IJNRD.ORG ISSN : 2456-4184
INTERNATIONAL JOURNAL OF NOVEL RESEARCH AND DEVELOPMENT (IJNRD) | IJNRD.ORG
An International Open Access, Peer-reviewed, Refereed Journal

# Binge Eating Disorder: A Review

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**Abstract:** Binge eating disorder (BED) is a state of mental disease which has symptoms of recurrent binge eating episodes in the absence of compensatory behaviors. Therefore, BED is strongly associated with obesity. This review is to provide an overview of the most important aspects of BED (e.g., clinical profile, and treatment approaches), to not only facilitate a better understanding of the disorder and its clinical consequences, but also to identify potential methods of prevention and intervention. Many risk factors have been associated with the disorder. Although some treatments for BED have proven to be effective in addressing different key aspects of the disorder, the rates of patients that have ever received specific treatment for BED are very low. The factors involved and how to implement effective treatments will be discussed for the purpose of addressing the eating symptomatology and comorbidities.

**Introduction:** Binge eating disorder (BED) is a type of eating disorder (ED) recently included in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5).[1] BED, like bulimia nervosa (BN), is characterised by recurrent binge eating episodes (i.e., episodes of overeating with loss of control), at least once a week for the preceding 3 months.[2] However, in contrast to BN, there is an absence of inappropriate compensatory behaviours (such as purging, fasting or excessive exercise). Due to the caloric overconsumption involved, BED is strongly associated with obesity.[3] BED is much more severe than overeating since it is associated with more distress regarding the eating behaviour. It frequently impacts quality of life, is associated with psychopathological features (mainly stress, negative affect, depression, or anxiety) and gives an increased risk of comorbid medical conditions (e.g., metabolic syndrome: mainly type 2 diabetes, hypercholesterolemia and hypertension).[4,5]

**Clinical and psychopathological features associated with BED:** A wide range of psychiatric disorders are present in most patients suffering from BED.[6] According to Grilo,[7] 67.0% of BED patients had at least one additional lifetime psychiatric disorder, and 37.0% had at least one current psychiatric disorder, mood and anxiety disorders being the most frequent comorbidities. Approximately, between 30.0 and 80.0% of individuals with BED present lifetime comorbid mood and anxiety disorders,[8,9] but also some related pathologies such as bipolar disorder.[10] Furthermore, evidence points to impulse control impairments in patients with BED.[11] Therefore, other common comorbidities reported in individuals with BED comprise numerous addiction disorders such as substance use/abuse (22.0%),[9] gambling problems (5.7–18.7%)[12] as well as compulsive buying (7.4–18.5%).[13] Additionally, comorbid personality disorders have been also identified in BED patients, especially avoidant, obsessive-compulsive, and borderline personality disorders.[14] Moreover, some patients with BED also meet criteria for comorbid attention deficit and hyperactivity disorders (ADHD).[10]

**Specific risk and maintenance factors for BED:** From a holistic perspective, a wide range of factors must be considered to understand the complexity of BED, such as environmental (socio-cultural and family patterns) and individual factors (biological, genetic, and psychological), as well as the interactions between these.[15]

**Individual risk factors:**

*Psychological and personality risk factors:* There is broad evidence indicating that some specific psychological features, such as negative self-evaluation, low self-esteem, and high body dissatisfaction, might aid the development of BED.[16] Likewise, low self-esteem interacts with other factors to predict BED.[2] Striegel- Moore et al.[17] reported that both negative affect and perfectionism increase the risk of BED. All these findings suggest a complex aetiology of BED and reinforce the idea that the combination of multiple psychological factors, not just the presence of one, influences the onset of the disorder. A large body of research suggests a link between emotion regulation and BED.[18] This relationship is bidirectional, and it can

sometimes be maladaptive, that is to say: emotional dysregulation precedes the appearance of binge-eating episodes and overeating, in turn, exacerbating the guilt and embarrassment from losing control and overeating, generating a vicious circle.[19] On the other hand, BED has frequently been associated with specific personality traits such as high levels of impulsiveness, sensation seeking and lack of persistence.[2,20]

*Biological-genetic risk factors:* Despite the high prevalence of BED, its aetiology remains understudied, and little is known about the biological – genetic features underlying this disorder.[21] The fact that obesity exists in the absence of BED and vice versa reveals that the genetic and environmental factors which contribute to both pathologies are not the same ones.[2] Notably, some studies have assessed potential biological substrates which might contribute to the pathogenesis of the disorder (mainly genetic factors). Recent genetic research (based on family, twin, and molecular studies) suggests that familiar and genetic factors may act as risk factors for BED.[22] BED is moderately heritable, with heritability estimated to range from 41.0 to 57.0%.[23] However, the genetics of BED are complex and imply interactions with environmental factors. Genetic association studies in BED have mostly investigated genes implicated in dopamine, serotonin, and appetite systems. Genetic underpinnings of BED include alterations in the dopaminergic (related to reward) and opioidergic (responsible for the food-related hedonic response) systems, which contribute to impulsivity-compulsivity and reward-related processes.[23] Although other neurotransmitter systems (e.g., serotoninergic, noradrenergic, or glutamatergic) have been associated with the neuropathophysiology of BED, dopaminergic genes seem to be the most important contributors to BED due to their relationship with the reward system.[2,23]

