Chapter 12
Food Addiction

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Contents

Abstract For years, the concept of food addiction has been gaining increased attention in the scientific literature and the media. A conceptualization of the condition from a substance use disorder and behavioral addiction perspective has emerged. Even though food addiction remains a controversial diagnosis, with research being in the nascent stages, this condition can have a devastating impact on the lives of those afflicted. In this chapter, the authors provide a comprehensive review of the literature targeting food addiction. Topics for discussion include epidemiology and risk factors, pathophysiology and neurobiology, symptomatology framework based on the diagnostic criteria of behavioral and nonbehavioral

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addictions, physical and psychiatric comorbidities, and proposed treatment avenues of food addiction.

Keywords Food · Hyperpalatable · Addiction · Nutrition · Eating · Mental health

12.1 Introduction to Food Addiction: Is Food Addiction a True Medical Disorder?

Food addiction is a controversial construct and proposed medical diagnosis. Back in 1956, Theron Randolph was the first to introduce the term "food addiction" in the scientific literature. However, earlier in the twentieth century, "eating addiction" was used to refer to individuals with binge eating patterns. Nowadays, the scientific community still debates the validity of food addiction as a medical diagnosis. The use of objective tools, such as the Yale Food Addiction Scale (YFAS), has allowed researchers to better assess and describe the concept of food addiction (Penzenstadler et al. 2019). Furthermore, over the past decade, an increased number of studies have tried to describe the phenomenology of this condition, epidemiology, neurobiology, symptomatology, and the impact it has on affected individuals, as well as its possible treatment avenues. Although ample research has investigated the intricate relationship between food addiction and substance use, eating disorders, and obesity, much more is yet to be understood.

Certain foods, primarily processed foods, refined carbohydrates, and high-fat foods, can contribute to an increased addictive potential. One recent systematic review supports the notion that food addiction is a unique entity that meets similar criteria to other substance use disorders (SUDs) (Gordon et al. 2018).

In this chapter, the authors explore the current evidence about food addiction and provide clinical guidance on its assessment and treatment. First, the epidemiology and risk factors of food addiction will be discussed, followed by a review of research findings of its neurobiology and the conceptualization of food addiction from a SUD perspective. Food addiction symptomatology framework based on the Diagnostic and Statistical Manual of Mental Disorders (DSM-5-TR) criteria will be explored. Key comorbid or associated physical and mental health conditions, encountered among individuals with food addiction, will also be described. Objective tools used to assess food addiction will be highlighted, as well as the role of society and the mainstream media in shaping this condition. Finally, a comprehensive review of available evidence-based treatment interventions will be discussed, with recommendations for future research.

12.2 Epidemiology and Risk Factors of Food Addiction

According to the YFAS, the adult prevalence rates of food addiction range between approximately 5 and 10% in nonclinical samples, 15 and 25% in obese samples, and 30 and 50% in morbidly obese bariatric patients or obese individuals with binge eating disorder (Oliveira et al. 2021). Among children and adolescents, the estimated prevalence of food addiction is estimated to be around 15% (12% among community samples and 19% among overweight/obese samples) (Yekaninejad et al. 2021).

Food addiction is a multifactorial construct that is postulated to emerge secondary to the interplay between biological, genetic, psychological, and social factors.

Biological factors include hormonal imbalances, abnormalities in brain structures, side effects to certain medications, and genetic inherited factors (Liu et al. 2010; Murray et al. 2014). The genetic influence on the risk of becoming overweight or obese is substantial. Changes in gene expression occurring during intrauterine development, or epigenetics changes, can significantly impact an individual's subsequent risk of developing obesity (Rhee et al. 2012; Şanlı and Kabaran 2019). The relationship between genetics and food addiction, on the other hand, is certainly complicated and not yet clear. A recent review described the intricate association of genetic and epigenetic research related to addictive tendencies toward food (Davis and Bonder 2019). A genome-wide association study of 9314 females of European ancestry who were diagnosed with food addiction did not identify a significant association with single nucleotide polymorphisms or genes already implicated in drug addiction (Cornelis et al. 2016). However, a review of neurogenetic evidence in obesity implicated a relationship between obesity and gene polymorphisms that code for dopamine receptor types 2, 3, and 4; the dopamine active transporter; and enzymes associated with dopamine degradation such as catechol-o-methyltransferase, monoamine oxidase A, and monoamine oxidase B (Stanfill et al. 2015).

Biological factors interact with lifestyle and psychosocial factors. For instance, physical exercise can reduce the genetic effects on obesity measures, such as body mass index (BMI) and weight circumference (Lin et al. 2019). Psychological factors that can mediate food addiction include emotional distress and behavioral difficulties associated with weight gain, such as feelings of guilt and weight stigmatization (Adams et al. 2019). Compulsive eating patterns are thought to occur secondary to ineffective self-control schemes (Kayloe 1993), as a means to treat negative emotions triggered by depressive symptoms (Dêbska et al. 2011), or due to psychosocial factors such as high stress, low coping mechanisms, and low emotional support (Mazur et al. 2011). Certain character traits have also been implicated in the development of food addiction. A recent systematic review of 45 studies assessed the role of impulsivity and reward sensitivity in food addiction. While self-reported impulsivity was found to be consistently associated with food addiction, this was not the case with reward sensitivity. The latter, defined as the degree to which an individual's behavior is motivated by reward-relevant stimuli, was inconsistently associated with food addiction. Along the lines of impulsivity, food addiction was

consistently associated with negative and positive urgency (the tendency to act impulsively when experiencing extremely negative and positive emotions, respectively) and lack of perseverance (the tendency to quit when a task becomes difficult or boring) (Maxwell et al. 2020). Lastly, social factors including family dysfunction, peer pressure, social media influence, social isolation, and lack of a support system can also trigger or worsen food addiction. A 2019 review illustrates a biopsychosocial model of risk factors and triggers of food addiction (Adams et al. 2019).

The interplay among the above-described risk factors has been proposed to likely drive the development of food addiction. Food addiction can, therefore, be defined as an entity caused by the interplay between several factors that encourage or stimulate food cravings to reach a state of heightened pleasure or stress relief. Further understanding of these different elements is necessary to identify and implement individualized, targeted, holistic, patient-centered treatment interventions.

