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A review of binge eating disorder and obesity

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Summary Binge eating disorder (BED) is a mental illness characterised by recurrent binge eating episodes in the absence of appropriate compensatory behaviours. Consequently, BED is strongly associated with obesity. The current review aims to provide an update of the most relevant aspects of BED (e.g., clinical profile, aetiology and treatment approaches), in order not only to facilitate a better understanding of the disorder and its clinical consequences, but also to identify potential targets of prevention and intervention. Patients with BED often present high comorbidity with other medical conditions and psychiatric disorders. Numerous risk factors have been associated with the development and maintenance of the disorder. Moreover, 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 comorbid obesity.

Keywords Binge eating disorder · Obesity · Risk factors · Treatment · Prevalence

Übersicht zu Binge-eating und Adipositas

Zusammenfassung Die Binge-eating-Störung (BES) ist eine psychische Erkrankung, die durch wiederkehrende Essanfälle und das Fehlen eines angemessenen Kompensationsverhaltens gekennzeichnet ist. In der Folge besteht eine starke Assoziation mit Adipositas. Der vorliegende Beitrag bietet eine aktuelle Übersicht zu den wichtigsten Gesichtspunkten von BES, unter anderem zu klinischem Bild, Ätiologie und Behandlungsansätzen. Ziel ist nicht nur ein besseres Verstehen der Erkrankung und ihrer klinischen Folgen, sondern auch die Identifikation potenziel-

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ler Ansatzpunkte für Prävention und Intervention. Patienten mit BES zeigen oft eine hochgradige Komorbidität mit anderen somatischen und psychischen Erkrankungen. Für zahlreiche Risikofaktoren wurde ein Zusammenhang mit der Entwicklung und Erhaltung der Störung ermittelt. Obwohl sich einige therapeutische Ansätze in Bezug auf verschiedene Kernaspekte der BES als wirksam erwiesen haben, hat nur ein sehr geringer Anteil der Patienten je eine spezifische Behandlung erhalten. Es werden Einflussfaktoren erörtert und die Frage diskutiert, wie sich wirksame Therapien umsetzen lassen, um die Esssymptomatik und komorbide Adipositas zu beeinflussen.

Schlüsselwörter Binge-eating-Störung · Adipositas · Risikofaktoren · Behandlung · Prävalenz

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]. According to DSM-5 criteria, 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. 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 [2–4]. BED is much more severe than overeating, since it is associated with more subjective distress regarding the eating behaviour. BED frequently impacts quality of life, is associated with psychopathological features (mainly stress, negative affect, depression or anxiety) and confers an increased risk of comorbid medical conditions (e.g. metabolic syndrome: mainly type 2 diabetes, hypercholesterolemia and hypertension) [5–7].

Epidemiology of binge eating disorder

The prevalence of BED varies depending on the diagnostic criteria used for its assessment and the type of sample evaluated. Two large-scale studies have assessed BED prevalence in community samples using a text revision of the DSM-IVE (DSM-IV-TR) criteria [8, 9]. Whereas the US National Comorbidity Survey Replication study reported a BED lifetime prevalence of 2.8% [8], an average lifetime prevalence of 1.9% was obtained when a total of 24,124 adult respondents from 14 different countries on three continents (America, Europe and Oceania) were evaluated by the WHO World Mental Health Survey [9]. Subsequently, the US National Health and Wellness Survey assessed another community sample but taking into account the DSM-5 criteria, observing a similar BED lifetime prevalence of 2.0% [10]. On the other hand, when the lifetime occurrence of BED is examined in samples comprising individuals with overweight or obesity, the estimated rates obtained are notably higher. In the last above-mentioned study, the lifetime prevalence of BED varied depending on body mass index (BMI) of the participants: 1.2% in overweight subjects, as well as 2.6% and 4.5% in obese subjects with a BMI = 30–35 and BMI ≥35, respectively [10]. Similarly, a recent study which evaluated prevalence rates of mental disorders in individuals with obesity showed BED prevalence rates between 3.3 and 5.5% [11]. These results suggest a direct correlation between BED prevalence and BMI, with BED lifetime prevalence becoming higher while BMI increases.

Given that BED is strongly associated with BMI, some studies have calculated the percentage of overweight and obesity in people who suffer from BED. The WHO World Mental Health Survey reported that 30.7% of individuals with lifetime BED had overweight and 32.8% suffered from obesity [9]. Nevertheless, the percentage of lifetime obesity in BED substantially increased to 87.8% in another investigation where the sample was composed of 1383 ED female patients diagnosed by experienced psychologists according to DSM-IV-TR criteria [3]. According to the authors, the prevalence of obesity within BED might be biased by the way BED was diagnosed. Therefore, a formal diagnosis of the patient's pathology is highly important when examining the relation between BED and obesity. This diagnosis should be made by a specialist psychologist/psychiatrist following the diagnosis criteria described in the DSM-5 for BED.