*Neuropsychological and brain activity risk factors:* Until very recently, little was known about the neuropsychological mechanisms which could be involved in the development and maintenance of BED. Nevertheless, growing evidence suggests that cognitive processes may underlie both eating behaviours and BED, behaving as potential risk factors; however, these processes are still barely understood.[24] Included within the mentioned neuropsychological mechanisms one finds the executive function (EF), which involves higher-level cognitive processes implicated in the formation of successful goal-directed behaviour.[25] Previous research suggests that eating disorders are associated with impairments in EF,[26,27] although studies examining EF deficits in BED are currently limited.[28] At present, the EF problems observed in subjects with BED consist of deficits in set-shifting,[29] inhibitory control[28], decision-making,[30] problem solving,[28] as well as working

memory.[31] In addition, delay discounting impairments (i.e., the extent to which the perceived value of a reward decreases as the reward is delayed) were also observed in individuals with BED, who displayed higher prioritization of immediate versus delayed rewards in comparison with subjects without BED.[28] In this line, individuals with BED reported altered reward/ punishment sensitivity.[2,32]

*Behavioural risk factors:* Dieting is the most described behavioural risk factor for BED onset. It is well-documented that dieting increases the risk of overeating to counteract the caloric deprivation and, therefore, weight gain over time. Furthermore, some of the above-described psychological risk factors such as body dissatisfaction, perceived pressure to be thin, emotional eating and negative affect interact and mutually reinforce each other, promoting dieting and, therefore, increasing the risk for BED.[33] Similarly, a sedentary lifestyle and the preference for high-calorie foods are critical during BED patients with obesity, so they are also important maintenance factors to consider.[34] Likewise, it is worth noting that binge eating also predicts obesity onset.

*Environmental risk factors:* It has been widely described that some specific socio-environmental conditions may act as shared risk factors for BED and obesity. Of the most frequent vulnerability factors, the following are highlighted: weight teasing by family and peers, perceived weight-related social pressure, frequent negative comments, or bullying.[35] Furthermore, images on television or social media promoting society's focus on thinness and ideals of beauty contribute to body dissatisfaction, which, in turn, may also act as a risk factor for eating disorders, including BED.[36] Similarly, Fairburn et al.[37] found that parental criticism about shape, weight and eating, as well as high expectations of the family, were also relevant risk factors for developing BED. Furthermore, traumatic life events or life stressors (such as death or separation from a friend or family member, the end of a relationship, etc.) and adverse childhood experiences (sexual and physical abuse and parental problems) also increase the risk of BED.[37] In addition, stress of pregnancy and overvaluation of pregnancy- related weight gain have also been related to BED.[38]

**Treatment approaches for BED:**

BED treatment rates seem to be low; in fact, only 38.0% of patients with a lifetime diagnosis of BED have ever received specific treatment for an eating disorder.[39]

*Psychological and behavioural first-line treatments for BED:* Leading psychological and behavioural treatments include cognitive behavioural therapy (CBT), interpersonal psychotherapy (IPT), dialectical behaviour therapy (DBT) and behavioural weight loss treatment (BWL).[2,40]

CBT directly targets the normalization of eating patterns and promotes behavioural and cognitive changes to prepare the patient for coping with stressful situations, which reduces binge eating episodes. As primary outcomes, the CBT model presents high long-term effectiveness (i.e., long-lasting absence of binge-eating symptomatology), but also great improvements in comorbid psychopathology at 48-month follow-up.[41] The IPT model, which focuses on the interpersonal difficulties that develop and maintain the disorder, has shown similar marked long-term effectiveness to CBT.[41] In this regard, recent studies report recovery rates of 50.0–65.0% (absence of binge eating), which are maintained at follow-up.[42] Although both CBT and IPT have a sustained effect on binge eating and slowing down the course of weight gain, they fail to produce weight loss in patients with BED, which often present obesity.[43] In this regard, the study by Agüera et al.[44] suggests that some typical features of BED, such as dissatisfaction with body shape and the urge to lose weight, may be responsible for the high dropout rates of these therapies. To address this weakness, some authors have carried out research using a behavioural therapy based on caloric reduction and promotion of physical activity (namely BWL). BWL is widely used to treat obesity and appears to be useful for weight loss; however, it is less effective than CBT or IPT in maintaining binge eating reduction at follow-up.[42]

*Pharmacological first-line treatments for BED:* Pharmacotherapy is not the primary treatment for BED, but it is often indicated as a supplement in therapeutic interventions, mainly to address depressive symptoms and weight management. Some antidepressants have been shown to be effective in reducing binge eating frequency, but they do not have the desired effect on weight loss.[45] On the other hand, weight management medication, such as Orlistat (a lipase inhibitor used to treat obesity), has been shown to be useful for treating weight loss in non-BED individuals with obesity, but it reveals ineffectiveness among those with BED.[46]

Currently, the first-choice drug used in BED is topiramate, an anti-epileptic medication. Randomized placebo-controlled trials have demonstrated that CBT plus topiramate improved the efficacy of the psychological therapy in severe cases, reducing both binge-eating frequency and weight.[47] However, topiramate should occasionally be withdrawn due to its adverse effects, such as headache, paresthesias, amenorrhea or sedation.[47]

**Conclusions:** This review presents a brief overview of BED, addressing its relevance, clinical characteristics, treatment options and treatment outcomes. As mentioned throughout this review, BED is an eating disorder which presents high comorbidity with other medical and psychiatric disorders. A lot of risk factors have been associated with the development and maintenance of the disorder, from individual to environmental risk factors. Although some treatments for BED have proven to be effective in addressing different key aspects of the disorder, not many patients have received specific treatment for this disorder. Conversely, BED patients usually have a strong urge to lose weight, which interferes with psychological treatments and increases the risk of dropout. It is important to consider the clinical implications and it is necessary to determine which therapeutic tools might be effective for improving the treatment of patients with BED. For this, further studies are needed to assess the extent to which patients with BED can benefit from other therapeutic approaches.

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