disorder symptoms of 15.9 years.[16] BED generally occurs among diverse ethnic and/or racial backgrounds.[20] Longitudinal developmental community studies suggest a peak onset of BED in early adolescence in addition to a peak onset in early adulthood.[21,22]

ETIOLOGY

BED develops along a complex etiology involving multiple psychological, biological, and sociocultural influences.[23] Retrospective correlates of risk for BED were mostly shared with the other eating disorders, and included premorbid negative affectivity, perfectionism, conduct problems, substance abuse, childhood obesity, family weight concerns and eating problems, parenting problems and family conflict, parental psychopathology, and physical and sexual abuse.[24]

A few longitudinal studies on the onset of BED indicated that childhood loss of control eating predicts development of BED in adolescence.[25,26] Risk factors longitudinally predicting BED onset in young women were overeating, body dissatisfaction, dieting, negative affect, and mental health impairment.[27] The sociocultural background of an idealization of thinness likely fosters perceived pressure to be thin and internalization of the thin ideal, which have been found to predict the onset of BED in adolescent girls.[28]

Formal genetic studies suggest a heritability of BED of about 41-57%, independent of obesity.[29,30] Molecular genetic research focusing on genes related to reward processing, homeostatic control, and mood processing is limited. Candidate gene studies have indicated the involvement of both dopamine (e.g., DRD2) and μ-opioid (e.g., OPRM1) receptor genes in the etiology of BED. Thus, the risk for binge eating in BED may be conveyed through a hypersensitivity to reward, which is likely to foster binge eating given our current obesogenic environment with high availability of palatable, calorically-dense processed foods. In contrast, research addressing an involvement of the melanocortin 4 receptor gene (MC4R) or of the serotonin transporter gene (5HTT) in BED has yielded inconsistent results.

Overall, in light of the complex etiology of BED, prospective studies testing mediational and interactional models may contribute to understanding how various risk factors work together.

MAINTENANCE

Emotional, social, and cognitive dysfunctions have been assumed to be maintaining factors of BED. According to the prominent affect regulation model,[31] binge-eating episodes are triggered by and serve to provide relief from negative affect. Indeed, descriptive, laboratory, and ecological momentary assessment studies confirmed binge eating in BED to be preceded by negative affect.[32] However, no clear evidence exists that binge eating actually reduces negative affect. Binge-eating episodes are likely to occur against a general background of a reduced emotional awareness and difficulties in emotion regulation, that were significantly more pronounced in BED versus weight-matched controls, and similar, albeit slightly less pronounced than in the other eating disorders.[33,34] Negative affect often arises from interpersonal problems that are reported more frequently by individuals with BED than controls.[35] Interpersonal problems were cross-sectionally found to be associated with eating disorder psychopathology, partially mediated by negative affect, thus lending some support to the interpersonal model of binge eating.[36]

Binge eating is further regarded as resulting from underlying neurocognitive dysfunctions, including difficulties in inhibitory control and reward processing, especially in the processing of disorder-relevant stimuli such as food cues, as determined using behavioral measures.[33,37-39] Neuroimaging studies, mostly based on functional magnetic resonance imaging, documented a related differential brain activation, for example, hypoactivity in prefrontal networks and hyperactivity in the medial orbitofrontal cortex when compared to weight-matched controls.[38,39] Current research on reward-based decision-making showcased an impaired behavioral adaptation, including an increased switching behavior in BED when compared to weight-matched controls. This behavior was accompanied by reduced activation in the anterior insula/ventro-lateral prefrontal cortex, typically associated with reversing behavior, and thus potentially relevant in the maintenance of binge eating despite its adverse consequences.[40] Negative mood seems to exacerbate inhibitory deficits in BED and resulted in less thorough conflict monitoring on the N2 component of EEG event-related potentials and in more inhibition error deficits regarding food cues in an antisaccade task than in weight-matched and normal weight controls,[41] supporting an interaction of emotional and cognitive factors in the maintenance of BED.