Gender differences have also been found in BED lifetime prevalence, which appears to be higher in women (2.1–3.5%) than in men (0.9–2.0%) [8, 10]. Personal and family history of obesity also play an important role in the development of BED; for instance, the previously mentioned study exploring obesity in ED patients, found that 62.8% of patients with BED had a family history of obesity and almost 29.0% suffered from childhood obesity [3]. Furthermore, a family history of BED has been considered a risk factor for suffering obesity in adulthood [12].

Clinical and psychopathological features associated with BED

A wide range of psychiatric disorders are present in most patients suffering from BED [13]. According to Grilo [14], 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 [15–20], but also some related pathologies such as bipolar disorder [10]. Furthermore, evidence points to impulse control impairments in patients with BED [21]. Therefore, other common comorbidities reported in individuals with

BED comprise numerous addiction disorders such as substance use/abuse (22.0%) [16], gambling problems (5.7–18.7%) [22, 23] as well as compulsive buying (7.4–18.5%) [24, 25]. Likewise, food addiction (FA) is a concept of growing interest which has recently been closely related to BED [26]. FA is a term employed to describe addictive-like compulsive overeating which involves cravings and difficulties in abstaining from high-calorie foods [27]. Even though FA may appear to be similar to BED, it is important to note that while BED is a classified and diagnosable mental disorder, FA remains a controversial concept which has not yet been recognized as a diagnostic entity. In addition, FA has been linked to a greater extent to addictive disorders than to ED due to neurochemistry similarities [28, 29], specifically in terms of the dopaminergic reward system. Previous studies found that around 50.0% of obese adults diagnosed with BED also met criteria for FA [30, 31] assessed with the Yale Food addiction Scale (YFAS) [32]. Similarly, recent studies showed a high percentage (i.e. ranging from 76.9 to 87.2%) of FA in patients with BED who were screened with the YFAS [33–36]. These findings suggest that some cases of compulsive overeating, an essential feature of BED, share pronounced similarities with conventional addictions such as substance abuse [37]. Additionally, comorbid personality disorders have been also identified in BED patients, especially avoidant, obsessive-compulsive and borderline personality disorders [38]. Moreover, some patients with BED also meet criteria for comorbid attention deficit and hyperactivity disorders (ADHD) [10].

Focusing on obesity, previous studies reported that individuals with obesity and BED presented significantly higher levels of general psychopathology in comparison with those without BED [39–41]. In relation to the onset of the disease, individuals that developed BED during their childhood presented greater comorbidity rates than those with an onset in adulthood [42]. More severe psychopathology and poorer daily functioning also correlate with childhood onset, although no associations were found between age of onset and higher frequency of binge eating or higher BMI [18].

Additionally, most BED patients present impairments in their psychological well-being, including aspects such as autonomy, environmental mastery, self-acceptance [43] as well as emotional regulation [44]. Another frequent characteristic of BED is the overvaluation of body shape and weight, which is strongly related to a greater severity of eating-related psychopathology, higher psychological distress and a poorer prognosis of the disease [14]. Psychological impairments could also have direct consequences on suicidal behaviour, especially on those BED individuals with depressive symptoms [45, 46].

Specific risk and maintenance factors for BED

From a holistic perspective, a wide range of factors must be considered in order 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 [47, 48] (see Fig. 1).

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 [49, 50]. 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 [50].

