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A multi-perspective analysis of dissemination, etiology, clinical view and therapeutic approach for binge eating disorder

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Summary

Eating disorders (ED) constitute the third most common group of chronic diseases among people aged 14–19 years after asthma and obesity, and one of their forms is binge eating disorder (BED). The purpose of the present review was to summarize new research findings on BED and overview the epidemiology, characteristics, criteria, etiopathogenesis, and treatment. Etiopathogenesis of BED is still poorly understood, and the current state of knowledge leads to the conclusion that the pathomechanism of the development and persistence of the symptoms of that disorder is very complicated – factors influencing these symptoms have a genetic, neurobiological, biochemical, cognitive, and emotional background. Treatment targeted at selected pathogenetic mechanisms – i.a., disturbance in the corticostriatal circuit, neurohormonal dysregulation or incorrect regulation of emotions – may be of help for people with binge eating disorder. Often comorbid mental, e.g., mood, anxiety and personality disorders, psychoactive substance abuse, suicidal ideation and suicide attempts, and somatic problems are particularly crucial in the context of primary care physicians and psychiatrists work and should encourage the expanding knowledge about BED and the creation of interdisciplinary therapeutic teams.

Key words: BED, obesity, binge eating disorder

1. Introduction

Eating disorders (ED) constitute the third most common group of chronic diseases among people aged 14–19 years after asthma and obesity [1], and one of their forms is binge eating disorder (BED). Its prevalence is 3–7%, so it is higher than anorexia

nervosa (AN) or bulimia nervosa (BN), despite this, it remains poorly understood probably because it is newly characterized and classified disease. The first scientific descriptions of binge eating were published by Albert Stunkard (1959), who studied eating habits in obese patients. Interest in this phenomenon increased primarily in the 1980s, due to inclusion in DSM-III the diagnosis of bulimia nervosa with binge eating episode as one of its symptoms. In the following years, the researchers [2] began to pay attention to the correlation of the signs of binge eating and obesity, reporting that up to 30% of obese children have binge eating episodes without compensatory behaviors. Another research [3–5] emphasized two main features of binge eating – eating a large amount of food and losing control over it – and described accompanying circumstances, behaviors, and emotions.

As a result of these studies, BED was finally included in DSM IV-TR in the group of eating disorders not otherwise specified (EDNOS) and described in detail in Appendix B, which contained criteria for further research. In DSM-5 published in 2013, BED was established as a separate feeding and eating disorder, and its criteria are presented in Table 1. It is not possible to refer it to ICD-10 because it does not include BED, but only overeating associated with other psychological disturbances (F50.4) without specific diagnostic criteria. It is proposed to include BED as one of the main eating disorders in ICD 11, classifying it under the reference number 6B82 [6]. The suggested diagnostic criteria are slightly different from those described in DSM-5. The same frequency of binge episodes (once a week) is predicted, but the duration of the disorder is not specified, using the term "several months". Moreover, binge eating has been characterized in more detail considering that the patient can eat not only more but also different than usual. Not just the quantity but also the quality of the consumed food takes on meaning. Furthermore, the feeling of lack of control is described in detail – it means not only the inability to stop eating but also to limit it, both quantitatively and also qualitatively. In addition, the criteria B1, B2, B3, and B4 included in DSM-5 are not required to diagnose BED according to ICD-11.

2. Etiopathogenesis of BED

Biological factors

Etiopathogenesis of BED is still poorly understood, and the current state of knowledge leads to the conclusion that the pathomechanism of development and persistence of the symptoms of this disorder is very complex. Research involving patients with BED and their families has shown a significant role of both genetic and environmental factors, with an inheritance of 57% [7].

So far, no genome-wide association study (GWAS) has been carried out in the BED patient population. It has only been proven [8] that there is a positive correlation between bipolar disorder and eating disorders and the relationship between single

nucleotide polymorphisms (SNPs) in PRR5-ARHGAP8 and the presentation of BED symptoms in patients with bipolar disorder. Therefore it is necessary to continue genetic research in this area.

So far, there are no unambiguous conclusions from studies on the BED relationship with organic and functional changes of the CNS. Most authors suggest that in the course of BED, there is a disturbance in the corticostriatal circuit that is important for the regulation of such processes as motivation or impulse control. These abnormalities are similar to those occurring in other disorders related to impulsive or compulsive behaviors, including addictions [9]. Moreover, Balodis et al. [10] showed that people with obesity and BED have reduced activity of the ventromedial, prefrontal, inferior frontal, and insular regions, compared to people with obesity without concomitant BED.