Neurocognitive similarities of BED and obesity with substance use disorders resulted in the prominent, yet controversial food addiction hypothesis, according to which certain foods (e.g., high-sugar, high-fat) may elicit addictive responses in vulnerable individuals with high impulsivity and reward sensitivity.[42] Although BED, viewed as an "eating addiction," and substance use disorders may share these vulnerabilities, other important characteristics are distinct: For example, substance use disorders are defined by specific addictive agent, withdrawal, or tolerance, none of which have been demonstrated regarding food.[43] Further critical aspects are that food addiction in humans is mainly operationalized through self-report and has unclear clinical relevance.

Overall, new research on the maintenance of BED should be used to refine clinical maintenance models providing treatment rationales, for example, the cognitive-behavioral model that was developed for bulimia nervosa,[44] without specifically adapting it to BED. Biopsychological aspects of the maintenance of binge eating await further clarification.

PROGNOSIS

Regarding long-term outcome, limited longitudinal evidence suggests that the natural course of BED is more variable than that in anorexia nervosa or bulimia nervosa, with tendencies towards recovery and relapse likely embedded in a chronic course.[45,46] Further, BED seems to be less likely to migrate to another eating disorder diagnosis over time in community samples,[45,47] although in some clinical samples, an increased odds of migration to other eating disorder diagnoses, mostly bulimia nervosa, has been noted.

Little is known about the long-term mental or physical health outcome of BED. Significant negative mental health outcomes of binge-eating behaviors in youth are depressive symptoms and substance use.[48,49] Childhood binge-eating behaviors also predicted excess weight gain in adolescents,[48,50] as did diagnosis of BED in young women.[46] BED or binge-eating behaviors were found to independently increase the risk of the obesity-related sequelae in adolescents and adults, including metabolic symptoms.[7,8,50]

TREATMENT

A range of psychological and pharmacological treatments for patients with BED have been designed and evaluated in randomized-controlled trials (RCTs), with psychotherapy, self-help treatment, pharmacotherapy, behavioral weight loss (BWL) treatment, and combined treatment representing the most frequently evaluated treatment categories.

Psychotherapy

Current meta-analyses of RCTs in patients with BED demonstrated that psychotherapy, specifically the most frequently used approach of cognitive-behavioral therapy (CBT), has significant effects on the reduction of binge-eating episodes and remission from binge eating when compared to inactive control groups, mostly wait-list, at post-treatment.[52-54] In addition, psychotherapy significantly reduced eating disorder psychopathology, whereas effects on depression were inconsistent and weight loss effects were non-significant, indicating a stabilization of body weight. The long-term maintenance of effects was demonstrated in RCTs with CBT and interpersonal psychotherapy up to four years following treatment cessation.[55,56] A superiority of CBT over other conceptually and procedurally distinct psychotherapies that specifically addressed the symptomatology of BED, for example, interpersonal psychotherapy or psychodynamic therapy, was not found.[53,57] Thus, while CBT is the most well-established psychotherapy for BED, other psychotherapies with specific interventions for BED may be as efficacious.

Structured Self-help Treatment

Structured self-help treatment has mostly applied CBT manuals in book, electronic (e.g., video), or Internet-based format, with or without guidance from a mental health professional. Meta-analyses of RCTs showed significant effects of self-help treatment on the reduction of binge-eating episodes and remission when compared to wait-list control conditions.[53,54] As with psychotherapy, eating disorder psychopathology was significantly improved when compared to wait-list control conditions. In contrast, effects on depression were inconsistent and there were no significant effects on body weight. Long-term maintenance of effects in RCTs was found up to 24 months following treatment.[56,58] In comparative RCTs of guided versus unguided self-help treatment, differences in efficacy were not found.[53] Thus, guidance does not seem to be indispensable element for patients with BED, although optimal intensity and types of guidance require further clarification. In comparative RCTs with psychotherapy, self-help treatment led to lower rates of remission from binge eating at follow-up and higher drop-out, suggesting a lower efficacy and acceptance than in the more intense specialist psychotherapeutic treatment. In general, self-help treatment is a viable option helping many patients to fully remit from BED. It was found to be less costly than psychotherapy, however, not necessarily more cost-effective.[59] Further work is needed in this area.

