Binge Eating Disorder and Food Addiction

Ashley N. Gearhardt, M.S., M. Phil,1*, Marney A. White, Ph.D., M.S.,2 and Marc N. Potenza, M.D., Ph.D.2,3

1Department of Psychology, Yale University, New Haven, CT
2Department of Psychiatry, Yale School of Medicine, New Haven, CT
3Departments of Child Study and Neurobiology, Yale School of Medicine, New Haven, CT

Abstract

Binge eating disorder (BED) shares many characteristics with addictive behaviors (e.g., diminished control, continued use despite negative consequences), and a body of scientific literature is building to support addiction conceptualizations of problem eating. Despite similarities, BED and “food addiction” may represent unique yet overlapping conditions. Although the exploration of food addiction is relatively new, understanding the relationship between food addiction and BED may be informative in understanding the mechanisms underlying the development and maintenance of problem eating. In the following paper, we 1) examine the theoretical similarities and differences between BED and addiction, 2) review recent empirical evidence that speak to the relationship between BED and food addiction and 3) discuss the implications of associations between BED and food addiction with respect to clinical interventions.

Keywords

Food Addiction; Binge Eating Disorder; Eating Disorders; Obesity; Cognitive-Behavioral Therapy

Interest in the concept of food addiction has recently received greater attention, in part due to the similarities between the behavioral indicators of addiction and binge eating disorder (BED), such as diminished control over consumption and continued excessive consumption despite negative consequences [1]. Animal models suggest a relationship between binge eating and addictive-like eating. Rats given food elevated in highly palatable or processed ingredients (e.g., sugar, fat) display behavioral indicators of binge eating, such as consuming elevated quantities of food in short time periods and seeking out highly processed foods despite negative consequences (i.e., electric foot shocks) [2,3]. In combination with these behavior changes, these rats exhibit neural changes implicated in drug addictions, like...
reduced dopamine D2 receptor availability [3]. These findings suggest that highly processed foods may possess addictive properties that may underlie problematic eating for some individuals. Given that BED and substance dependence share certain diagnostic indicators (see below), it might be possible that the adaptation of empirically validated therapeutic approaches to drug addiction for BED may improve treatment outcomes. In order to more fully consider this possibility, it is important to examine the relationship between BED and food addiction. In the following paper, we will 1) describe theoretical similarities and differences between BED and addiction (food and drug), 2) review recent empirical data examining the relationship between BED and food addiction and 3) discuss the implications of associations between BED and food addiction with respect to clinical interventions.

Theoretical Similarities and Differences between BED and Food Addiction

Although the term “addiction” has been used for centuries to describe excessive, harmful patterns of substance (alcohol or drug) consumption, the term “dependence” has been used in the Diagnostic and Statistical Manual (DSM) for decades. The decision to employ the term “dependence” was debated and remains controversial. While members of prior DSM substance use disorders task forces are reported to have agreed that addiction refers to compulsive drug use, there was concern regarding the use of the term addiction (e.g., with respect to stigma) [4]. Presently, there is renewed interest in the possible use of the term in the DSM, particularly as the term dependence may also indicate non-addictive patterns of substance use (e.g., hypertensive individuals being treated with beta-adrenergic antagonists may become physically dependent on a drug without being addicted) [4,5]. As such, the core features of addiction have been discussed and non-drug behaviors (e.g., gambling) and substances that have not traditionally been viewed as addictive (e.g., food) have been considered within addiction frameworks [6,7].

While substance (alcohol, drug) dependence diagnoses have been defined in the DSM for decades, BED is not yet considered an established diagnosis and is currently included as a provisional diagnosis in Appendix B of the DSM IV-Text Revision (DSM-IV-TR) [8]. Diagnostic criteria for BED in the DSM IV-TR include recurrent episodes of binge eating that are not accompanied by inappropriate compensatory behaviors (e.g., vomiting, laxative abuse) [8]. BED is also associated with feelings of guilt and embarrassment about the episodes of binge eating [8] and with elevated concerns with shape and weight [9, 10]. Indicators of substance dependence (and the proposed criteria for food addiction) include the amount of time spent acquiring, using, or recovering from the substance and the discontinuing of previously important activities due to substance use, as well as tolerance and withdrawal [8,11]. Substance dependence and food addiction conceptualizations are also characterized by diminished control over substance use/consumption, continued use/consumption despite negative consequences, and diminished ability to cut down or abstain use/consumption of the substance (DSM) [8,11]. In other words, BED and substance dependence are both associated with diminished control during consumption, as well as a diminished ability to reduce the quantity or frequency of use [1, 12, 13, 14]. Further, BED and substance dependence are both marked by continued excessive patterns of consumption despite negative consequences, such as physical or emotional problems [1, 12, 13]. Factors such as impulsivity and mood dysregulation have also been associated with both BED and substance dependence, and these may be importantly involved in different stages of the disorders (initiation, maintenance, relapse) [14, 15]. BED and substance dependence also share similar triggers, like elevated cravings and negative affect [16, 17] (See Table 1).