In recent years, it has been suggested that a crucial role in generating motivation to eat is played by the dopaminergic pathways and the μ-opioid system located in the ventral tegmental area (VTA). Studying the response of BED patients to the μ-opioid receptor antagonist and placebo [11], by measuring the response to high-calorie and low-calorie foods, showed that the μ-opioid receptor antagonist decreased the activity of the right globus pallidus and putamen, as well as the motivation to reach for high-calorie foods. These results may indicate new therapeutic pathways for patients with BED.

In the search for pathomechanisms of the development of BED, the estrogen hypothesis [12] plays a significant role: both the estrogen receptor alpha (ERα) and serotonin neurons (5-HT) are located in the dorsal raphe nuclei (DRN). Estrogen replacement therapy increases the activity of DRN, and thus, through 5-HT neurons, it inhibits the symptoms of BED. Klump et al. [13] showed a negative correlation between the estradiol level and BED symptoms and a positive relationship between the progesterone level and its exacerbations.

Patients with BED show a significantly lower total cortisol level during the day than those without BED, with no difference in awakening cortisol response (ACR) [14]. Lavagnino et al. [15] confirmed that the 24-h urinary free cortisol concentrations show a negative correlation with the incidence and severity of symptoms of binge eating and other psychopathological symptoms of BED. Levels of cortisol before, during and after the Trier Social Stress Test (TSST) in patients with BED proved to be lower than in people with obesity without BED, and the release of cortisol in response to a stress stimulus – weaker [16].

Many of the studies on the etiopathogenesis of BED have concerned proteins involved in the processes of hunger and satiety and the regulation of metabolism. In the meta-analysis by Culbert et al. [17], higher fasting peptide YY (PYY) level were found in obese subjects with BED, compared to obese subjects without BED, but similar in postprandial measurements. These relationships may be due to reduced sensitivity to

PYY as a satiety signal, which, in consequence, may stimulate the feeling of hunger and overeating in BED patients.

Disputes among researchers are also caused by the participation of ghrelin in the etiopathogenesis of BED – in individual studies the results were contradictory, but already in the meta-analysis [17] it was shown that its concentration is significantly decreased among patients with BED.

The analyses carried out so far have indisputably proved a close, positive correlation between the level of leptin and the incidence of overweight and obesity, regardless of the prevalence of BED symptoms. Obese patients with BED have higher levels of leptin compared to people with healthy body weight, but in comparison with obese people without BED, the results seem to be very inconclusive [17]. Meanwhile, Brandao et al. [18] demonstrated that adiponectin concentrations and leptin/adiponectin ratio were lower in obese subjects with BED than in obese subjects without BED. They considered hypoadiponectinemia as a possible factor affecting the increased energy supply, and consequently, increased weight and binge eating episodes. The positive correlation was shown between the leptin level and the body dissatisfaction as well as loss of control (LOC) but this second correlation concerned only female group [19, 20].

The endocannabinoid system includes anandamide (arachidonoylethanolamide, AEA) and 2-arachidonoylglycerol (2-AG) which play a substantial role in the regulation of nutritional behaviors and the reward system. They are secreted in response to short reduction of food intake in the hypothalamus and modulate the activity of orexigenic and anorexigenic hormones, such as ghrelin, leptin or melanocortins [21, 22]. Moreover, they strengthen the motivation to find food through interactions with the dopaminergic mesolimbic pathways. The experiment carried out by Monteleone [23] showed a significantly higher plasma AEA concentration in patients with AN and BED compared to patients with BN, with no differences in 2-AG level compared to healthy ones [23].

The brain-derived neurotrophic factor (BDNF) is considered one of the main factors involved in the regulation of food intake, hunger and satiety, eating behaviors, and the development of eating disorders. There are few studies on its level among patients suffering from BED. However, its negative correlation with the incidence of overweight and obesity is undisputed. In a study conducted on rats [24], it was shown that BDNF infusions increased the activity of the serotonergic system in the hypothalamus and reduced the perception of appetite in a dose-dependent manner. It is suggested that mutations in the human BDNF gene and its TrkB receptor may disrupt the regulation of hunger-satiety processes and affect the development of obesity. People with the most common polymorphism of the BDNF gene, Val66Met, have a higher BMI compared to those with the Val/Val genotype and have a higher risk of developing obesity and eating disorders by up to 36% [25, 26]. It has been shown [27] that SNP 196G/A of the BDNF gene does not correlate with genetic susceptibility to BN or BED. However,

it may predispose to the presence of severe binge eating episodes and severe course of the disorder.