12.3 Neurobiology of Food Addiction

Three reviews provide an in-depth discussion about the neurobiology of food addiction. In 2010, Blumenthal and Gold reviewed the similarities in the physiology of addiction and food consumption, the published evidence of food addiction, and the freshly developed tools to better characterize pathological appetitive behaviors (Blumenthal and Gold 2010). Before then, in 2009, Dagher provided an overview of the signaling networks that regulate food consumption and the similarities between drug use and food addiction (Dagher 2009), while Wang et al. reviewed the neurobiology underlying food consumption, including the interactions between peripheral and central signaling systems involved in eating and the mediating role of dopamine (Wang et al. 2009).

At a structural level, four central nervous system (CNS) regions are involved in the regulation of eating: the amygdala/hippocampus, insula, orbitofrontal cortex, and striatum (Dagher 2009). Other implicated brain structures include the hypothalamus and arcuate nucleus, which have also been particularly involved in weight regulation (Wang et al. 2009). To further delineate the role of these structures in food addiction, one study compared functional magnetic resonance imaging (fMRI) in 39 healthy young women ranging from lean to obese. Results showed that participants with higher scores on food addiction scales had significantly greater activation in the dorsolateral prefrontal cortex and the caudate in response to anticipated receipt of food (i.e., a highly caloric and sweet drink). Participants also showed decreased activation in the lateral orbitofrontal cortex in response to receipt of food (Gearhardt et al. 2011). The findings of this study are similar to patterns of neuronal activation observed in SUDs. The activation of these brain regions stimulates implicit learning about “food rewards”, allocating efforts toward maximizing them, and integrating information about bodily energy stores and gut contents with information from the

outside world, such as food types and availability (Blumenthal and Gold 2010; Dagher 2009; Wang et al. 2009).

At a hormonal level, there is growing recognition of the involvement of hormones in food addiction (Blumenthal and Gold 2010). In the CNS, dopaminergic pathways regulate the motivation to consume food and the pleasurable feelings after eating (Dagher 2009; Wang et al. 2009). These dopaminergic pathways interact with other systems including opioid-mediated, GABAergic, and serotonergic circuits. Moreover, orexin and melanin play important signaling roles in hypothalamic circuits, whereas neuropeptide Y and alpha-melanocyte-stimulating hormones regulate neuronal signaling in the arcuate nucleus (Wang et al. 2009).

Disruption of the hypothalamic-pituitary-adrenal axis and levels of corticotropin-releasing factor have been described as mediators of food addiction. Cottone and colleagues demonstrated that rats withdrawn from access to a high-sucrose diet exhibited an increase in mRNA and peptide expression of corticotropin-releasing factor in the central nucleus of the amygdala. Rats also displayed anxious behaviors when unable to access their diet. Upon access to the hyperpalatable food, overeating was noted. Interestingly, these findings in rats were mitigated by pretreatment with a selective corticotropin-releasing factor antagonist (Cottone et al. 2009).

Peripherally, many signaling pathways are involved in regulating hunger and satiety. These pathways are highly interconnected with the central circuits. In particular, four gut- and fat-derived hormones mediate this complex homeostatic regulation: ghrelin, leptin, insulin, and peptide YY. Ghrelin, or the "hunger peptide," is released by the stomach and acts on the hypothalamus to stimulate the dopaminergic reward pathways, leading to an increase in food consumption. Ghrelin typically rises during a fasting period and falls following meal intake. In contrast, leptin acts on the hypothalamus to decrease food intake and increase metabolic rate, inhibiting dopaminergic circuits. Leptin relays information about available fat reserves to the CNS, therefore playing a significant role in long-term energy homeostasis. Insulin and peptide YY, secreted from the pancreas and small intestine, respectively, relay information to the brain about acute changes in energy levels (Wang et al. 2009).

12.4 Food Addiction Conceptualized from a Substance-Use Disorder Perspective

The recent expansion in the food industry allowed the creation of food products with increased rewarding properties. These products typically have a low nutritional value and are saturated with carbohydrates, sugars, fat, and additives. They are referred to as hyperpalatable food and have been described in the literature as "addictive" (Dimitrijević et al. 2015). This paved the way for a framework of food dependence as an analog to SUDs, characterized by the three stages of addiction: binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation.

The binge/intoxication stage involves the dopamine and opioid pathways in the nucleus accumbens and dorsal striatum. The innate reinforcing nature of food is a result of dopamine release in the striatum (Wang et al. 2004, 2009). However, food is found to activate dopaminergic pathways differently than drugs of abuse. In particular, hyperpalatable food causes a delayed increase in dopamine level as a function of increased glucose and insulin. Meanwhile, the opioid system exerts a synergistic effect with dopaminergic pathways to promote food intake. In contrast, drugs of abuse typically increase dopamine through direct pharmacologic effects or, indirectly, through opioid, GABAergic, and cannabinoid systems (Barbano and Cador 2007; Volkow et al. 2008). Interestingly, both food and drugs activate the endogenous opioid system. As such, naltrexone, an opioid receptor antagonist, is used in both weight loss and SUD treatment (Colantuoni et al. 2001; Gearhardt et al. 2009a). The withdrawal/negative affect stage activates the extended amygdala and medial part of the nucleus accumbens, whereas the preoccupation/anticipation stage engages the prefrontal cortex, hippocampus, and insula (Koob and Volkow 2016). Dopamine pathways in these brain structures were found to be modulated in food addiction, as will be discussed below.

Food addiction can be conceptualized from the lens of SUDs. At a physiological level, a hyperpalatable food product activates the reward system. At a behavioral level, this previously neutral stimulus (i.e., hyperpalatable food product) becomes paired with a rewarding effect (i.e., satisfaction or relief) via dopamine and other rewarding brain circuits, leading to conditioned reinforcement. Subsequently, the neutral stimulus becomes a reinforcer of the behavior, leading to an "urge" to consume the food product to achieve the rewarding effect.