Pharmacotherapy

For pharmacotherapy of BED, the majority of RCTs used second generation antidepressants (e.g., fluoxetine), while a few RCTs used anticonvulsants (e.g., topiramate) and the central nervous system stimulant lisdexamfetamine, the only medication approved by the US Food and Drug Administration in 2015 for the treatment of BED.[52,53,60] Meta-analytically, pharmacotherapy using these agents outperformed pill placebo in most RCTs, showing significant effects on binge-eating episodes and remission. A significant weight loss effect was especially demonstrated for lisdexamfetamine,[52,53] while results on eating disorder psychopathology and depression were inconsistent. Data are lacking on long-term maintenance of effects and long-term administration. The incidence rate of adverse events and the related odds of premature discontinuation were significantly increased,[53] necessitating careful consideration in the pharmacotherapy of BED. Of note, lisdexamfetamine is marketed with a limitation of use, not being suited for weight loss and having a potential of serious cardiovascular adverse events.

Weight Loss Treatment

Behavioral weight loss (BWL) treatment is not a treatment for BED, but represents the standard treatment for obesity, which is according to the 2013 Obesity Guideline generally indicated for individuals with BMI $\geq$ 30.0 kg/m^2 or for individuals with BMI $\geq$ 25 kg/m^2 (or increased waist circumference) and obesity-related comorbidities.[61] In a few RCTs, psychotherapy outperformed BWL treatment in reducing binge-eating episodes and eating disorder psychopathology at post-treatment and led to a significantly higher remission from binge eating at long-term follow-up.[53,62] Results were inconsistent as to whether BWL treatment produced a superior post-treatment weight loss outcome than psychotherapy, while at follow-up no differences were observed. Thus, BWL treatment was found to be less suited for the treatment of binge-eating symptomatology than psychotherapy. Other weight loss treatments that were evaluated in a few RCTs of BED are self-help weight loss treatment and antiobesity medications such as orlistat.[53,60] Preliminary data suggest a decreased efficacy regarding binge-eating outcome in BED.

Combined Treatment

Combinations of CBT, BWL treatment, and/or pharmacological interventions did not show meta-analytical effects on binge-eating episodes and remission in RCTs, but eating disorder psychopathology, depression, and body weight were significantly reduced.[53] In direct comparisons, there were no differential effects of combined treatment versus psychotherapy

on these outcomes, but a higher attrition from treatment was found. An additive effect being absent, combined treatment may be offered when psychotherapy alone is insufficient. However, combined treatment was superior to pharmacotherapy in reducing binge-eating episodes, but not in weight loss.[63] These results suggest that combined treatment may be favored over pharmacotherapy alone.

Conclusions

Psychotherapy, self-help treatment, and pharmacotherapy have empirical support in the treatment of BED. Because of low quality of many RCTs for BED,[52,53] more high quality research on diverse treatment approaches is warranted, with a focus on long-term maintenance and comparative efficacy. Pretreatment predictors informing about which patients with BED show a more favorable treatment response have not consistently been identified.[64] More research is also warranted on treatment-specific moderators, which could elucidate for whom a specific treatment works. The presumably most well-established treatment process factor for BED is rapid response.[65] Rapid response, typically defined as a 65-70% reduction in binge eating over the first four weeks of treatment, is a positive prognostic indicator of sustained remission from binge eating across a range of psychotherapeutic and self-help treatment approaches, highlighting a particular relevance of monitoring a patient's response early in treatment.[66]