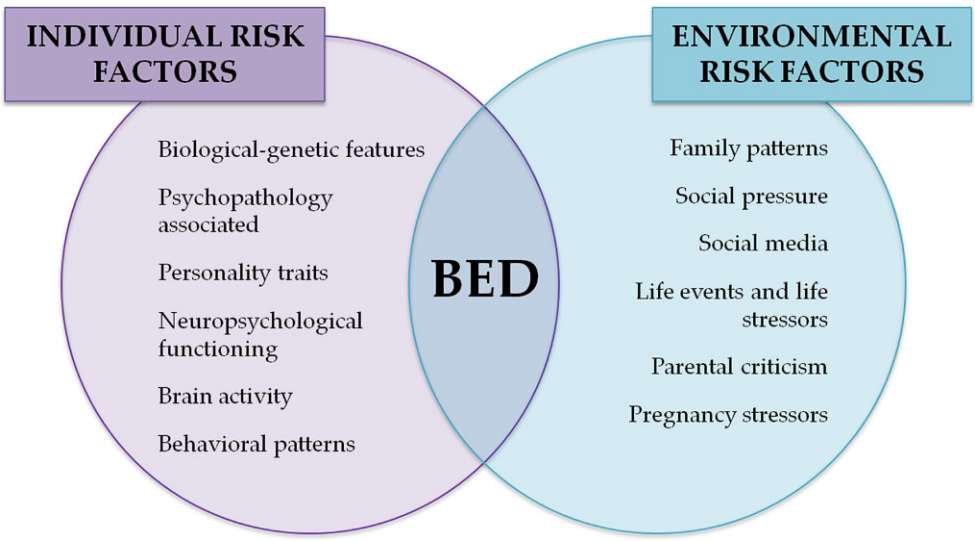

Fig. 1 Individual and environmental risk factors of binge eating disorder (*BED*)

Several family risk factors, such as family overeating, having a parent with a mood, anxiety or substance use disorder, family discord, high parental demands or parental perfectionism as well as parental separation, have been identified as possible predictors involved in the onset of BED [51]. Similarly, Fairburn et al. [52] 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 [52–54]. In addition, stress of pregnancy and overvaluation of pregnancy-related weight gain have also been related to BED [55].

Individual risk factors

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 [56]. 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 exactly the same ones [57]. 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 [58]. BED is moderately heritable, with heritability estimated to range from 41.0 to 57.0% [59, 60]. 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 [60]. 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 [60, 61]. Also, the potential role of other additional candidate genes for BED has been explored. However, research is sparse, with contradictory findings and a lack of replications. In this regard, while some studies have found that different variants of genes such as MC4R (*melanocortin 4 receptor*) [62], BDNF (*brain-derived neurotrophic factor*) [63] or FTO (*Fat Mass and Obesity related gene*) [64, 65] may be candidate genes for BED, others failed to find these associations [66, 67]. The lack of genome-wide association studies (GWAS) or epigenetic research in BED hinder advances towards a better understanding of its aetiology and, therefore, its prevention and treatment [68].

Psychological and personality risk factors

There is broad empirical evidence indicating that some specific psychological features, such as low self-esteem, negative self-evaluation and high body dissatisfaction, might contribute to the development of BED [52, 69]. Likewise, low self-esteem interacts with other factors to predict BED [70]. Whereas Striegel-Moore et al. [51] reported that both negative affect and perfectionism increase the risk of BED, Sehm and Warschburger [70] found that the lack of interoceptive awareness and body dissatisfaction were the most robust predictors 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 [71]. This relationship is bidirectional and it can sometimes be maladaptive, that is to say: when negative affect and emotional dysregulation precede the appearance of binge-eating episodes and overeating, in turn, exacerbating the guilt and embarrassment from losing control over eating, generating a vicious circle [72].

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 [73]. Likewise, it has also been described that BED patients with obesity report higher harm avoidance (highly correlated with neuroticism) and lower scores in self-directedness and persistence than non-BED individuals with obesity [41].

Both high neuroticism and high impulsivity were significantly associated with a higher lifetime prevalence of BED [74]. This combination of high impulsivity and neuroticism may be conceptualized as *negative urgency*—the tendency to engage in rash actions and risky behaviours (i.e. impulsivity) when experiencing strong negative emotions (i.e. neuroticism) [75]. High scores on negative urgency have been associated with vulnerability to stress, reduced capacity to problem solving and poor coping strategies such as escape-avoidance coping [74]. Similarly, the study of Wolz et al. [76] suggests that low self-directedness and emotion dysregulation predict negative urgency in BED patients and are highly associated with binge-eating symptomatology.

However, in a study assessing individuals with obesity seeking weight loss treatment, the authors found that those with and without BED scored similarly on negative urgency, suggesting that negative urgency may also be elevated in obese non-BED populations [77]. These discrepancies indicate that research on

BED is limited and findings have been inconsistent. These inconsistencies open a new line of debate, which implies the need for further studies comparing personality traits among BED individuals with and without obesity, and patients with obesity but without BED.

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 [78]. 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 behaviours [79]. Previous research suggests that eating disorders are associated with impairments in EF [80, 81], although studies examining EF deficits in BED are currently limited [82]. At present, the EF problems observed in subjects with BED consist of deficits in set-shifting [83–86], inhibitory control [82, 86–88], decision-making [83, 89], problem solving [82, 84, 86], attention [87] as well as working memory [84]. 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 [82]. In this line, individuals with BED reported altered reward/punishment sensitivity [90]. Furthermore, set-shifting impairments seem to decrease the ability to handle negative affect and increase loss of control over eating in individuals with BED [91].