Findings from brain imaging studies validate the presence of similarities in central processes between individuals with food addiction and SUDs (Lindgren et al. 2018). Clinical studies of positron-emission tomography imaging in alcohol, stimulant, and opioid use disorders show that a defining trait of SUDs is a reduction in dopamine release and a decrease in dopamine receptor D2 (DRD2) availability in the nucleus accumbens and dorsal striatum (Martinez et al. 2005, 2007). To assess whether food addiction has a comparable pattern, several studies examined dopamine release and availability of DRD2 in the brains of individuals with obesity. Studies have shown that, compared to individuals with low BMI, those with high BMI had less striatal dopamine release in response to consuming glucose (Wang et al. 2014), and reductions in striatal DRD2 availability correlated with increasing BMI (Wang et al. 2001). The lack of dopamine-induced signaling results in decreased functional modulation of reward brain regions, which is thought to mediate the compulsive administration of hyperpalatable food or drugs (Wang et al. 2004).

Imaging studies indicate similar changes in regional brain glucose metabolism in individuals with either food addiction or SUDs. In both groups, an association is seen between DRD2 availability and glucose metabolism in the orbitofrontal cortex and anterior cingulate gyrus (Volkow et al. 2001, 1993). Reduced glucose metabolism in these prefrontal brain regions was found to be correlated with reduced striatal dopamine availability and signaling in individuals with obesity (Volkow et al. 2001).

Decreased glucose metabolism in the executive control centers reflects a decrease in their functioning, which appears to contribute to the lack of inhibitory control over food/drug-taking behaviors observed in these individuals (Volkow et al. 2008). One major difference between addiction to food and drugs is glucose metabolism changes observed in the somatosensory cortex, which is associated with the subjective perception of taste. Individuals with obesity had increased glucose metabolism in the postcentral gyrus in the left and right parietal cortex of the somatosensory cortex (Wang et al. 2002). Given this enhanced activity, palatability is thought to be increased in individuals with obesity, which potentiates the reinforcing properties of food, the intense desire to consume high-calorie meals, and the subsequent risk of developing food addiction (Wang et al. 2004).

In terms of regulation of μ-opioid receptors, individuals with obesity share features similar to those with opioid use disorder. One study showed that compared to lean individuals, those with obesity have significantly lower availability of μ-opioid receptors in brain regions of reward processing, including the ventral striatum, insula, and thalamus. This may promote overeating to compensate for a blunted μ-opioid receptor response (Karlsson et al. 2015).

Another hormone possibly involved in food addiction is norepinephrine. Imaging results in individuals with morbid obesity show a decrease in norepinephrine transporter availability in the thalamus compared to healthy controls (Li et al. 2014). Also, higher emotional eating patterns in individuals with obesity, defined as increased food consumption in response to negative emotions, correlates with lower norepinephrine transporter availability in the locus coeruleus and higher availability in the left thalamus (Bresch et al. 2017). Similar alterations are described in SUDs. In comparison to healthy controls, individuals with cocaine use disorder have significant upregulation of norepinephrine transporters in the thalamus (Ding et al. 2010). Collectively, these findings suggest that fluctuations in norepinephrine availability in the brain are present in individuals with obesity, emotional eating, and SUDs.

Numerous brain imaging studies have elucidated neurostructural correlates between obesity and SUDs. Drug cues have consistently produced activation of the ventral striatum, amygdala, prefrontal cortex, anterior cingulate cortex, orbitofrontal cortex, and insula (Courtney et al. 2016). A similar response is seen when individuals with obesity are exposed to food-related cues in comparison to controls (Rothemund et al. 2007) and upon consuming high- versus low-calorie beverages (Feldstein Ewing et al. 2017). Furthermore, an overlap in activated brain regions was described following the provision of food and drug cues in individuals with cocaine use (Tomasi et al. 2015). Alternatively, BMI was found to be positively correlated with response in CNS regions implicated in reward and attention, including the lateral orbitofrontal and ventrolateral prefrontal cortex, during the initial provision of food cues (Yokum et al. 2011). This suggests that, following exposure to food cues, individuals with higher activation of reward and attention regions might be at greater risk of obesity. Conversely, gains in body weight and fat were found to be associated with an increase in the responsivity of reward and attention brain regions to food cues, including putamen and mid-insula (Stice and Yokum

2016). Individuals with obesity also show, compared to individuals with normal BMI, differences in the activation of regions of food reward and salience before and after food intake (Hogenkamp et al. 2016) and after a 48-h fasting period (Wijngaarden et al. 2015). Finally, higher levels of body fat were found to be associated with frontal gray matter atrophy, particularly in the prefrontal cortex (Willette and Kapogiannis 2015). These findings hint at the loss of executive function and inhibitory control, which are common themes in SUDs, as processes associated with obesity (Lindgren et al. 2018).

12.5 Food Addiction and DSM-5-TR Criteria

Food addiction is a multidimensional and complex condition. Whether or not it has an inherently addictive quality similar to drugs of misuse or it represents a behavioral addiction, similar to gambling disorder, continues to be debated (Lindgren et al. 2018). In this section, the authors explain food addiction from both perspectives.

One of the original conceptualizations of food addiction was to compare it to the DSM criteria of SUDs. The DSM-5 was the first version to include the diagnosis of binge eating disorder, which emphasized the presence of escalated ingestion of food (in amounts larger than what most people would eat in a discrete period) and loss of control over overeating as core symptoms of the illness. This parallels the description of food addiction as a compulsive "food-seeking" behavior (Koob and Volkow 2010). However, the DSM-5 may still fail to capture the full pathology and spectrum of symptoms behind food addiction (Lindgren et al. 2018). There is disagreement about the needed criteria for diagnosing this condition (Meule and Gearhardt 2014b), as the translation of the DSM SUD criteria to food addiction is not straightforward. Empirical evidence for the applicability of some of these criteria, such as tolerance and withdrawal symptoms, is based on preclinical studies (Avena et al. 2008). Nevertheless, almost all symptoms can be found in and applied to humans (Gearhardt et al. 2009a). Cassin and von Ranson showed in their cross-sectional study that all participants with binge eating disorder received a diagnosis of SUD when, in a diagnostic interview, the word "substance" was substituted with "binge eating" (Cassin and von Ranson 2007).

Table 12.1 is an application of the DSM-5-TR criteria of SUDs to food addiction (Dimitrijević et al. 2015; Meule and Gearhardt 2014b). Previous studies showed that the most common symptoms of food addiction are eating large amounts of food over a long period, an attempt to reduce consumption, and continued use despite negative consequences. Rare symptoms include tolerance and time spent on the purchase and consumption of food. Alternatively, the least common or inapplicable symptoms are intoxication, withdrawal, and reduction of social, occupational, or recreational activities secondary to food consumption (Dimitrijević et al. 2015; Meule and Gearhardt 2014b).