FUTURE DIRECTIONS

Despite these clinical advances, BED is still not sufficiently being recognized, diagnosed, and treated.[67] For example, Kessler et al. documented in 24,124 adults from 14 countries that only 38.3% of lifetime cases with BED ever received a specific treatment for their eating disorder.[20] This "treatment gap" may be attributable to multiple system factors (e.g., lack of screening for eating disorders) and patient factors (e.g., lack of information, perceived stigma, shame).[68] Contributing to this gap, BED is publicly perceived as less impairing than the other eating disorders and attributable to a lack of self-discipline, which forms the basis of the stigma associated with BED.[69] Although it remains unclear how these stereotypes affect health care professionals' interventions for patients with BED, on the patients' side, perceived stigma is one major barrier to seek help for an eating disorder such as BED.[70] In addition, a "research-practice gap" was identified, including a discrepancy between evidence-based treatments and actual treatment delivery: For example, eating disorder therapists commonly offer eclectic combinations of interventions, not adhering to evidence-based treatment

protocols.[71] These results highlight a substantial challenge of disseminating and implementing evidence-based treatments for BED into clinical practice.

In order to further increase the efficacy of evidence-based treatments for BED, current research on the nature or maintenance of this disorder warrants translation into new interventions and/or treatments. For example, interventions to improve inhibitory control, especially regarding disorder-relevant stimuli, may have potential, including technology-assisted bias modification training,[72] cue exposure,[73] or non-invasive neuromodulation using EEG neurofeedback.[74]

Regarding the classification of BED, further evidence is needed on its reliability and validity, taking into account the changes to diagnostic criteria in DSM-5 and ICD-11. Difficulties exist in the differentiation of BED from other eating disorders, for example, bulimia nervosa with non-purging compensatory behaviors. More research is warranted on the operationalization of diagnostic criteria, especially binge eating with its components of loss of control and size, the behavioral abnormalities, and severity rating. In addition, overvaluation of shape and weight, characteristic of the majority but not all individuals with BED, has been recommended, for example, as a diagnostic specifier, indicating greater illness severity.[75]

REFERENCES

1. American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
2. World Health Organization. International Classification of Diseases and Related Health Problems Tenth Edition (ICD-10). Geneva: World Health Organization; 1992.
3. Forney KJ, Bodell LP. Incremental validity of the episode size criterion in binge-eating definitions: An examination in women with purging syndromes. Int J Eat Disord 2016; 49:651-62.
4. Ziobrowski H, Brewerton TD, Duncan AE. Associations between ADHD and eating disorders in relation to comorbid psychiatric disorders in a nationally representative sample. Psychiatry Res 2018;260:53-9.
5. Global BMI Mortality Cooperation, Di Angelantonio E, Bhupathiraju S, et al. Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. Lancet 2016;388:776-86.
6. Ma C, Avenell A, Bolland M, et al. Effects of weight loss interventions for adults who are obese on mortality, cardiovascular disease, and cancer: systematic review and meta-analysis. BMJ 2017;359:j4849.