The evidence from neuroimaging studies reports structural abnormalities and neural vulnerability factors related to obesity and BED. Regarding obesity, a review of Val-Laillet et al. [92] elucidated a decrease in the basal metabolism of the prefrontal cortex and striatum as well as dopaminergic alterations in individuals with obesity; at the same time, they presented increased activation of reward brain areas in response to palatable food cues, which could trigger food craving and weight gain. On the other hand, according to another review by Kessler et al. [9], BED may be related to altered reward sensitivity and food-related attentional biases. These authors suggest that individuals with BED present corticostriatal circuitry alterations similar to those observed in individuals with substance abuse disorders; these abnormalities include altered function of prefrontal, insular and orbitofrontal cortices and the striatum.

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 [93]. Similarly, a sedentary lifestyle and the preference for high-calorie foods are critical in the course of BED patients with obesity, so they are also important maintenance factors to consider [94, 95]. Likewise, it is worth noting that binge eating also predicts obesity onset [93].

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 [9]. However, the current literature provides optimal data on the most effective treatments for BED. Rigorous studies have highlighted the need to address the following key aspects in order to guarantee the effectiveness of the treatment: eating symptomatology (reducing binge eating), associated psychopathology (especially depression and anxiety), emotional problems, psychosocial functioning (identifying and managing stressful situations such as traumatic events, improving interpersonal skills and relationships and enhancing coping strategies and quality of life), as well as excess weight and metabolic problems ([96]; see Table 1).

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) [97, 98].

The *National Institute of Clinical Excellence* (NICE) guideline [99] identifies CBT as the first choice treatment for BED. CBT directly targets the normalization of eating patterns and promotes behavioural and cognitive changes in order 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 [100]. The IPT model, which focuses on the interpersonal difficulties that develop and maintain the disorder, has shown similar marked long-term effectiveness to CBT [100]. In this regard, recent studies report recovery rates of 50.0–65.0% (ab-

Table 1 Treatment approaches for binge eating disorder

Treatment approaches	Treatment targets						Limitations and strengths based on evidence
	Binge eating reduction	Comorbid psychiatric pathology	Weight and metabolic problems	Quality of life	Emotion regulation	Psychosocial functioning	
Psychological treatments							
CBT[1,2,3,4]	++	++	–	+	+	++	Empirical evidence; long-term effectiveness
IPT[2,3,4]	++	++	–	+	+	++	Empirical evidence; long-term effectiveness
DBT[3,4]	+	+	–	+	++	+	Further research is needed
Behavioural treatment							
BWL[4]	+	–	++	++	–	–	Less effectiveness in reducing binge eating
Pharmacotherapy							
SSRIs[5]	+	++	–	+	+	–	Unwanted effects on weight
Orlistat[5, 6]	–	–	++	–	–	–	Ineffective in reducing binge eating frequency
Topiramate[5,7,8]	+	+	+	+	–	–	Adjuvant treatment; adverse side effects
LDX[5,9,10]	+	+	+	+	–	–	HR and BP should be closely monitored
Other adjuvant treatments							
Exercise[11,12]	+	+	+	++	–	+	Further research is needed
Mindfulness[4]	+	+	–	++	++	+	Further research is needed

– = No effect, + = discrete or variable effect, ++ = relevant effect
CBT cognitive behavioural therapy, *IPT* interpersonal therapy, *DBT* dialectical behaviour therapy, *BWL* behavioral weight loss treatment, *SSRIs* selective serotonin reuptake inhibitors, *HR* heart rate, *BP* blood pressure, *LDX* Lisdexamfetamine
Sources: [1]National Institute for Clinical Excellence (NICE), 2004 [99]; [2]Hilbert, Bishop, Stein, Tanofsky-Kraff, Swenson, Welch, Wilfley, 2012 [100]; [3]Wilson, Wilfley, Agras, Bryson, 2010 [101]; [4]Iacovino, Gredysa, Altman, Wilfley, 2012 [102]; [5]Davis Attia, 2017 [109]; [6]Grilo &White, 2013 [110]; [7]McElroy, Shapira, Arnold, Keck, Rosenthal, Wu, Capece, Fazzio, Hudson, 2004 [111]; [8]Claudino, de Oliveira, Appolinario, Cordás, Duchesne, Sichieri, Bacaltchuk, 2007 [112]; [9]McElroy, Mitchell, Wilfley, Gasior, Ferreira-Cornwell, McKay, Wang, Whitaker, Hudson, 2016 [114]; [10]Bello and Yeomans 2017 [115]; [11]Vancampfort, De Herdt, Vanderlinden, Lannoo, Soundy, Pieters, Adriaens, De Hert, Probst, 2014 [107]; [12]Mathisen, Rosenvinge, Friborg, Vrabel, Bratland-Sanda, Pettersen, & Sundgot-Borgen, 2020 [106]