The diagnosis of SUDs requires at least two symptoms of the DSM criteria and a clinically significant functional impairment. In food addiction, distress has been

Table 12.1 Food addiction conceptualized as a DSM-5-TR substance use disorder

DSM-5-TR substance use disorder criterion	Food addiction equivalent	Evidence and explanation
Substance often taken in larger amounts or over a longer period than was intended	Food often consumed in larger amounts or over a longer period than was intended	• Core feature in binge eating disorder and bulimia nervosa • Observed in nonclinical population, including individuals with normal weight • Characterized by eating faster than others, eating alone, when not hungry, and until becoming ill • Associated feelings of shame and guilt • Triggers include hyperpalatable food rich in sugar and fat, and pervasive marketing
Persistent desire or unsuccessful efforts to cut down or control substance use	Persistent desire or unsuccessful efforts to cut down or control food intake	• Core feature in binge eating disorder and bulimia nervosa • Recurrent, and commonly failed, engagement in fitness programs, diet plans, and medical intervention (including over-the-counter medications and surgeries) • Weight loss market in the United States achieved a record of 72 billion dollars in 2019 (LLC 2021)
Great deal of time is spent in activities necessary to obtain or use the substance or recover from its effects	Great deal of time is spent in activities necessary to obtain or overeat food or recover from its effects	• Criterion might not be applicable • Might consider engaging in dietary habits as a tool to recover from food addiction
Craving, or a strong desire or urge to use the substance	Craving or a strong desire or urge to eat specific types of food	• Concept of food cravings previously described in the literature (Hormes and Rozin 2010), with noted cultural differences in types of craved food (Komatsu 2008; Lawson et al. 2020; Weingarten and Elston 1991) • Neurostructural activation patterns in the setting of craving overlap across different substances, including food (Kühn and Gallinat 2011; Naqvi and Bechara 2009; Pelchat et al. 2004) • Craved food can be consumed in an addictive-like

(continued)

Table 12.1 (continued)

DSM-5-TR substance use disorder criterion	Food addiction equivalent	Evidence and explanation
		manner • Overeating can be associated with more intense and frequent experiences of food cravings: higher scores on self-reported food craving measures have been found in patients with binge eating disorder, bulimia nervosa, and obesity (Abilés et al. 2010; Van den Eynde et al. 2012) • Association of food consumption with external cues, such as advertisements, sights, smells, and sounds can trigger food cravings or increased preference for highly palatable foods (Corsica and Pelchat 2010)
Recurrent substance use resulting in a failure to fulfill major role obligations at work, school, or home	Recurrent overeating resulting in a failure to fulfill major role obligations at work, school, or home	• Criterion might not be applicable • Might consider morbid obesity and reduced mobility secondary to food addiction as a culprit for failure to fulfill obligations
Continued use despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of the substance	Continued overeating despite having persistent or recurrent social or interpersonal problems caused or exacerbated by the effects of excessive or specific types of food	• Increased social isolation described in obese individuals compared to those with normal weight (Anderson et al. 2006) • Interpersonal problems such as interpersonal distrust, social insecurity, or hostility linked to binge eating behavior, independent of BMI (Fassino et al. 2003; Lo Coco et al. 2011)
Important social, occupational, or recreational activities are given up or reduced because of substance use	Important social, occupational, or recreational activities are given up or reduced because of overeating of food	• Criterion might not be applicable • Shame and guilt in the setting of unhealthy eating patterns can nonetheless trigger feelings that might interfere with social and recreational activities
Recurrent substance use in situations in which it is physically hazardous	Recurrent overeating in situations in which it is physically hazardous	• Criterion might not be applicable • Clinically, this can refer to

(continued)

Table 12.1 (continued)

DSM-5-TR substance use disorder criterion	Food addiction equivalent	Evidence and explanation
		inappropriate food consumption in the context of an acute health condition: eating excessive sugar despite living with a diagnosis of diabetes mellitus or eating excessively following bariatric surgery • Can be applied to eating while driving, as it impairs driving performance and increases the risk for crashes (Alosco et al. 2012; Stutts et al. 2005)
Substance use is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by the substance	Overeating is continued despite knowledge of having a persistent or recurrent physical or psychological problem that is likely to have been caused or exacerbated by overeating	• In preclinical studies, rats exposed to hyperpalatable food continue to consume it despite negative consequences, such as electric shock (Novelle and Diéguez 2018) • Clinically, this can refer to inappropriate food consumption in the context of an acute health condition: eating excessive sugar despite living with a diagnosis of diabetes mellitus or eating excessively following bariatric surgery
Tolerance: the need for markedly increased amounts of the substance to achieve intoxication or desired effect; markedly diminished effect with continued use of the same amount of the substance	Tolerance: the need for markedly increased amounts of food to achieve the desired effect; markedly diminished effect with continued use of the same amount of food	• Evidence from preclinical studies: – Rats provided with intermittent or excessive access to sugar solutions significantly increase their intake over time and display neurochemical changes similar to those observed in drug use (Colantuoni et al. 2001; Rada et al. 2005) – Rats displayed voluntary tolerance for punishment by electrical shock to obtain a particular palatable food (Oswald et al. 2011) • Evidence from clinical studies: – Females who crave carbohydrates develop an increased preference for this food category and tolerance to

(continued)

Table 12.1 (continued)

DSM-5-TR substance use disorder criterion	Food addiction equivalent	Evidence and explanation
		its ability to ameliorate dysphoria (Corsica and Pelchat 2010) – Males provided with chocolate for 3 weeks gradually increased their chocolate intake while reporting a reduction in the pleasantness of taste and desire to eat the chocolate (Hetherington et al. 2002)
Withdrawal syndrome: differs by substance; substance is taken to relieve or avoid withdrawal symptoms	Withdrawal syndrome: when refraining from eating specific foods; specific foods are eaten to relieve or avoid withdrawal symptoms	• Evidence from preclinical studies: – Withdrawal symptoms (tremors, irritability, increased body temperature) occur when animals are deprived of their hyperpalatable food (Di Segni et al. 2014; Novelle and Diéguez 2018) • Evidence from clinical studies: – In a qualitative study, withdrawal-like symptoms were reported by youth attempting to reduce the intake of pleasurable food, including persistent cravings and irritability (Pretlow 2011)
Substance intoxication	Not applicable to food intoxication	• Criterion is not applicable

previously noted by Schwartz and colleagues. In their study of 4283 people, 46% of participants expressed that they would waive 1 year of their life rather than be fat, 15% would waive 10 years, 25% would agree to have no children, 30% would rather be divorced than obese, and 14% alcohol dependent rather than overweight (Schwartz et al. 2006).