7. Mitchell JE. Medical comorbidity and medical complications associated with binge-eating disorder. Int J Eat Disord 2016;49:319-23.
8. Olguin P, Fuentes M, Gabler G, et al. Medical comorbidity of binge eating disorder. Eat Weight Disord 2017;22:13-26.
9. Heaner MK, Walsh BT. A history of the identification of the characteristic eating disturbances of Bulimia Nervosa, Binge Eating Disorder and Anorexia Nervosa. Appetite 2013 Dec;71:445-8.
10. Masheb RM, Grilo CM, White MA. An examination of eating patterns in community women with bulimia nervosa and binge eating disorder. Int J Eat Disord 2011;44:618-24.
11. Schulte EM, Grilo CM, Gearhardt AN. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. Clin Psychol Rev 2016; 44:125-39.
12. Lewer M, Bauer A, Hartmann AS, et al. Different facets of body image disturbance in binge eating disorder: A review. Nutrients 2017;9:E1294.
13. Leslie M, Turton R, Burgess E, Nazar BP, Treasure J. Testing the addictive appetite model of binge eating: The importance of craving, coping, and reward enhancement. Eur Eat Disord Rev 2018. [Epub ahead of print]
14. Espel-Huynh HM, Muratore AF, Lowe MR. A narrative review of the construct of hedonic hunger and its measurement by the Power of Food Scale. Obes Sci Pract 2018;4:238-49.
15. Agh T, Kovács G, Supina D, et al. A systematic review of the health-related quality of life and economic burdens of anorexia nervosa, bulimia nervosa, and binge eating disorder. Eat Weight Disord 2016;21:353-64.
16. Udo T, Grilo CM. Prevalence and correlates of DSM-5-defined eating disorders in a nationally representative sample of U.S. adults. Biol Psychiatry 2018. [Epub ahead of print]
17. Watson HJ, Jangmo A, Smith T, et al. A register-based case-control study of health care utilization and costs in binge-eating disorder. J Psychosom Res 2018;108:47-53.
18. Hay P, Girosi F, Mond J. Prevalence and sociodemographic correlates of DSM-5 eating disorders in the Australian population. J Eat Disord 2015;3:19.
19. Lindvall Dahlgren C, Wisting L, Rø Ø. Feeding and eating disorders in the DSM-5 era: a systematic review of prevalence rates in non-clinical male and female samples. J Eat Disord 2017;5:56.

20. Kessler RC, Berglund PA, Chiu WT, et al. The prevalence and correlates of binge eating disorder in the World Health Organization World Mental Health Surveys. Biol Psychiatry 2013;73:904-14.
21. Swanson SA, Crow SJ, Le Grange D, et al. Prevalence and correlates of eating disorders in adolescents. Results from the national comorbidity survey replication adolescent supplement. Arch Gen Psychiatry 2011;68:714-23.
22. Stice E, Marti CN, Rohde P. Prevalence, incidence, impairment, and course of the proposed DSM-5 eating disorder diagnoses in an 8-year prospective community study of young women. J Abnorm Psychol 2013;122:445-57.
23. Bakalar JL, Shank LM, Vannucci A, et al. Recent advances in developmental and risk factor research on eating disorders. Curr Psychiatry Rep 2015;17:42.
24. Hilbert A, Pike KM, Goldschmidt AB, et al. Risk factors across the eating disorders. Psychiatry Res 2014;220:500-6.
25. Hilbert A, Brauhardt A. Childhood loss of control eating over five-year follow-up. Int J Eat Disord 2014;47:758-61.
26. Tanofsky-Kraff M, Shomaker LB, Olsen C, et al. A prospective study of pediatric loss of control eating and psychological outcomes. J Abnorm Psychol 2011;120:108-18.
27. Stice E, Gau JM, Rohde P, et al. Risk factors that predict future onset of each DSM-5 eating disorder: Predictive specificity in high-risk adolescent females. J Abnorm Psychol 2017;126:38-51.
28. Stice E, Marti CN, Durant S. Risk factors for onset of eating disorders: evidence of multiple risk pathways from an 8-year prospective study. Behav Res Ther 2011;49:622-7.
29. Mayhew AJ, Pigeyre M, Couturier J, et al. An evolutionary genetic perspective of eating disorders. Neuroendocrinol 2018;106:292-306.
30. Yilmaz Z, Hardaway JA, Bulik CM. Genetics and epigenetics of eating disorders. Adv Genomics Genet 2015;5:131-50.
31. Polivy J, Herman CP. Etiology of binge eating: Psychological mechanisms. In: C. G. Fairburn, G. T. Wilson, editors. Binge Eating: Nature, Assessment, and Treatment. New York, NY: Guilford Press, 1993, pp. 173-205.
32. Nicholls W, Devonport TJ, Blake M. The association between emotions and eating behaviour in an obese population with binge eating disorder. Obes Rev 2016;17:30-42.
33. Kittel R, Brauhardt A, Hilbert A. Cognitive and emotional functioning in binge-eating disorder: A systematic review. Int J Eat Disord 2015;48:535-54.

