

# [Binge-eating disorder: theories, risk factors and treatments](https://assignbuster.com/binge-eating-disorder-theories-risk-factors-and-treatments/)

Binge-Eating Disorder

Introduction

Although the addition of Binge-Eating disorder (BED) as a distinct eating disorder diagnosis is new to the Diagnostic and Statistical Manual of Mental Disorders , Fifth Edition, the concept of binge eating is not new to humanity.  Evolutionarily speaking, binge eating was once advantageous.  In the time of hunting and gathering, food availability was scarce and unknown.  Thus, consuming excessive amounts of food was merely a survival instinct.  In the 21 st century however, consuming large amounts food has become significantly maladaptive.  Many of the foods lining shelves of grocery stores are easily accessible, highly processed, and calorically dense.  Consistent consumption of these foods can eventually alter brain function and compromise healthy eating behaviors (Davis, 2015).  Prolonged presence of altered brain function in combination with environmental aspects such as negative emotions results in the psychopathology of Binge-Eating Disorder.

Binge-Eating Disorder is defined by the following characteristics in DSM-5 (2013). BED is an eating disorder involving consuming excessive amounts of food in a finite amount of time.  The amount of food consumed is far greater than one normally would consume in the same setting.  The amount of time is usually defined as in one sitting and less than 2 hours.  This definition does not include eating centered around special events such as a holiday or celebration (i. e. Thanksgiving or birthday party).  Episodes are not restricted to one setting however.  An episode may begin in an area such as a restaurant, but upon returning home the individual continues to consume food even though experiencing feelings of “ fullness” previously.  BED episodes also require feeling a lack of control and marked distress.  While post-binge feelings or shame or remorse are common in both bulimia nervosa and BED, BED does not have any compensatory behaviors such as vomiting or excessive exercise associated with diagnosis.  These episodes must take place at least once a week for 3 months.  This is in contrast with the former criteria of DSM IV, which required the duration to be for at least 6 months (DSM IV).

According to DSM-5 (2013), binge-eating disorder has two subtypes: in partial remission, and in full remission.  To be considered for partial remission, binge eating episodes must happen an average of less than one episode per week.  To be considered in full remission, one cannot meet any criteria for a sustained period after previously meeting the criteria for a binge-eating disorder diagnosis.  BED also has four severity levels. “ Mild” presents with 1-3 binge eating episodes per week, “ Moderate” has 4-7 episodes per week, “ Severe” has 8-13 episodes per week, and “ Extreme” has 14 or more binge eating episodes per week.

The prevalence of BED is most often based on United States measures making it seem like a “ culture bound syndrome” (Davis, 2015).  DSM-5 (2013) reported the prevalence at 1. 6% for U. S.  females and 0. 8% for U. S. males.  Davis (2015) however discovered data reporting a 3% prevalence rate with interview protocols and a 6. 6% prevalence rate using self-questionnaires.  As with many eating disorders, the ratio of females to males diagnosed is much higher.  Binge-Eating Disorder follows this pattern but is less severe than other eating disorders. For example, the ratio of females to males diagnosed with anorexia nervosa is 10: 1 whereas the ratio for BED is closer to 2: 1.  Prevalence is also higher in those awaiting a bariatric procedure.  Although this disorder is common in those seeking weight loss treatment (DSM-5), BED is associated with an elevated or normal body weight.  This may be due to the lack of compensatory behaviors.  Some common comorbidities are disorders of anxiety, depression, and bipolar disorder.  There is also some degree of comorbidity with substance use disorders.

Theories of Binge-Eating Disorder

Many of the theories and models surrounding Binge-Eating Disorder center experiences of negative affect and the different responses.  Across multiple literature works, the definition of negative affect can vary slightly, but the core meaning remains the same.  Negative affect can describe a range of emotions and states such as sadness, anger, grief, or a general negative emotional state.  Consequently, the words affect and emotion will be used interchangeably through the descriptions of the theories and models of Binge-Eating Disorder.

Restraint Theory: Restraint theory suggests that food restriction in those chronically diet requires significant cognitive effort, but when negative affect is present, disruption of the cognitive controls over food and eating behaviors can lead to an episode of Binge-Eating Disorder.  This disorder does not suggest maintenance of binge eating behaviors through minimizing negative affect (Haedt-Matt and Keel, 2011; Fisher and Munsch, 2012).