Gambling disorder was added in the DSM-5 as a non-substance-related disorder. Similar to SUDs, some of the gambling disorder symptoms can conceivably be applied to food addiction, as denoted in Table 12.2 (Meule and Gearhardt 2014a).

Whether food addiction resonates more with the criteria of SUDs or with those of the non-substance-related gambling disorder remains to be eluded. Regardless, symptoms of food addiction seem to echo several features of both conditions. As such, it may represent a mixture of nonbehavioral and behavioral addictions and, therefore, constitute a separate and novel entity on the spectrum of potentially addictive disorders.

Table 12.2 Food addiction conceptualized as a DSM-5-TR gambling disorder

DSM-5-TR gambling disorder criterion	Food addiction equivalent	Evidence and explanation
Need to gamble with increasing amounts of money to achieve the desired excitement	Need to eat increasing amounts of food to achieve the desired satisfaction	• Equals the tolerance criterion in SUDs
Restlessness or irritability when attempting to cut down or stop gambling	Restlessness or irritability when attempting to cut down or stop overeating	• Equals the withdrawal criterion in SUDs
Repeated unsuccessful efforts to control, cut back, or stop gambling	Repeated unsuccessful efforts to control, cut back, or stop overeating	• Core feature in binge eating disorder and bulimia nervosa
Preoccupation with gambling	Preoccupation with food and eating	• Food addiction is strongly associated with a preoccupation with food, eating, and overeating when feeling distressed (Gearhardt et al. 2009a, 2012; Meule and Kübler 2012)
Gambling when feeling distressed	Eating/overeating when feeling distressed	• Food addiction is strongly associated with a preoccupation with food, eating, and overeating when feeling distressed (Gearhardt et al. 2009a, 2012; Meule and Kübler 2012)
After losing money gambling, often return another day to get even	After dieting or holding off from food intake, may return or relapse into overeating patterns	• Criterion might not be applicable • One can hypothesize that, in food addiction, failed attempts to decrease food intake can be counteracted by unhealthy eating patterns
Lying to conceal the extent of involvement with gambling	Lying to conceal the extent of involvement with overeating	• Core feature in binge eating disorder and bulimia nervosa • Characterized by feelings of shame and guilt associated with excessive eating and trying to conceal the behavior
Jeopardizing or losing a significant relationship, job, or educational or career opportunity because of gambling	Jeopardizing or losing a significant relationship, job, or educational or career opportunity because of overeating	• Criterion might not be applicable • Might consider morbid obesity secondary to food addiction as jeopardizing life opportunities. For instance, with stigma playing a role, human resource professionals underestimate the occupational prestige of obese individuals and are less likely to

(continued)

Table 12.2 (continued)

DSM-5-TR gambling disorder criterion	Food addiction equivalent	Evidence and explanation
		hire them (Giel et al. 2012) • In women, excessive weight is associated with an increased likelihood of taking sick leave days and long-term absenteeism from work (Reber et al. 2018)
Relying on others to provide money to relieve desperate financial situations caused by gambling	Relying on others to provide money to relieve desperate financial situations caused by overeating	• Money spent on binge eating was found to markedly affect the quality of life in individuals with binge eating disorder and bulimia nervosa, the former being particularly bothered by financial problems (Agras 2001)

12.6 Signs and Symptoms of Food Addiction

Food addiction is recognizable by the following cluster of symptoms:

- **Symptoms related to the amount of food consumed.** This includes food consumed in larger amounts or over a longer period than was intended, continuing to eat certain types of foods when no longer hungry, cravings, and persistent desire or repeated failures to reduce the amount of food intake.
- **Symptoms related to time spent on food-related activities.** This includes spending a great deal of time in activities necessary to obtain food, overeat, or recover from food's effects.
- **Symptoms related to consequences of food addiction.** This includes failure to fulfill major obligations at work, school, or home; continued overeating despite persistent or recurrent social or interpersonal problems and despite knowledge of having physical or psychological problems caused or exacerbated by overeating; giving up or reducing important activities; spending a significant amount of money on the behavior; and recurrent overeating in physically hazardous situations.
- **Associated physiological dependence.** This includes food tolerance and withdrawal symptoms.
- **Associated psychological symptoms.** This includes decreased energy, difficulty concentrating, sleep disturbances, restlessness, and irritability.
- **Associated physical symptoms.** This includes chronic fatigue, headache, and gastrointestinal symptoms.

12.7 Food Addiction Scales

In an attempt to provide a standardized measure for the diagnosis of food addiction, the YFAS was constructed in 2008 (Gearhardt et al. 2009b). It is a 25-item instrument that encompasses dichotomous and Likert scale questions. It measures the presence of food addiction symptoms based on the DSM-IV substance dependence criteria (7 items) and associated clinically significant impairment or distress (2 items). The former seven items include diminished control over consumption, persistent desire or repeated unsuccessful attempts to quit, consumption of large amounts of food over a longer period than intended, spending much time obtaining food, giving up important activities, withdrawal, and tolerance. When at least three out of the seven items are met *and* clinical impairment is present, a diagnosis of food addiction can be provided. The YFAS has shown good internal reliability and good convergent validity with measures obtained from similar constructs. Since its creation and validation, this scale has been used in almost all food addiction research and has been translated into several languages (Gearhardt et al. 2009b; Meule and Gearhardt 2014b; Oliveira et al. 2021).

In 2014, the modified YFAS (mYFAS) was created as a briefer assessment tool of food addiction (Flint et al. 2014). The mYFAS consists of nine self-report questions: seven that assess the DSM-IV SUD criteria and two that evaluate clinical impairment or distress. The mYFAS performed similarly on indicators of reliability and validity as the YFAS and yielded similar rates of food addiction symptoms and diagnostic threshold scores (Flint et al. 2014; Lemeshow et al. 2016).