Analyses of hunger and satiety in people with BED are ambiguous: Mirch et al. [28] indicates that patients with BED take meals with a higher energy value and feel postprandial satiety for a shorter time than people without eating disorders.

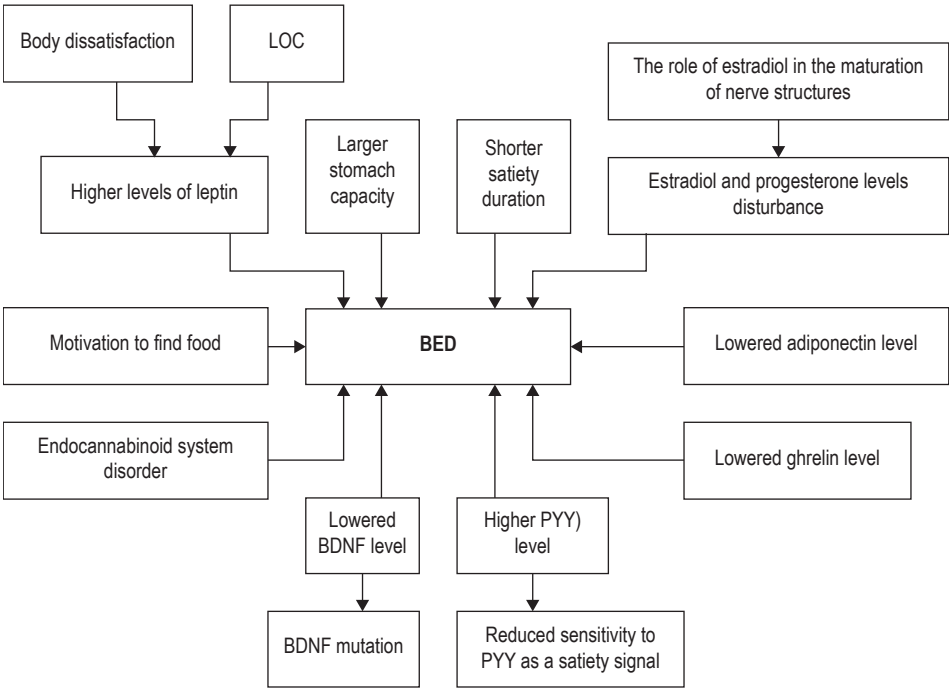

Figure 1. **Biological factors for BED development**

However, these results are not confirmed by the study by Tanofsky [5]. It is also postulated [29] that people with obesity and concomitant BED have a larger stomach capacity, which in turn stimulates eating larger meals. Fluctuations in postprandial glucose concentrations were statistically insignificant for obese patients with and without BED [30].

All of the above studies were conducted on small groups of patients and needed to be expanded. At the same time, they indicate the directions of further research on factors and pathomechanisms important for the onset and the persistence of BED.

Psychological factors

Psychological factors affecting the development of BED include incorrect regulation of emotions and the use of coping strategies based on overeating [31]. The control of emotions through eating is substantially different among obese people without BED symptoms and in those with the diagnosis of the disorder. The factor triggering the binge eating episodes among patients with BED is depressed mood and/or experiencing negative emotional states, while eating food temporarily improves the mood. Such a mechanism was not reported among obese people without BED [32]. It has also been proved that the presence of disturbances in the body perception (overestimation of body size and weight) can be an important factor – it is significantly associated with greater problems in the regulation of emotions in women diagnosed with BED [33].

Studies [34, 35] focusing on the identification of differences in predictors of BED development in children and adolescents and a later age of onset indicate that significant risk factors for developing BED during adolescence are: the female sex and being perceived by parents/significant persons as a child with the excessive body weight. Teenage girls with BED differ significantly in the level of interpersonal sensitivity and psychoticism from girls with other eating disorders, and the tendency to internalize symptoms is more widespread in this group of patients [36].