34. Dingemans A, Danner U, Parks M. Emotion regulation in binge eating disorder: A review. Nutrients 2017;9:E1274.
35. Brugnera A, Lo Coco G, Salerno L, et al. Patients with binge eating disorder and obesity have qualitatively different interpersonal characteristics: Results from an Interpersonal Circumplex study. Compr Psychiatry 2018;85:36-41.
36. Ivanova IV, Tasca GA, Proulx G, et al. Does the interpersonal model apply across eating disorder diagnostic groups? A structural equation modeling approach. Compr Psychiatry 2015;63:80-7.
37. Giel KE, Teufel M, Junne F, et al. Food-related impulsivity in obesity and binge eating disorder-a systematic update of the evidence. Nutrients 2017;9:E1170.
38. Kessler RM, Hutson PH, Herman BK, et al. The neurobiological basis of binge-eating disorder. Neurosci Biobehav Rev 2016;63:223-38.
39. Kober H, Boswell RG. Potential psychological and neural mechanisms in binge eating disorder: Implications for treatment. Clin Psychol Rev 2018;60:32-44.
40. Reiter AM, Heinze HJ, Schlagenhauf F, et al. Impaired flexible reward-based decision-making in binge eating disorder: evidence from computational modeling and functional neuroimaging. Neuropsychopharmacol 2017;42:628-37.
41. Leehr EJ, Schag K, Dresler T, et al. Food specific inhibitory control under negative mood in binge-eating disorder: Evidence from a multimethod approach. Int J Eat Disord 2018;51:112-23.
42. Gearhardt AN, Davis C, Kuschner R, Brownell KD. The addiction potential of hyperpalatable foods. Curr Drug Abuse Rev 2011;4:140-5.
43. Schulte EM, Grilo CM, Gearhardt AN. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. Clin Psychol Rev 2016;44:125-39.
44. Fairburn CG, Cooper Z, Shafran R. Cognitive behaviour therapy for eating disorders: a "transdiagnostic" theory and treatment. Behav Res Ther 2003;41:509-28.
45. Agras WS, Crow S, Mitchell JE, et al. A 4-year prospective study of eating disorder NOS compared with full eating disorder syndromes. Int J Eat Disord 2009;42:565-70.
46. Fairburn CG, Cooper Z, Doll HA, et al. The natural course of bulimia nervosa and binge eating disorder in young women. Arch Gen Psychiatry 2000;57:659-65.
47. Schaumberg K, Jangmo A, Thornton LM, et al. Patterns of diagnostic transition in eating disorders: a longitudinal population study in Sweden. Psychol Med 2018;18:1-9.

48. Field AE, Sonneville KR, Micali N, et al. Prospective association of common eating disorders and adverse outcomes. Pediatrics 2013;130:e289-95.
49. Tanofsky-Kraff M, Shomaker LB, Olsen C, et al. A prospective study of pediatric loss of control eating and psychological outcomes. J Abnorm Psychol 2011;120:108-18.
50. Tanofsky-Kraff M, Yanovski SZ, Schvey NA, et al. A prospective study of loss of control eating for body weight gain in children at high risk for adult obesity. Int J Eat Disord 2009;42:26-30.
51. Mitchell JE. Medical comorbidity and medical complications associated with binge-eating disorder. Int J Eat Disord 2016;49:319-23.
52. Brownley KA, Berkman ND, Peat CM, et al. Binge-eating disorder in adults: A systematic review and meta-analysis. Ann Intern Med 2016;165:409-20.
53. Hilbert A, Petroff D, Herpertz S, et al. Meta-analysis of the effectiveness of psychological and medical treatments for binge-eating disorder (MetaBED). J Consult Clin Psychol. In press.
54. Linardon J, Wade TD, de la Piedad Garcia X, et al. The efficacy of cognitive-behavioral therapy for eating disorders: A systematic review and meta-analysis. J Consult Clin Psychol 2017;85:1080-94.
55. Hilbert A, Bishop ME, Stein RI, et al. Long-term efficacy of psychological treatments for binge eating disorder. Br J Psychiatry 2012;200:232-7.
56. Wilson GT, Wilfley TE, Agras WS, et al. Psychological treatments of binge eating disorder. Arch Gen Psychiatry 2010;67:94-101.
57. Spielmans GI, Benish SG, Marin C, et al. Specificity of psychological treatments for bulimia nervosa and binge eating disorder? A meta-analysis of direct comparisons. Clin Psychol Rev 2013;33:460-9.
58. de Zwaan M, Herpertz S, Zipfel S, et al. Effect of internet-based guided self-help vs individual face-to-face treatment on full or subsyndromal binge eating disorder in overweight or obese patients. JAMA Psychiatry 2017;74:987-95.
59. König HH, Bleibler F, Friederich HC, et al. Economic evaluation of cognitive behavioral therapy and Internet-based guided self-help for binge-eating disorder. Int J Eat Disord 2018;51:155-64.
60. McElroy SL. Pharmacologic treatments for binge-eating disorder. J Clin Psychiatry 2017;1:14-9.