Back in 2016, the YFAS 2.0 emerged to reflect the diagnostic criteria of SUD in the DSM-5 (Gearhardt et al. 2016). The YFAS 2.0 includes 35 items that assess food addiction based on the 11 criteria of SUD in DSM-5. It also lowers the diagnostic threshold of food addiction from three to two symptoms, along with clinically significant impairment or distress. To determine the severity of food addiction, the YFAS 2.0 uses mild, moderate, or severe specifiers. The updated scale appears to have better internal consistency but similar convergent, incremental, and discriminant validity with eating-related constructs compared to the original YFAS (Gearhardt et al. 2016; Schulte and Gearhardt 2017). The YFAS 2.0 has been translated and adapted to multiple contexts (Oliveira et al. 2021).

The YFAS 2.0 was later adapted into mYFAS 2.0, a 13-item measure, with good reliability and similar convergent, discriminant, and incremental validity compared to the full YFAS 2.0 (Schulte and Gearhardt 2017). It was translated into several languages such as French (Brunault et al. 2020) and Chinese (Li et al. 2021; Zhang et al. 2021). The French version of the mYFAS 2.0 was found to have close psychometric properties to the YFAS 2.0 in both nonclinical and clinical samples (Brunault et al. 2020).

12.8 Food Addiction and Impact on Physical Health

Food addiction has a direct impact on well-being. Individuals suffering from food addiction are at increased risk of physical and psychological consequences. Overeating can predispose to the early development of chronic metabolic diseases such as obesity, which may contribute to high cholesterol and glucose impairment, ultimately leading to increased risk of cardiovascular diseases and organ dysfunction. Gastrointestinal issues may also develop secondary to food addiction, possibly due to disruption of gut microbiota and brain-gut interactions, especially if an eating disorder is comorbid (Santonicola et al. 2019). Other debilitating physical consequences described in food addiction may be chronic fatigue, sleep disorders, reduced sex drive, lethargy, headaches, arthritis, kidney or hepatic disease, osteoporosis, malnutrition, and chronic pain. Directly or indirectly, the physical changes caused by food addiction may have an increased toll on the mental health of affected individuals (Wenzel et al. 2020).

Food addiction can occur in healthy individuals with no obesity, who are capable of maintaining normal body weight. Some of these individuals engage in behavioral modifications, such as increasing the intensity or the frequency of their physical activity, to counteract the potential effects of uncontrolled overeating. However, food addiction may still lead to obesity, defined as a BMI greater than or equal to 30. This can be largely considered a preventable disease. Worldwide, by 2016, over 650 million adults were obese (WHO 2021b). The presence of obesity is a major risk factor for stroke, heart disease, diabetes, musculoskeletal disorders, and neoplasms such as endometrial, breast, ovarian, prostate, liver, gallbladder, kidney, and colon cancer (Pi-Sunyer 2009). When obesity occurs in childhood, the individual is at increased risk of premature death and living with disability as an adult. As obesity can be a difficult disease to treat and overcome, some obese individuals may pursue bariatric surgery as a treatment option to improve health outcomes. A 2017 review of literature on food addiction and bariatric surgery found that presurgical food addiction was related to psychopathology (with mixed findings related to substance misuse), but not related to presurgical or postsurgical weight outcomes (Ivezaj et al. 2017). Still, further research is needed to examine any positive or negative, short- and long-term, effects of bariatric surgery on food addiction symptoms (Koball et al. 2020).

Unstable blood glucose levels or low insulin sensitivity as a result of food addiction can lead to diabetes. A study examining a sample of individuals with type 2 diabetes found a link between obesity and food addiction in this population, with impulsivity and food addiction significantly predicting higher BMI (Raymond and Lovell 2015). Although obesity and type 2 diabetes are treatable conditions, living with diabetes represents a management challenge requiring strict treatment, glucose monitoring, and lifestyle modifications to maintain adequate glucose control and prevent comorbid medical complications such as retinopathy, kidney failure, cognitive decline, depression, and neuropathy. A recent 2020 cross-sectional study exploring the relationship between food addiction and type 2 diabetes found that the

presence of food addiction in individuals with diabetes worsens glycemic control and increases BMI and hemoglobin A1C levels. Moreover, there was an increased presence of diabetic retinopathy, neuropathy, nephropathy, and depressive symptoms among individuals with type 2 diabetes and food addiction (Nicolau et al. 2020).

As many individuals suffer from cardiovascular disease, which is the leading cause of death globally (WHO 2021a), it is imperative to understand the role of unhealthy eating patterns in the risk of developing heart conditions. Addictive behaviors, such as smoking cigarettes and drug use, have been associated with an increased risk for premature heart disease (Mahtta et al. 2021; Thylstrup et al. 2015). To minimize the cardiovascular risk, in addition to promoting tobacco, alcohol, and drug use cessation, individuals should monitor and reduce their salt and red meat intake, eat more fruits and vegetables, and engage in regular physical activity. A cross-sectional study among Peruvian adults assessing food addiction, saturated fat intake, and BMI found no differences among men or women but identified a positive correlation between those with reported increased weight, food addiction, and higher saturated fat intake (Lopez-Lopez et al. 2021). Many longitudinal studies have demonstrated the association between the so-called unhealthy lifestyle (high-saturated-fat diet, high caloric intake, poor sleep, and physical inactivity) and cardiovascular disease. Alternatively, studies have suggested that a Mediterranean-type diet, with an emphasis on plant food sources, reduces the risk of obesity and cardiovascular diseases (Anand et al. 2015). Therefore, promoting a low-fat diet with minimally processed low-glycemic content may ultimately help prevent obesity and cardiovascular disease.

No data has been published on food addiction and infertility. However, among many factors, eating disorders and poor diet (lower intake of fruit and higher intake of fast food) have been associated with either longer time to pregnancy, infertility, or reproductive health negative outcomes such as increased risk of miscarriage (Grieger et al. 2018; Linna et al. 2013).

12.9 Food Addiction and Mental Health

An association between food addiction and a wide variety of mental health symptoms has been described in the literature (Burrows et al. 2018; Piccinni et al. 2021). Individuals with food addiction may experience comorbid psychiatric conditions, such as eating disorders, mood disorders, anxiety disorders, and SUDs (Piccinni et al. 2021). Food addiction has also been found to have a significant direct relationship with binge eating, depression, and anxiety (Burrows et al. 2018). This has also been established in adolescents (Skinner et al. 2021).

