Current Behavioral Neuroscience Reports (2018) 5:281–294
https://doi.org/10.1007/s40473-018-0169-2

ADDICTIONS (M POTENZA AND M BRAND, SECTION EDITORS)

The Concept of Food Addiction: a Review of the Current Evidence

Katherine R. Naish[1] · James MacKillop[1] · Iris M. Balodis[1]

Published online: 17 November 2018

Abstract

Purpose of Review With the literature on food addiction expanding rapidly, we aim to provide an overview of what is known about this topic, including its assessment, prevalence, and associated behavioral, clinical, and neurobiological characteristics.

Recent Findings The literature presents compelling evidence for the validity of the concept of food addiction, revealing numerous substantive parallels between compulsive overeating and substance use disorders. Research published since the introduction of a formal measure of food addiction has examined neurobiological characteristics associated with food addiction, providing evidence of similarities between neural responses in individuals with food addiction and those with substance use disorders. Furthermore, food addiction has been associated with heightened depression, anxiety, and eating psychopathology. There is also evidence of poorer treatment outcomes in clinical populations, highlighting the importance of continued investigation of this condition.

Summary The extent to which food addiction is equivalent to substance use disorders remains an open question; however, it is clear that the presence of food addiction has implications for physical and psychological health outcomes. A focus for future research should be identifying specific symptoms of food addiction that contribute to these poor outcomes and greater psychopathology, to inform the development of therapeutic interventions for food addiction.

Keywords Food addiction · Drug · Binge eating · Neurobiology · YFAS · Obesity

Introduction

The past decade has seen a surge in research on the topic of "food addiction" [1]. Although the rewarding nature of food has been recognized for some time [2], it is only recently that an empirical measure of food addiction was developed [3]. Sophisticated neuroimaging techniques and behavioral paradigms have revealed overlap in responses to food and addictive drugs, and studies in both humans and animals have identified neural and behavioral parallels between compulsive eating and substance use disorders. Despite these advances, however, the idea that people can be *addicted* to food items in the same way as they can to drugs of abuse remains controversial (e.g., [4]). The broad scope of the current paper is to summarize the state of this research field, discussing what is currently known about food addiction. The first section of our review defines food addiction, describing its classification, prevalence, and commonly associated characteristics. We then discuss research findings that support the notion of food addiction, describing first the similarities between food and drugs of abuse, and then parallels between overeating and substance use disorders. In the third section, we discuss studies on the neurobiology of food addiction. Finally, we discuss the validity and usefulness of the food addiction model of overeating.

This article is part of the Topical Collection on *Addictions*

✉ Katherine R. Naish
naishek@mcmaster.ca

[1] Peter Boris Centre for Addictions Research, Department of Psychiatry and Behavioural Neurosciences, McMaster University and St. Joseph's Healthcare Hamilton, Hamilton, ON, Canada

What Is Food Addiction?

Classification

The idea that food has addictive properties is not new [2]. Indeed, the first use of the word "addiction" by one of the earliest addiction journals (the *Journal of Inebriety*) in 1890 was in reference to chocolate [5]. The year 1960 saw the introduction of "Overeaters Anonymous"—a self-help group based on an

addiction model of overeating, followed two decades later by the "Foodaholics Group Treatment Program" [6]. Although the addictive potential of food was being discussed in the late nineteenth century [2], it was only in 2009 that a formal measure of food addiction was introduced [3]. The Yale Food Addiction Scale (YFAS; [3]) is a self-report measure based on the Diagnostics and Statistical Manual of Mental Disorders' (DSM) criteria for diagnosing substance-related and addictive disorders [7, 8]. The original version of the YFAS was based on symptoms of substance use disorders listed in the DSM-IV, and assessed seven symptoms: (1) food taken in a larger amount or for a longer period than intended; (2) persistent desire or repeated unsuccessful attempts to reduce or stop intake of certain foods; (3) Much time/activity to obtain, use, recover from eating certain foods; (4) important social, occupational, or recreational activities given up or reduced as a result of symptoms; (5) continued intake of the food(s) despite knowledge of adverse consequences; (6) tolerance (requiring increasingly more of a food to obtain the desired effects, or a markedly reduced effect of eating the same amount); and (7) withdrawal (physical or psychological symptoms when the individual reduces or stops intake of the food). In line with the DSM's criteria for diagnosing substance use disorders, food addiction is considered present if three or more symptoms are endorsed and the respondent reports distress or impairment associated with their symptoms (assessed using additional items on the YFAS). Responses on the YFAS are also used to calculate a continuous symptom count (0–11), or to classify the level of food addiction as "mild," "moderate," or "severe" based on the number of symptoms endorsed [9]. As discussed later in this review, symptom count is sometimes used to define "low" and "high" food addiction in research participants who do not necessarily meet the criteria for food addiction.