Behavioral and clinical similarities between BED and addiction may be reflected in shared neural correlates. Dopamine (DA) activation is implicated in the motivation to consume drugs and food [18]. Animal studies have identified similar patterns of DA activity in
response to both binge eating and substance dependence. In response to novel palatable foods, rats show increased DA response, which typically wanes with repeated consumption of the food. However, in rats trained to “binge” eat, DA activity does not diminish with repeated exposure – that is, opportunities to binge on familiar foods continue to result in elevated DA responses [2]. This parallels the pattern of sustained DA response found during repeated nicotine administration [2, 19, 20]. BED and substance dependence also share similar patterns of neural activation in response to food and drug cues, respectively. Schienle and colleagues found that individuals with BED exhibited elevated activation in the medial orbitofrontal cortex (OFC) in response to food pictures relative to overweight healthy control subjects, normal-weight healthy control subjects, and normal-weight patients with bulimia nervosa [21]. Further, BED participants relative to normal-weight healthy control subjects exhibited greater grey matter volume in the medial OFC, which may relate to neural dysfunction in this region [22]. Elevated medial OFC activation has also been linked to drug-related cravings [23] and a greater motivation to consume drugs among substance dependent individuals [24]. A recent study also found enhanced dorsal striatal dopamine release in obese BED participants relative to non-BED obese participants during exposure to food cues [25]. In substance dependence, activation in the dorsal striatum has been implicated in the habitual, rigid, and automatic nature of drug consumption in drug-addicted individuals [26]. Thus, BED and substance dependence appear to share multiple behavioral, clinical, and neurobiological similarities.

Despite the similarities, BED and drug addictions also differ in important ways. Although diminished control over consumption is a core feature of both BED and addiction, the definition differs by disorder. In BED, an episode of out-of-control eating (i.e., binge eating) occurs when an objectively large quantity of food is consumed during a discrete period of time and is accompanied by a subjective sense of feeling out-of-control [8]. Thus, overeating that occurs over the course of a day, rather than during a discrete period of time, would not fit the BED definition of binge eating. In contrast, diminished control in substance dependence is marked less by a specific time course of consumption, but rather by substance use that occurs in a greater quantity or higher frequency than intended [8]. For example, if someone plans to have only one or two drinks after work, but proceeds to drink late into the night, this person would exhibit signs of diminished control over consumption according to substance dependence. In contrast, this type of pattern of food consumption may not meet the criteria for a binge eating episode. Given less specificity in the criterion for diminished control in substance dependence, individuals with clinically relevant disordered eating that would not meet BED criteria, but could possibly result in obesity, may be captured under an addiction framework.

Another difference between eating disorders and substance dependence is the role of the substance (e.g., food). For BED, a greater emphasis is typically placed on understanding and treating the role of individual factors, such as eating-related cognitive distortions, shape/weight concern, and dietary restraint, but less attention is paid to the attributes of the problematic foods. In contrast, the role of the substance in triggering the problematic behavior, in combination with individual factors, occupies arguably a larger focus in addiction. For example, the hedonically rewarding nature of an addictive substance is thought to elicit strong approach motivations (e.g., cravings) and behaviors [27] and to reinforce substance-seeking behaviors [28]. The ingestion of an addictive substance is also thought to sensitize the dopaminergic system to related cues [29] and alter physiological systems that result in tolerance and withdrawal [30]. Excess consumption of highly processed foods also appears related to many of these factors. High-calories foods are associated with elevated cravings [16] and are strongly reinforcing [31]. With regard to sensitization, elevated dopamine-related neural activations in response to food cues have been demonstrated in participants exhibiting features of food addiction [32]. Although the
exploration of tolerance and withdrawal to food is in its nascent stages, rats exhibit features of tolerance and withdrawal to sugar \[2\] and processed foods \[3\]. Thus, the adoption of an addiction perspective to problematic eating may reveal more information about the psychological and physiological impact of chronic overconsumption of highly processed foods.