Common eating disorders are anorexia nervosa, bulimia nervosa, and binge eating disorder. Food addiction may lead individuals to develop any of these eating disorders given the unhealthy patterns of food restriction and binges. Excessive episodes of food restriction, either via reducing caloric intake, skipping meals, or

excessive fasting, may signal the body to perceive extreme hunger, eventually leading to binge episodes or overeating. A recent review article provided evidence that food addiction may be a completely distinct phenomenon separated from other eating disorders (Hauck et al. 2020). Meanwhile, an important criterion in eating disorders is experiencing thoughts of shame or guilt related to overeating after the excessive ingestion of food. As food addiction may lead to the development of eating disorders, it is worth pointing out that eating disorders can also occur in combination with other SUDs, major depression, and anxiety disorders, which may complicate the clinical assessment and management of individuals with food addiction.

Mood disorders are commonly encountered among individuals suffering from food addiction. Depression, isolation, and low self-esteem may occur secondary to food addiction. Food addiction may be highly mediated by mood and impulsivity, which can be affected by chronic stress exerting a negative effect on the hypothalamic-pituitary-adrenal axis function (Kalon et al. 2016). Moreover, as dopamine has been associated with reward, food intake, stress regulation, and gastrointestinal motility, one study explored the relationship of peripheral dopamine levels on depressed patients meeting YFAS criteria for food addiction. The study found that plasma dopamine levels positively correlated with eating behaviors in females and negatively with food addiction in males (Mills et al. 2020). The presence of obesity has been directly linked with the risk of developing depression, and a bidirectional relationship has been described as depressed individuals are also at elevated risk of developing obesity (Luppino et al. 2010). As a wide variety of factors can predispose an individual to develop mood disorders when struggling with food addiction, attention to the severity of any eating disorder is crucial. Binges may be perceived as temporary relief of depressive symptoms. Isolation, ensuing regret, shame, and guilt may precipitate a recurrence of depressive symptoms.

Regardless of the presence or absence of food addiction, it is well established that eating disorders are associated with the highest mortality rate among mental illnesses. Once an individual with food addiction struggles with major depression and/or eating disorder, self-harm behaviors and risk of suicide become potentially fatal complications. Adolescents living with eating disorders co-occurring with depression are at increased risk of suicidal ideations (Patel et al. 2021). Eating disorders remain the third most common chronic illness among adolescents. Adolescents struggling with comorbid depression and eating disorders were found to be at five times increased odds of experiencing suicidal ideations but lower odds of having a suicide attempt (Patel et al. 2021). As per the National Association of Anorexia Nervosa and Associated Disorders (ANAD), the mortality rate associated with anorexia nervosa is 12 times higher than the death rate of all causes of death for females aged 15–24 (ANAD 2021). In particular, suicide accounts for at least two-thirds of the nonnatural deaths among those diagnosed with eating disorders. Specifically, in anorexia nervosa, individuals are significantly more likely to engage in serious suicide attempts, exhibit a higher expectation of dying, and have an increased risk of disease severity, compared to individuals without an eating disorder or with bulimia nervosa (Guillaume et al. 2011).

Food addiction has also been associated with anxiety symptoms, especially in certain populations such as obese individuals and adolescents (Benzerouk et al. 2018; Parylak et al. 2011). Studies have described that individuals who struggle with food addiction and exhibit irrational beliefs (defined as "habitual affect-eliciting thought patterns leading to dysfunctional emotional and/or behavioral responses") show high trait anxiety, depression, and emotional eating patterns (Nolan and Jenkins 2019). Comorbid anxiety disorders in food addiction may add to the burden of the illness, with anxious individuals exhibiting more challenges and increased maladaptive mechanisms to cope with daily stressors.

Alternatively, efforts have been made to understand if there is any relationship between food addiction and cognitive dysfunction. The strength of cognitive inhibitory control exerted by the orbitofrontal, prefrontal, and parietal cortices appears to be weakened in food addiction (DiLeone et al. 2012). A meta-analysis found that adults with binge eating disorder or food addiction have difficulties with core executive functions, performing poorly when completing cognitive tasks related to cognitive flexibility, inhibitory control, attention, and planning (Iceta et al. 2021). When exploring the effects of food addiction on cognition, one study found that adolescents with food addiction exhibited a higher probability of making errors after an incorrect answer, a higher probability of false alarm, and a poorer target sensitivity. They also scored higher on self-reports assessing executive functioning difficulties, binge eating, depressive symptoms, and impulsivity levels compared to adolescents without food addiction (Rodrigue et al. 2020).

12.10 Mindful Eating Amid Fast Food Availability

Life in the twenty-first century is characterized by an unprecedented availability of ready-made foods as perhaps never seen before by our species. The discovery of refrigeration, which started thousands of years ago and has been progressively perfectioned, has helped our species to store and maintain edible organic foods for longer periods. Canning, mass production, and food preservatives are other important advancements toward storing and distributing nutrients (Guerrant et al. 1946; Jones and Jones 1937).

In the twentieth century, the world witnessed the beginning of a restaurant industry known as "fast food." Never had existed a restaurant that would produce warm, ready food with a preset menu before the consumer places their order. Developed nations and wealthy sectors of some developing countries also experienced the concept of "drive-in" restaurants, where one would consume fast food near or inside their car. A new relationship with food started to bud: one where our days and meals do not have to be significantly planned around the procurement and preparation of food with some time in advance. Eventually, many of these places went on to provide services 24 h a day.

Since the arrival of the "fast food revolution" in the United States, Europe, and most of the Western world, the mass production process has become significantly

more efficient (Kearney 2010). Developing countries have also seen an increase in fast food presence, and what some describe as an "obesity epidemic." There is growing concern that they are strongly correlated. The industry is regulated differently in each country, with some countries limiting advertisement and setting stricter nutritional standards in the name of population health.