sence of binge eating), which are maintained at follow-up [101]. To a lesser extent, DBT, which is mainly focused in emotion regulation, distress tolerance and interpersonal effectiveness, has also shown promising results in reducing binge eating and associated psychopathology [102]. However, further research is needed to elucidate its long-term effectiveness (by means of 24- to 48-month follow-up). 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 [96]. In this regard, the study by Agüera et al. [103] 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 [101].

Furthermore, adjuvant treatment to usual therapy, such as mindfulness, has shown preliminary usefulness and effectiveness for improving overall health, well-being and sustainability of outcomes [102, 104]. In other words, both mindfulness and emotion regulation strategies might be effective for addressing negative urgency (a key feature involved in the development of BED) [105].

On the other hand, some studies cover the benefits of adding exercise to CBT for BED, showing both a reduction in binge-eating symptomatology and an improvement in comorbid depression and anxiety [95, 106]. BED patients often report physical complications due to comorbid obesity and sedentary lifestyle, the majority of them being extremely inactive [94, 107]. Recent findings indicate that BED patients, when compared to obese non-BED patients and normal weight controls, display the greatest limitations in physical activity and self-perception [108]. In addition, the positive effect of enhancing physical activity/exercise in these patients has been shown to be associated with improved quality of life and physical self-perception [107].

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 [109]. 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 [110].

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 [111, 112]. However, topiramate should occasionally be withdrawn due to its adverse effects, such as headache, paresthesias, amenorrhea or sedation [111]. Lisdexamfetamine (LDX) is a stimulant of the central nervous system which has also proved its efficacy for treating patients with BED. Recent studies treating BED patients with LDX showed good results in reducing binge eating behaviours, comorbid symptoms and decreasing weight in these patients [114, 115]. However, although LDX is generally safe and well-tolerated, some emergent adverse effects such as elevations in heart rate and blood pressure should be closely monitored by physicians. In sum, the gaps in the literature and the methodological limitations of the studies make the understanding of pharmacotherapy in BED still far from conclusive [113].

Predictors, mediators and moderators of treatment outcomes for patients with BED

A large number of demographic and clinical variables (including eating-related and general psychopathology, as well as personality) have been tested as possible predictors, mediators and/or moderators of treatment outcomes in patients with BED. The baseline predictors most frequently associated with both short-term and long-term treatment outcomes include: motivation and self-efficacy, self-esteem, psychopathology and negative affect, weight/shape concerns and frequency of binge behaviours [102, 116, 117]. Regarding mediational factors, a rapid response (i.e. early symptom improvement) was found to be the most salient mediator of treatment outcome [117]. Finally, a few studies analysing moderators of treatment outcome in patients with BED found that overvaluation of body shape and weight was the most robust moderator between the intervention received and its outcomes, specifically in relation to the reduction in eating-related psychopathology and depression levels [116]. Unfortunately, the results in the current literature are still conflicting and inconclusive [96].

Conclusions and future perspectives

The current review briefly presents a general overview of BED, addressing its prevalence, aetiology, clinical characteristics, treatment options and treatment outcomes. As specified throughout this review, BED is an eating disorder which presents high comorbidity with other medical and psychiatric disorders. Numerous risk factors have been associated with the development and maintenance of the disorder, from individual to environmental risk factors. Moreover, 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 the eating disorder are very low. This brings to the table a debate about which factors may be responsible for the low rates of BED treatment. For example, although BED has been a nosological entity since 2013 with the publication of DSM-5, it is still an unknown disorder. Therefore, patients with BED seeking treatment are sometimes referred to other non-specific units of eating disorders. On the other hand, BED patients usually have a strong urge to lose weight, which interferes with psychological treatments and increases the risk of dropout. In the light of these data, it is important to consider the clinical implications. It would be necessary to determine which therapeutic tools might be effective for improving the treatment of patients with BED, especially for reducing the dropout rates. In this regard, further studies are needed to assess the extent to which patients with BED can benefit from other therapeutic approaches, for example, by designing comprehensive treatments which include psychological therapies complemented by adjuvant treatments, such as exercise or mindfulness.

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