**Empirical Evidence of the Relationship between BED and Food Addiction**

Despite the theoretical similarities and differences between BED and an addiction perspective of excessive or problematic eating, empirical exploration of food addiction and its relationship with BED has just started. One possible reason for the delay had been the lack of a validated tool to operationalize food addiction. The Yale Food Addiction Scale (YFAS) \[11\] was developed to fill this gap in the literature. The YFAS adapts the diagnostic criteria for substance dependence to apply to eating behaviors. The original validation of the YFAS in a non-clinical sample found evidence of adequate internal reliability, as well as convergent and incremental validity \[11\]. A recent examination of the YFAS in a clinical sample of obese patients with BED further supported the psychometric properties of the scale and provided information about the relationship between food addiction and BED \[33\]. First, a “diagnosis” of food addiction was met by 57% of BED patients, which suggests that despite multiple similarities, the constructs do not entirely overlap. Additionally, compared to patients without food addiction, participants who met the food addiction criteria had significantly higher levels of poor self-esteem, depression, negative affect, emotion dysregulation, and eating disorder psychopathology, but did not exhibit significantly different levels of dietary restraint. Although other factors related to binge eating (i.e., negative affect, eating disorder psychopathology) did not predict binge eating frequency, higher food addiction scores were related to more frequent binge eating episodes. Thus, the subset of BED patients classified as having food addiction by the YFAS may represent a more impaired group, with the greater impairment potentially related to the impact of an addictive process or pre-existing vulnerability factors \[33\].

Another recent study investigated the neurobiology of food addiction in individuals who do not currently meet criteria for BED. In a functional magnetic resonance imaging paradigm, the neural correlates of food addiction as measured by the YFAS were examined in a sample of young women, none of whom had an eating disorder diagnosis \[32\]. Participants who endorsed a greater number of food addiction features exhibited greater activation in the amygdala, anterior cingulate cortex, caudate, dorsolateral prefrontal cortex, and medial orbitofrontal cortex (OFC) during exposure to a palatable-food cue (i.e., picture of milkshake) relative to a neutral cue (i.e., picture of a glass of water) \[32\]. This pattern of neural activation has been implicated in cue-induced craving and incentive salience in substance dependence \[24, 34, 35\]. Further, participants scoring highly on food addiction severity also displayed reduced activation in the lateral OFC during consumption of a milkshake relative to a tasteless solution \[32\], similar to neural patterns observed in association with disinhibition in cocaine abusers \[36\]. In sum, behavioral indicators of food addiction in the absence of BED appear related to patterns of neural activations implicated in drug addictions. Examining the neural correlates of food addiction in the presence of BED will be an important future direction in understanding the potential role of addiction in binge eating in BED.

**Obesity and the Relationship between BED and Food Addiction**

Food addiction, BED and the co-occurrence of these two constructs may have implications for understanding and combating the current obesity epidemic. Specifically, food addiction and BED both appear to be characterized by excess food consumption, which could result in
elevated body mass indexes (BMIs). For example, BED is associated not only with binge eating episodes, but also with periods of overeating between binge eating episodes [37], which may speak to why BED is significantly and strongly associated with obesity [37, 38]. BED is associated with severe obesity (BMI > 40) [37], is a risk factor for prospective weight gain [39], and prospectively predicts the development of metabolic problems above and beyond the risk attributable to obesity [40]. Although cognitive-behavioral treatments of BED are effective in reducing binge-eating episodes in BED, they do not appear to result in significant weight loss [41, 42]. However, the prevention or successful treatment of BED may prevent future weight gain.

Although evidence of food addiction has been found in lean participants [11, 32], higher endorsement rates are present in obese samples who also have BED [33]. The utility of an addiction perspective in dealing with obesity may come from the type of substance-focused approach previously used to reduce the impact of widespread tobacco use. In combination with more effective behavioral and pharmacological treatment, much of the reduction in tobacco use in the United States has been attributed to changes in the environment, such as increased taxes, reduced availability of cigarette vending machines and restricted advertising [43]. If highly processed foods are also capable of triggering an addictive process, similar efforts that focus on changing the current food environment may provide substantial public health benefit [44].