61. Jensen, Ryan DH, Apovian CM, et al. 2013 AHA/ACC/TOS guideline for the management of overweight and obesity in adults. J Am Coll Cardiol 2013;63:2985-3023.2017;9:E299.
62. Palavras MA, Hay P, Filho CA, et al. The efficacy of psychological therapies in reducing weight and binge eating in people with bulimia nervosa and binge eating disorder who are overweight or obese. Nutrients 2017;9:E299.
63. Grilo CM, Reas DL, Mitchell JE. Combining pharmacological and psychological treatments for binge eating disorder: current status, limitations, and future directions. Curr Psychiatry Rep 2017;18:55.
64. Linardon J, de la Piedad Garcia X, Brennan L. Predictors, moderators, and mediators of treatment outcome following manualised cognitive-behavioural therapy for eating disorders: A systematic review. Eur Eat Disord Rev 2017;25:3-12.
65. Brauhardt A, de Zwaan M, Hilbert A. The therapeutic process in psychological treatments for eating disorders: a systematic review. Int J Eat Disord 2014;47:565-84.
66. Grilo CM. Psychological and behavioral treatments for binge-eating disorder. J Clin Psychiatry 2017;1:20-4.
67. Kornstein SG. Epidemiology and recognition of binge-eating disorder in psychiatry and primary care. J Clin Psychiatry 2017;1:3-8.
68. Kazdin AE, Fitzsimmons-Craft EE, Wilfley DE. Addressing critical gaps in the treatment of eating disorders. Int J Eat Disord 2017;50:170-89.
69. Puhl R, Suh Y. Stigma and eating and weight disorders. Curr Psychiatry Rep 2015;17:552.
70. Ali K, Farrer L, Fassnacht DB, et al. Perceived barriers and facilitators towards help-seeking for eating disorders: A systematic review. Int J Eat Disord 2017;50:9-21.
71. Waller G. Treatment protocols for eating disorders: clinicians' attitudes, concerns, adherence and difficulties delivering evidence-based psychological interventions. Curr Psychiatry Rep 2016; 18:1-8.
72. Schmitz F, Svaldi J. Effects of bias modification training in binge eating disorder. Behav Ther 2017;48:707-17.
73. Ferrer-Garcia M, Gutierrez-Maldonado J, Pla-Sanjuanelo J, et al. A randomised controlled comparison of second-level treatment approaches for treatment-resistant adults with bulimia nervosa and binge eating disorder: Assessing the benefits of virtual reality cue exposure therapy. Eur Eat Disord Rev 2017;25:479-90.

74. Blume, M., Schmidt, R. & Hilbert, A. Abnormalities in the EEG power spectrum in bulimia nervosa, binge-eating disorder, and obesity: A systematic review. In revision.
75. Grilo CM. Why no cognitive body image feature such as overvaluation of shape/weight in the binge eating disorder diagnosis? Int J Eat Disord 2013;46:208-11.