Comparison of factors in the family system predisposing to the development of particular eating disorders [37] proved that AN, BN as well as BED are correlated with psychopathological features in parents of teenage female patients. However, in the group of parents of female patients with BED, significant differences concerned the level of phobic anxiety and psychoticism in the mothers. Analyzes of the functioning of family systems [38] in which a teenage child suffers from BED revealed less emotional involvement and expression of emotions among the family members in comparison with the control group. From the perspective of teenagers with BEDs, in the family system there is a difficulty in matching emotional needs, safety needs of individual family members, and additionally, rigidity in the context of autonomy and non-adaptive rigidity concerning the changing environment are noticeable.

Dietary factors

Factors contributing to the development of BED also include nutritional factors [39]. Particular attention is paid to the application of dietary restrictions. The theory of containment [40] assumes that the habit of overeating may occur in people who impose on themselves dietary restrictions, inadequate to their physiological drive of eating. This theory is based on the assumption of a biologically determined set point associated with the body weight – higher in the case of people who are overweight and obese compared with people with the normal body weight. The restrictions are

understood as voluntary abstinence from eating aimed at achieving a lower body weight than that biologically determined by the so-called set point.

Among people who impose on themselves dietary restrictions, there is an observed tendency to compensate for emotional tension in the form of excessive consumption, specific excessive cognitive control (rigid, non-adaptive beliefs in the form of rules and orders related to nutrition), dominating over the physiological system of appetite regulation [39]. Stimuli that diminish the individual's ability to maintain this rigid cognitive control may predispose to binge eating episodes [39]. Experiments using very low-calorie diets (VLCD) [41] confirm that imposing dietary restrictions may be a factor triggering episodes of overeating even among people who have not previously had a problem with binge eating.

Some retrospective studies on the nutritional behaviors of people suffering from BED were conducted [42] – they did not reveal the relationship with the frequency of applying restrictive diets, while a significant correlation with the age of starting a restriction was observed. Patients who met the diagnostic criteria of BED made attempts to lose weight at an earlier age than obese people without the problem of binge eating. Moreover, people with BED are distinguished from the healthy population by significantly higher body weight fluctuations during weight-loss attempts and a more frequent occurrence of the yo-yo effect [43].

3. Clinical characteristics

Data on the prevalence of BED vary depending on the study method, oscillating between 2.8% in studies based on patient evaluation by a specialist [44], and 6.6% based on self-assessment questionnaires [45]. The prevalence and course vary depending on the sex [44] – the lifetime prevalence among women is 3.5% and among men 2%. Moreover, men tend to overeat more often than women, who in turn are more likely to lose control over eating [46].

Although the diagnosis rate of BED is higher in obese people actively seeking treatment, e.g., among those awaiting bariatric surgery, it should be noted that only 42% of patients with BED and 28% with subclinical BED reach BMI values above 30 kg/m^2 [44, 47]. 50% of patients have body weight within a normal range or slightly above.

BED more often coexists with other mental disorders [44, 48] – up to 80% of patients may have one, and 50% – three or more additional diagnoses, such as anxiety disorders (65%), mood disorders (46%), post-traumatic stress disorder (26%), or abuse of psychoactive substances (23%). Furthermore, it may coexist with chronic headaches and spinal pains, insulin resistance, and type 2 diabetes, as well as metabolic syndrome [48].

4. BED in the child-adolescent population

Although eating disorders can occur at any age, adolescents and young adults are a particularly vulnerable group. Two BED morbidity peaks are observed: the first at puberty, around 14 years of age [49] and the second around 18–20 years of age [50]. Even 26% of female and 13% of male teenagers experience at least one episode of binge eating within 12 months [51]. BED, like AN and BN, is more common in girls. However, the men to female ratio is smaller compared to other eating disorders. In this age, the diagnosis of BED may cause some difficulties that result from an ambiguous picture of the illness, including lower frequency and irregularity of episodes, than that specified in DSM-5.

The first adult onset of a binge eating episode is twice as frequent as the child-adolescent onset, although the earlier age of onset correlates with a higher risk of full-blown BED and BN, more severe course of the disorder, faster development of overweight and obesity with all consequences. Moreover, it turns out [52] that it is more often associated with a history of trauma, the presence of symptoms of post-traumatic stress disorder, depression, smoking, abuse of alcohol and other psychoactive substances.