**Clinical Implications of the Relationship between BED and Food Addiction**

The relationship between BED and food addiction may have many important clinical implications. Cognitive-behavioral therapy (CBT), which is the best-established treatment for BED [45], effectively targets aspects of BED in a manner similar to how CBT targets aspects of substance dependence [46–48]. For example, CBT for BED and CBT for substance dependence monitor consumption, identify automatic thoughts about use, develop alternative coping strategies, and identify triggers for problematic behaviors [46–49]. Furthermore, most techniques associated with CBT for BED and CBT for substance dependence do not inherently conflict with one another [49] (See Table 2). Thus, for BED patients that exhibit attributes of food addiction, CBT techniques used in the treatment of BED (e.g., cognitive challenges to overvaluation of shape/weight) and CBT techniques used in the treatment of substance dependence (e.g., craving-focused coping skill development) could be incorporated. An important next step will be to evaluate whether the integration of other interventions developed for substance dependence (e.g., motivational enhancement, contingency management) or other addictive behaviors (e.g., imaginal desensitization used in the treatment of pathological gambling) into treatments for BED provides any added benefit to treatment outcomes in individuals experiencing addictive-like eating behaviors.

One component that is inherently incompatible between CBT for BED and CBT for substance dependence is the role of abstinence. Although harm-reduction approaches to substance dependence do not require abstinence [50], most empirically supported drug-addiction treatments focus on achieving and maintaining drug abstinence. In contrast, elevated dietary restraint and strict avoidance of “forbidden” foods may be associated with an increased risk for binge eating behavior [51–53]. Thus, a treatment that requires participants to strictly avoid certain foods could possibly result in the exacerbation of binge eating. Alternatively, substances may have a priming effect in addictions. For example, in people with alcohol dependence, a drink of alcohol can lead to biological changes that promote additional drinking [54, 55]. Similarly, animal models of eating behavior have identified a priming effect of highly palatable food consumption in promoting binge eating [56, 57]. As restraint-related cognitive processes (e.g., restrictive food rules, identification of forbidden foods) may not play a substantial role in rat eating behaviors, the impact of highly
palatable foods in these models may be reflective of an addiction-like biological priming effect. An important empirical question for future research will be to examine whether abstaining from all or some “trigger” foods is beneficial or harmful for participants exhibiting addictive eating behaviors.

In sum, although the exploration of food addiction is relatively new, understanding the relationship between food addiction and BED may be informative in identifying the mechanisms underlying the development, maintenance, prevention, and treatment of problematic eating. Among the eating disorders, BED seems particularly linked to addiction given specific features of the disorder (e.g., compulsive eating, excess consumption despite adverse consequences, and diminished self-control over eating behaviors). However, food addiction may also be linked to other patterns of eating behaviors that are associated with non-BED-related obesity. Incorporation of addiction assessments like the YFAS into studies of obesity and eating disorders will help clarify the extent to which patterns of eating reflect addictive engagement in these conditions. Conceptualizing eating behaviors within an addiction framework may help improve prevention strategies, identify potentially novel avenues for developing effective treatments, and inform policy makers with respect to how best to address the obesity epidemic.

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References


Table 1
The Relationship between BED and Substance Dependence

<table>
<thead>
<tr>
<th>Similarities between BED and Substance Dependence</th>
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<tbody>
<tr>
<td>1. Diminished control over consumption</td>
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<tr>
<td>2. Continued use despite negative consequences</td>
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<tr>
<td>3. Diminished ability to cut down or abstain from problematic substance</td>
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<td>4. Elevated levels of impulsivity</td>
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<td>5. Elevated comorbidity with mood/anxiety disorders</td>
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<td>6. Triggered by cravings and negative affect</td>
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<tr>
<td>7. Similar patterns of neural activations</td>
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</table>

<table>
<thead>
<tr>
<th>Differences between BED and Substance Dependence</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. BED is associated with elevated concerns with shape or weight, but substance dependence is not</td>
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<tr>
<td>2. BED diagnosis specifies that consumption must occur during a discrete period of time, but substance dependence does not</td>
</tr>
<tr>
<td>3. Substance dependence diagnosis assesses withdrawal, tolerance, amount of time spent on substance-focused activities and activities given up due to substance use, but BED does not</td>
</tr>
<tr>
<td>4. Substance dependence diagnosis places a greater emphasis on the contribution of the substance (e.g., addictive potential of substances), BED diagnosis does not consider specific types or properties of food consumed (merely the amount)</td>
</tr>
<tr>
<td>5. Substance dependence treatments typically focus on abstaining from the problematic substance, but BED treatments do not</td>
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Table 2
Components of CBT for Substance Use Disorders and Binge Eating Disorder: Similarities, Differences and How an Addiction Perspective May Hold Implications for Treatment Development