Escape Theory: Escape theory proposes that binge eating episodes take place to fend off or reduce negative affect by re-directing cognitive thought processes from an area of negative feelings of self to an environmental factor not associated with the negative feelings (i. e. seeking out food).  The binge eating episode takes place in order to intersect the cognitive awareness of negative thoughts.  This contrasts with the previous model of restraint because the reduction of negative affect happens during the episode and tends to reappear after the binge eating episode (Haedt-Matt and Keel, 2011; Fisher and Munsch, 2012).

Expectancy Theory: Expectancy theory suggests that holding onto the belief that negative affect will be reduced after food consumption is what makes binge eating episodes more likely.  Food becomes a reward system and anticipation of the reward can lead to binge eating disorder episodes (Haedt-Matt and Keel, 2011; Fisher and Munsch, 2012).

Affect Regulation Model: Affect regulation models are common in other psychiatric disorders such as substance or alcohol abuse.  As indicated by the above theories, negative affect is strongly associated with BED as well.  Affect regulation model posits that feelings associated with negative affect happen closely before an episode of binge eating, and binge eating is linked to a decrease in negative emotionality.  Binge-eating is then reinforced by negative reinforcement.  In a meta-analysis by Haedt-Matt and Keel (2011), Ecological Momentary Assessments (EMAs)were used to test the hypotheses that “ 1) negative affect would be greater prior to binge eating compared to other times during the day, and 2) negative affect would be lower after binge eating compared to before binge eating” (p. 7).  EMA’s are somewhat effective because they consist of ratings taken at specific time and are capture the short moments during negative affect before a binge eating episode.  The study found evidence supporting negative affect feelings prior to binge eating.  Negative affect levels rose to higher than normal before an episode.  Other findings were contrary to the second hypothesis.  The study found that after an episode of binge eating, negative affect increased instead of decreased.  One explanation for this is that an episode of binge eating may reduce negative affect, but only for a very short period of time.  Another possible reason for these findings is because one necessary characteristic of binge eating episodes is a loss of control or “ numb” feelings during them.  EMA’s may not be able to accurately capture emotions during a binge because of the lack of, or desire to escape from, self-awareness and related negative emotions (Haidt-Matt and Keel, 2011).

Emotion Regulation Model: Emotion Regulation Model was seen first as a model of Borderline Personality Disorder as an elucidation for self-harm behaviors seen in the disorder (Kenny & Singleton & Carter, 2017).  Unlike Affect Regulation model, emotion regulation model does not posit binge eating as a result of attempts alleviate negative affect.  Emotion regulation model for binge-eating disorder sets out to combine all the above theories into one comprehensive model (Leehr et al., 2015).  The study done by Leehr et al. (2015) hypothesized there could be a trigger component and a relief component of emotion regulation as well as wondering if there were emotion regulating differences in obese individuals with or without BED.  The results showed evidence for the trigger component of emotional regulation, but only for adults with BED.  Obese adults without BED were not affected by negative emotion manipulation.  The relief component could not be calculated because only a few studies reported data on post-meal emotionality (Leehr et al., 2015).  Testing the predictions made by an emotional regulation model had some discrepancies in results (Kenny, Singleton & Carter, 2017).  Kenny, Singleton and Carter (2017) found evidence supporting the theory that individuals with Binge-Eating Disorder tend to report more difficulties in impulsive emotion responses, more difficulties with self-awareness and emotions, and more difficulties finding constructive ways to handle negative affect.  The study found compelling evidence for accepting the comprehensive model of emotion regulation.

Food Addiction Model: There are some parallels that can be drawn between addictions seen in substance use disorders and between the behaviors and emotions characteristic of binge-eating disorder.  Drugs and certain foods containing high fat and high sugars both activate the striatal dopamine system. One study even found that 92% of the women in their study met the criteria (in DSM IV) for substance dependence when all instances of the word “ food” were replaced with the word “ drug” (Davis, 2015).  Dopamine and opioid pathways interact in response to pleasurable stimuli.  During opioid abuse, activation of these systems leaves the user feeling in an excited state.  Researchers found that opioid agonist injections in animals tended to increase consumption of foods containing high fat and sugar as well as reward seeking behaviors and willingness to work towards the reward.  Conversely, opioid antagonists tended to decrease consumption and related food-seeking behaviors.  With unmediated long-term consumption of both food and drugs, the brain’s structure becomes altered.  Similar to drug use, habits can be formed and negatively reinforce the behaviors of binge-eating (Smith and Robbins, 2013).