5. Diagnostic difficulties

The diagnostic criteria of BED (Table 1) are quite detailed, although they can cause some problems. For example, the definition "Eating, in a discrete period of time (...), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances" is ambiguous, both for obese people who usually eat more, as well as for children and adolescents, who grow up and are more active and therefore have different energy requirements. This unspecified amount of food causes that some authors emphasize the difficulty in referring it to this population and claim that this feature of binge eating is less important than previously thought [3, 4, 53]. Moreover, DSM-5 was completed by a supplement where the importance of a full interview with the patient, the context and the conditions under which episodes occur were pointed out. A large portion of a meal can be associated with special circumstances, such as birthdays or holidays, and therefore should not be considered as a bigger portion of food than usual. Similarly, the term "discrete period of time" is unclear. While in the course of BN the end of the binge eating may be determined by the beginning of compensatory behaviors, in BED it may be difficult to assess it.

Most researchers [54, 55] argue that the loss of control (LOC) over eating, understood as the inability to stop it, regardless of its quantity, quality or taste, should be the most crucial factor for the diagnosis. It seems that this is also not the precise criterion because patients suffering from BED can stop binge eating, for example, when someone calls or suddenly enters the room where they eat. The most common episodes are

sudden and spontaneous, but it happens that patients plan them at a specific time and place and prepare for them, which also contradicts the lack of control.

It is good to know that patients suffering from BED often show the preference for high-fat, sweet products, however, there are more and more patients with episodes of eating large amounts of fruit, yogurt, or healthy food products. Thus, the type of food should not imply a diagnosis or exclusion of BED. It is worth noting that many patients emphasize that during the binge episode they usually eat what they particularly avoid on a daily basis.

Some researchers introduce the definition of sub-clinical or sub-threshold BED (sub-threshold binge eating disorder). According to its diagnostic criteria published in the National Comorbidity Survey – Adolescent Supplement [56], it is enough to consume an above-average large amount of food, accompanied by a sense of losing control, and these episodes need to be present at least twice a week for three months. The criteria of low mood – thus omitting the other diagnostic criteria.

6. BED treatment

Psychotherapeutic treatment

Scientific reports focusing on the long-term effectiveness of the types of psychotherapy dedicated to BED patients show that cognitive behavioral techniques (CBT) [57], interpersonal psychotherapy (IPT) [58] and dialectical behavioral therapy (DBT) [59] are characterized by similar efficacy. A randomized study comparing CBT and IPT with behavioral techniques focused on body weight reduction proves the lower efficacy of only behavioral impacts in the context of weight loss [58].

Due to the numerous dysfunctions observed in the family systems of the patients, including adolescents, with BED problem, the authors of the study [37] suggest that it would also be beneficial to conduct therapeutic intervention focused on the family system. However, no results of studies showing the effectiveness of system therapy in BED have been presented so far.

The influence of group therapy and self-help groups on the reduction of BED symptoms also suggests their usefulness in the treatment of BED [58]. Conclusions from an empirical study conducted on small groups of subjects using meditation techniques [60] indicate their potential value in supporting BED treatment.

Due to the often co-occurring psychological problems in patients with BED (e.g., dysregulation of emotions, non-adaptive coping strategies, mood disorders, anxiety disorders, personality disorders, psychoactive substance abuse, body image disorders), referring to the current scientific reports [61], it is recommended to pay attention to symptoms of comorbid mental disorders. They differentiate subgroups of patients with BED, which significantly affects psychotherapeutic management.

Dietary treatment

Dietary intervention for people with BED should be carried out as a complement to psychotherapeutic methods. The literature data [62] prove that regulating the mode of nutrition, consisting in a regulated and balanced diet, excluding engaging in restrictive diets or models based on the so-called 'cheat days', can reduce the amount of binge eating episodes. However, some empirical studies [60] have shown surprising results – in a group of patients with BED under the influence of a long-term low-calorie diet, e.g., VLCD, BED remission occurs, even taking into account 12 months follow-up [56]. Meta-analyses [43] have shown that caloric restriction can reduce BED symptoms and lead to the body weight reduction in people with overweight or obesity; however, only in the case of the diet therapy conducted in clinical conditions. Moreover, specialists in the treatment of BED [60] suggest that it is essential to develop mindfulness related to nutrition among patients, e.g., by recommending keeping a nutritional diary [62]. In addition to the normalization of eating habits, diet therapy should focus on supporting the treatment of metabolic disorders commonly occurring in this group of patients.