As the Western civilizations have become more unequal, with the rising wealth gap between groups of people, those of lower socioeconomic status have become more drawn to consuming quick lower cost meals. These are typically heavily advertised in major media outlets, and recent studies showed significant associations of addictive phone use (Domoff et al. 2020) and both overall and commercial TV viewing (Domoff et al. 2021) with addictive eating in adolescents. These meals are mass-produced with low cost, lower quality ingredients (saturated fats, refined sugars, and overall low nutritional value), and increased risk of poor health outcomes, such as obesity, hypertension, coronary artery disease, diabetes mellitus, and metabolic syndrome (Fuhrman 2018). The fat, sweet, carbohydrate, and salt contents of many of these hyperpalatable foods produce a positive reward effect when consumed. As such, in many ways, fast, affordable, and hyperpalatable food has become a viable solution to food insecurity for many struggling families who cannot buy whole foods with high nutritional value or take the time to prepare them.

Eating is a physiological need for the human species, and this behavior can be understood as having biological, psychological, sociological, and cultural elements. Humankind has embraced different levels of ritualistic behaviors around meals. If seen on a spectrum, it could be poised as the simple act of feeding in response to hunger, a complex event that involves significant planning, preset rituals, and different layers of sophisticated sociocultural elements. However, for many, modern life has overall decreased the opportunity to experience communal meals (with a rich social and cultural aspect of eating) and decoupled eating with those aspects that are less about satisfying the hunger sensation and more about sharing resources to maintain family and community well-being.

In the last half a century, the concept of mindfulness has been more and more embraced by the Western world, initially by outliers incorporating Eastern philosophy (mainly from some forms of Buddhism) into their daily lives. Nowadays, mindfulness can be considered a more "mainstream" concept, used by an ever-growing number of mental health clinicians, life coaches, and other wellness or mental health paraprofessionals. Within mindfulness, there has been a modern push to practice "mindful eating," the act to redirect our attention to the present moment when our senses are being stimulated while engaged in food consumption (Nelson 2017). More broadly, some argue that mindful eating can be defined as being fully attentive to all that is related to the act of eating—as one buys, prepares, serves, and consumes food. This includes food-related experiences, cravings, and physical cues that may arise (Nelson 2017).

Mindfulness is a process-oriented, rather than an outcome-driven, behavior. When it comes to mindful eating, the purpose is not to lose weight. It is generally the case that, within a mindful approach, the person's choices often are to eat less, savor eating more, and select foods consistent with desirable health benefits. Studies

have shown that obese patients with binge eating disorder using eating-specific mindfulness-based meditation exercises experience a reduction of binging frequency and intensity, a reduction in depressive symptoms, and a subjective increase in sense of control (O'Reilly et al. 2014). Two recent narrative reviews provide a comprehensive summary of the evidence about the role of mindfulness, mindful eating, and intuitive eating in changing eating behaviors in general (Brewer et al. 2018; Warren et al. 2017). However, because food addiction is a relatively new concept and construct, the search for any studies directly exploring the effect of mindful eating on food addiction did not yield any results at the time of this publication. The following section provides a summary of the currently available treatment avenues for food addiction.

12.11 Evidence-Based Treatment Interventions for Food Addiction

To provide a comprehensive effective treatment plan for individuals struggling with food addiction, it is imperative to identify all comorbid medical conditions and understand the best available treatments for each of them. Collaboration among providers, such as primary care physicians, nutritionists, mental health providers, and counselors, who attempt to identify the best treatment options in their areas of expertise, may offer relief to individuals with food addiction. Unfortunately, there is a lack of clear clinical practice guideline recommendations to target food addiction. Moreover, there are no approved therapies for food addiction among obese individuals. Vella and Pai provide a narrative review of potential pharmacological and psychological treatment strategies for food addiction (Vella and Pai 2017).

A systematic review describing available treatments for food addiction found that the most effective interventions to reduce self-reported food addiction outcomes were a combination of psychotropic medications such as naltrexone and bupropion, bariatric surgery, and lifestyle modifications (Leary et al. 2021). Despite their effectiveness, it is important to properly evaluate and identify the individuals who most likely will benefit from each of these treatments. For example, bariatric surgeries may not apply to normal-weight individuals struggling with food addiction. Rather, these individuals may benefit from early, cost-effective, and practical lifestyle modifications by assessing and changing their diet patterns and physical activity level. The risks and benefits of medication use should be weighed in the medical decision-making while considering the proper indication for any prescribed medication. In the case of bupropion and naltrexone, this combination is FDA approved for weight loss and may target brain pathways involved in food addiction.

A systematic review found no significant evidence for psychosocial interventions in food addiction, such as psychoeducation and intuitive eating (Cassin et al. 2020). However, more specific psychotherapeutic interventions, such as cognitive-behavioral therapy (CBT), may play a role in the treatment of food addiction.

Particularly, telephone-based CBT appears to improve symptoms of food addiction in individuals with binge eating disorder and psychiatric symptoms (depression, anxiety, and emotional eating) after bariatric surgery (Cassin et al. 2020; Sockalingam et al. 2019). In addition, many individuals may choose to participate in self-help groups as a potential treatment option. Given the similarities between food addiction and SUDs, some individuals enroll in 12-step programs, which are free and available worldwide. Some examples of support networks, intended to provide access to peers and mentors who overcame food addiction, are Overeaters Anonymous (OA), GreySheeters Anonymous (GSA), Food Addicts Anonymous (FAA), and Food Addicts in Recovery Anonymous (FA). Further research is needed to investigate how self-help groups, individually or in combination with other psychological strategies (i.e., CBT or motivational interviewing), may target food addiction, ameliorate symptoms, and improve medical and mental health outcomes.

12.12 Conclusions

The past decade has witnessed a surge in research on the topic of food addiction. Food addiction shares a similar neurobiological and behavioral framework with drug use. However, the extent to which it is equivalent to SUDs and behavioral addictions remains an open question deserving further exploration.

It has become evident that the presence of food addiction has implications on physical and psychological health outcomes. To better understand the relationship between the core symptoms of food addiction and other health-related outcomes, studies should examine the development and course of food addiction in clinical and nonclinical populations.

Other relevant research avenues should include the influence of comorbid food addiction when addressing other conditions, such as eating disorders, obesity, and mental health disorders. Future research should focus on identifying specific evidence-based therapeutic interventions for food addiction while keeping in mind the need to create an expert multidisciplinary task force to develop concise clinical guidelines for the management of this condition. Notwithstanding that scientific input on this topic exponentially increased in the last years, our understanding of food addiction remains in its infancy and, therefore, further research efforts are needed in the years to come.

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