<table>
<thead>
<tr>
<th></th>
<th>Substance Use Disorders</th>
<th>Binge Eating Disorder</th>
<th>Addiction Perspectives to be Evaluated for Effectiveness in Treating Problematic Eating</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Consumption</strong></td>
<td>Consumption of addictive substance monitored with goal of reduction and eventual abstinence.</td>
<td>Food consumption monitored with goal of omission of binge episodes (overall food intake is not addressed). Calorie counting/following a specific diet is discouraged. Goal of treatment is to normalize eating, by following a regular meal/snack pattern.</td>
<td>Consumption of trigger foods/addictive foods could be monitored with goal of abstinence from these foods. Caveat: May increase dietary restraint</td>
</tr>
<tr>
<td><strong>Triggers</strong></td>
<td>Identification of triggers: social/interpersonal, environmental, internal (e.g., mood), substance-related cues</td>
<td>Identification of triggers: social/interpersonal, environmental, internal (e.g., mood), food-related cues</td>
<td>Common across substance dependence and BED treatments</td>
</tr>
<tr>
<td><strong>Education</strong></td>
<td>Education on peer-group norms for consumption and health effects of substance use</td>
<td>Education on nutrition, weight, physical effects of binge eating</td>
<td>Education on similarities between addictive substances and processed foods Caveat: May increase dietary restraint</td>
</tr>
<tr>
<td><strong>Abstinence Violation Effect</strong></td>
<td>Development of skills in coping with lapses (e.g., unplanned substance use)</td>
<td>Development of skills in coping with lapses (e.g., unplanned food consumption)</td>
<td>Common across substance dependence and BED treatments</td>
</tr>
<tr>
<td><strong>Identify AutomaticThoughts</strong></td>
<td>Craving-focused, expectancy-focused</td>
<td>Automatic thoughts pertaining to eating concerns/dietary information as well as body shape and weight concerns</td>
<td>Greater attention given to craving and expectancy-focused automatic thoughts</td>
</tr>
<tr>
<td><strong>Behavioral techniques</strong></td>
<td>Cue-exposure response prevention: exposure to drug cues without use to promote extinction of emotional and physiological reactions of craving</td>
<td>Exposure: consumption of moderate amounts of “forbidden” or “trigger” foods</td>
<td>Exposure to food-related cues (e.g., advertisements) followed by consumption of a healthier food option Caveat: May increase dietary restraint</td>
</tr>
<tr>
<td><strong>Develop alternate coping strategies</strong></td>
<td>Develop, practice, and substitute alternative activities to substance use</td>
<td>Substitute alternative activities to eating</td>
<td>Common across substance dependence and BED treatments</td>
</tr>
<tr>
<td><strong>Social Support</strong></td>
<td>Social reinforcement of family, friends</td>
<td>Not a significant component of treatment</td>
<td>Integrate social reinforcement of family, friends</td>
</tr>
<tr>
<td><strong>Skills Training</strong></td>
<td>Social skills training – based on assumption that individuals abuse substances as a means of coping with interpersonal difficulties. Also focuses on developing skills for Drink/drug refusal and enhancing social support network</td>
<td>Social skills training – generally for assertiveness – e.g., to decline food</td>
<td>Include social skills that focus on coping with interpersonal difficulties to replace problematic food consumption and enhancing social support network</td>
</tr>
<tr>
<td></td>
<td>Substance Use Disorders</td>
<td>Binge Eating Disorder</td>
<td>Addiction Perspectives to be Evaluated for Effectiveness in Treating Problematic Eating</td>
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<td></td>
<td>to cope with risky situations to prevent substance use.</td>
<td>risky situations to prevent binge eating.</td>
<td></td>
</tr>
<tr>
<td>Contingency Management</td>
<td>Use of a motivational incentive that is provided if compliant with treatment</td>
<td>Not a significant component of treatment</td>
<td>Can evaluate for efficacy the use of a motivational incentive that is provided if compliant with treatment</td>
</tr>
<tr>
<td>Other</td>
<td>• Anger Management • Decision Making • Problem Solving • Pleasant Activities • Relaxation Training</td>
<td>• Pleasant Activities • Relaxation Training • Problem Solving</td>
<td>Can evaluate the efficacy of developing skills in anger management, decision making, and problem solving</td>
</tr>
</tbody>
</table>