Pharmacotherapy

BED often co-occurs with obesity, and both of these disorders are associated with the presence of many other diseases that significantly affect the length and quality of life of patients. Unfortunately, so far only a few studies have been conducted on this subject, and most of them cover a dozen or so people, mostly women over 18 years of age, and their observation time is not more than few weeks.

In connection with the efficacy of fluoxetine in the treatment of BN, most studies concern both this drug and other antidepressants. So far, it has been indicated that it may reduce the severity of some symptoms of BED, mainly in the first weeks of its use, although it is not clear whether it affects the achievement of remission or weight loss. Also, its advantage over cognitive behavioral therapy has not been demonstrated. Similar conclusions have been drawn from the studies of the other antidepressants, such as fluvoxamine, sertraline, escitalopram, and duloxetine.

One of the best-studied antiepileptic drugs used to treat BED is topiramate. Moreover, it turned out to be the only drug causing a clinically significant body weight loss and reduction in BED symptoms. However, it must not be forgotten that its use was associated with severe side effects and a high percentage of discontinuation of treatment. In one long-term study [63], as many as 68% of patients stopped taking the drug due to poor tolerability.

Obesity is not a diagnostic criterion for BED, but the small number of studies on drugs used in its treatment among patients with concomitant BED is surprising. It has been shown that drugs used to treat obesity, such as orlistat, caused a decrease

in body weight but did not diminish the severity of BED symptoms, which could have discouraged from further similar research. Recently, new drugs have been introduced for the treatment of obesity, of which bupropion/naltrexone is of great interest, and preliminary study results [64] indicate that it can also be useful in relieving BED symptoms.

So far, the only medicine in the world registered for the treatment of medium and severe BED episodes in the adult population is a psychostimulant group drug also used in the treatment of ADHD – lisdexamphetamine (not available in Poland). It has been shown that it reduces both the frequency of episodes of binge eating, allows remission and causes a decrease in body weight. However, it should be remembered that this is not a medicine used to treat obesity without comorbid BED. Interestingly, no RTC study has been conducted so far with the use of other psychostimulants in the treatment of BED, although it is known that they can both inhibit impulsive behavior and cause a decrease in appetite and, secondary to this, also in the body weight.

7. Conclusions

BED is a relatively new and poorly understood illness. Underestimating the number of patients, both among the obese people and those presenting the healthy body weight, is crucial for further research on the pathomechanisms as well as pharmacotherapy, diet therapy and psychotherapy, and perhaps also for the search for new approaches to treat obesity.

Previous studies have usually involved a small group of patients with little statistical significance, which in the context of extrapolation of results to the general population may lead to incorrect conclusions. Therefore, there is a need to research larger group of patients and control groups, also pediatric population, as well as to conduct a genomic analysis using the GWAS method of people diagnosed with BED, which could open further, safer and more effective therapeutic pathways.

Patients with BED significantly often present comorbidity of other psychiatric and somatic disorders, show a higher frequency of suicidal thoughts and attempts, lower quality of life and an inferior level of socio-economic functioning. This is particularly important in the context of the work of psychiatrists, whose current knowledge of this disease entity is still insufficient and requires constant expanding and updating, and should encourage the creation of interdisciplinary teams that would include physicians of various specialties, psychotherapists and dietitians.

Table 1. **BED diagnostic criteria – based on DSM-5**

A. Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following:
1. Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than what most people would eat in a similar period of time under similar circumstances.
2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).
B. The binge-eating episodes are associated with three (or more) of the following:
1. Eating much more rapidly than normal.
2. Eating until feeling uncomfortably full.
3. Eating large amounts of food when not feeling physically hungry.
4. Eating alone because of feeling embarrassed by how much one is eating.
5. Feeling disgusted with oneself, depressed, or very guilty afterward.
C. Marked distress regarding binge eating is present.
D. The binge eating occurs, on average, at least once a week for 3 months.
E. The binge eating is not associated with the recurrent use of inappropriate compensatory behavior as in bulimia nervosa and does not occur exclusively during the course of bulimia nervosa or anorexia nervosa.

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