Genetic Risk factors of Binge-Eating Disorder

Several genes have been identified as possible risk factors for eating disorders, specifically Binge-Eating Disorder.  Further studies into food restriction have found that food restriction tends to increase the risk of binge eating episodes in healthy individuals as well as those with irregular eating patterns (Akkerman, Hiio, Villa, & Harro 2010).   Brain derived neurotrophic factor (BDNF) gene and receptor mutations may be an indicator for irregular eating patterns and obesity.  A common polymorphism of BDNF is Val666Met. A meta-analysis study found that people carrying the Val666Met gene are 36% more likely to develop an eating disorder versus the normal Val666Val gene.  In addition, carriers of the Val666Met gene engaging in food restriction behaviors are more likely to engage in binge eating.  More severe food restrictions resulted in an even higher likelihood to engage in binge eating (Akkerman et al., 2010).  Another gene studied is A1 allele of Taq1A.  This gene’s presence results in a decrease in dopamine D2 receptors.  The dopamine system regulates the reward system and presence of A1 causes a dysfunction in in the reward system (Davis, 2015).  This phenomenon has also been observed in those suffering with drug and alcohol abuse and findings have been replicated repeatedly across disorders (Davis, 2015; Smith and Robbins, 2013).  Finally, OPRM1, an opioid receptor gene, a G allele presence can lead to individuals having a heightened sensitivity to outside cues such as highly palatable foods (Davis, 2015).

Treatments of Binge-Eating Disorder

Several therapies have been discovered as effective treatment methods for Binge Eating Disorder.  These treatments include different types of psychotheraputic methods and pharmacological methods.  Treatment plans can vary from involving one type of therapy to a combined type of both medication and therapy.

The first is cognitive behavioral therapy (CBT) has shown to be the most effective and has replicable results across different meta-analyses (Vocks et al., 2010; Brownley et al., 2016).  CBT addresses the irregular patterns of eating and how to cope with feelings of negative affect before a binge (Brownley et al., 2016).  CBT is effective in a variety of BED areas.  CBT has been shown to reduce frequency of binge eating episodes as well as increase binge eating abstinence (Brownley et al., 2016).  Furthermore, CBT has been shown to decrease not only binge eating behavior, but also some of the associated psychological aspects such as concern with weight and shape (Brownley et al., 2016).  CBT did not however, manage to reduce feelings of depression in participants (Vocks et al., 2010).  Vocks et al. (2010) proposed this finding may be caused by CBT being tailored specifically to address negative affect concerning binge eating and not overall depressive behaviors.

A second method of psychotherapy, Interpersonal therapy (IPT) was also considered.  IPT consists of reviews of the participant’s life events in order to identify the main areas of psychological concern (Rieger et al., 20110).  IPT operates much like CBT, however it deals with issues concerning self-identity and focuses on identifying and resolving these such interpersonal issues instead of dealing with cognitive processes and negative affect (Rieger et al., 2010). Rieger et al. (2010) reported finding success rate comparable to that of CBT.

Meditation has also shown to have some worth as an effective intervention for BED.  In a randomized clinical trial from Kristeller, Wolever, and Sheets (2013), Mindfulness-Based Eating Awareness Training (MB-EAT) was able to aid patients in identifying and paying attention to their body’s cues for satiety. According to their study, “…the mechanisms for change designed into the MB-EAT program involve reregulation of appetitive and emotional processes by cultivating awareness, increasing sensitivity to the hedonic process, and disengaging habitual reactivity” (p. 12).  MB-EAT research also identified a trend in improved self-regulation and self-control (Kristeller, Wolever, & Sheets, 2013).

Some pharmacological options include second generation antidepressants (SGAs), lisdexamfetamine, and selective serotonin reuptake inhibitors.  While most of these are effective in reduction of binge eating episodes, they are not comprehensive treatments.  Most of them reduce depressive symptoms only slightly.  Little to no effect was observed on body related or eating related cognitions.  Lisdexamfetamine and SGAs managed to lower obsessions and compulsions felt in the disorder (Vocks et al., 2010; Brownley et al., 2016).

Overall, the most effective method to treat Binge-Eating Disorder appears to be cognitive behavioral therapy.  Not only does it lead to reduced or abstained binge eating behaviors, it also teaches patients valuable cognitive skills that help them deal with the accompanying negative affect of the disorder.

Future studies of Binge-Eating Disorder

First, most of the studies done on binge eating disorder are studies done on Caucasian, adult, women in the United States.  Going forward inclusion of multiple races, genders, and nationalities could aid the generalizability of the information.  In addition, BED is highly prevalent in obese adults.  Exploring this relationship could help spread the study of BED worldwide because obesity is becoming a worldwide health epidemic.  According to the World Health Organization, 13% of the world’s population qualifies as obese.  Studies focusing on BED in children and adolescents should also be considered.  There is a distinct lack of longitudinal studies on Binge-Eating Disorder.  Relapse rates are unpublished, if even measured.  This could become a valuable statistic both for research purposes and for clinicians.  Finally, more studies should be done to test the theories in Food Addiction Model. The parallels between substance/alcohol disorders and eating disorders are compelling.  The presence of the same pathways, the same tendencies for reward seeking behaviors, and similar compulsions and rituals are all evidence that more studies into the theory of “ food addiction”.

REFERENCES

* Akkermann, K., Hiio, K., Villa, I., & Harro, J. (2011). Food restriction leads to binge eating dependent upon the effect of the brain-derived neurotrophic factor Val66Met polymorphism. Psychiatry Research, 185(1-2), 39-43. doi: 10. 1016/j. psychres. 2010. 04. 024
* American Psychiatric Association (2013). The diagnostic and statistical manual of mental disorders, fifth edition: DSM 5. Arlington, VA: American Psychiatric Association.
* Brownley, K. A., Berkman, N. D., Peat, C. M., Lohr, K. N., Cullen, K. E., Bann, C. M., & Bulik, C. M. (2016). Binge-Eating Disorder in Adults. Annals of Internal Medicine, 165(6), 409. doi: 10. 7326/m15-2455
* Davis, C. (2015). The epidemiology and genetics of binge eating disorder (BED). CNS Spectrums, 1-8. doi: 10. 1017/S1092852915000462
* Fischer, S., & Munsch, S. (2012). Self-Regulation in Eating Disorders and Obesity – Implications for Treatment. (English Version Of) Verhaltenstherapie, 22, 158-164. doi: 10. 1159/000341540
* Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the Affect Regulation Model of Binge Eating: A MetaAnalysis of Studies using Ecological Momentary Assessment. Psychol Bull, 137(4), 660-681. doi: 10. 1037/a0023660
* Kenny, T. E., Singleton, C., & Carter, J. C. (2017). Testing predictions of the emotion regulation model of binge-eating disorder. International Journal of Eating Disorders, 50(11), 1297-1305. doi: 10. 1002/eat. 22787
* Kristeller, J., Wolever, R. Q., & Sheets, V. (2013). Mindfulness-Based Eating Awareness Training (MB-EAT) for Binge Eating: A Randomized Clinical Trial. Mindfulness, 5(3), 282-297. doi: 10. 1007/s12671-012-0179-1
* Leehr, E. J., Krohmer, K., Schag, K., Dresler, T., Zipfel, S., & Giel, K. E. (2015). Emotion regulation model in binge eating disorder and obesity – a systematic review. Neuroscience & Biobehavioral Reviews, 49, 125-134. doi: 10. 1016/j. neubiorev. 2014. 12. 008
* Obesity and overweight. (n. d.). Retrieved from http://www. who. int/en/news-room/fact-sheets/detail/obesity-and-overweight
* Rieger, E., Buren, D. J., Bishop, M., Tanofsky-Kraff, M., Welch, R., & Wilfley, D. E. (2010). An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. Clinical Psychology Review, 30(4), 400-410. doi: 10. 1016/j. cpr. 2010. 02. 001
* Smith, D. G., & Robbins, T. W. (2013). The Neurobiological Underpinnings of Obesity and Binge Eating: A Rationale for Adopting the Food Addiction Model. Biological Psychiatry, 73(9), 804-810. doi: 10. 1016/j. biopsych. 2012. 08. 026
* Vocks, S., Tuschen-Caffier, B., Pietrowsky, R., Rustenbach, S. J., Kersting, A., & Herpertz, S. (2009). Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. International Journal of Eating Disorders. doi: 10. 1002/eat. 20696