To reflect updated criteria for substance use disorders in the DSM-5 [8], the YFAS 2.0 was introduced in 2016 [9]. The main change in the YFAS 2.0 was the addition of four additional symptoms: (8) continued use despite social or interpersonal problems; (9) failure to fulfill major role obligations; (10) intake of certain foods in physically hazardous situations; and (11) craving or strong desire/urge to eat certain foods (see Table 1 for a comparison of different versions of the YFAS). The YFAS has been translated into several different languages (e.g., [10–12]), and there are modified (shortened) versions of both the original [13] and version 2.0 of the YFAS [14]. In addition, Gearhardt and colleagues [15] adapted the content and reading level of the original YFAS items to create the YFAS for children (YFAS-C). A side-by-side comparison of the items on each version of the YFAS can be seen in Table 1.

Prevalence

The prevalence of food addiction is estimated to be around 10% in individuals who fall into the "normal" body mass index (BMI) category, and 25% in individuals classified as overweight or obese [16]. Studies of bariatric surgery candidates, who typically have a BMI in the obese range, have found the prevalence of food addiction to be around 15% [17–20], with the exception of one study that found a prevalence rate of 25% [21]. Food addiction appears to be significantly more prevalent in females than males; however, this could reflect an over-representation of females in studies using the YFAS [16]. Interestingly, studies so far suggest that food addiction is more common in individuals aged over 35 years compared to younger individuals [16], although studies should be conducted to examine age differences systematically.

The prevalence of food addiction is increased in individuals who meet diagnostic criteria for bulimia nervosa or binge eating disorder (BED; [16]). BED is characterized by recurrent binge eating episodes, during which the individual eats more than would be eaten by most people over the same time period, while experiencing a sense of loss of control. Bulimia nervosa is also characterized by binge eating episodes, but individuals also engage in compensatory behaviors such as purging or excessive exercise [8]. According to the current literature, the prevalence of food addiction is around 40–60% in individuals with BED [22–25], and 84–100% in those with bulimia nervosa [24, 26–28].

Associated Clinical and Behavioral Characteristics

The overlap and high comorbidity of food addiction and BED has led some to query whether food addiction represents a subtype of BED, rather than being a distinct disorder [1]. Indeed, the concepts of food addiction, BED, and obesity have been conflated by researchers in the past [1, 29]. Despite their similarities, however, several studies have shown that food addiction and BED do not always co-occur [22, 30, 31]; for example, Davis et al. [30] found that around half of those with BED do not meet the criteria for food addiction, and around 30% of those with food addiction do not meet criteria for BED. Likewise, not all individuals who meet criteria for food addiction are overweight or obese [16, 32].

It is important from a treatment standpoint to distinguish food addiction from BED and obesity, because food addiction is associated with heightened psychopathology and poorer treatment outcomes compared to BED and/or obesity alone. Individuals with both BED and food addiction score higher on measures of depression and emotion dysregulation, and are lower in self-esteem, than those with BED alone [23]. Similar to substance use disorders, food addiction is associated with heightened impulsivity (e.g., [30]), and is significantly comorbid with attention deficit hyperactivity disorder (ADHD; [30, 33]). Food addiction is associated with greater eating psychopathology, such as emotional and external eating, binge-eating, and nighttime eating syndrome [2, 3, 19, 23, 24, 30, 34]. Food cravings are consistently higher in

Table 1 Table compares the items on four versions of the Yale Food Addiction Scale (YFAS): the original YFAS (labeled here as YFAS 1.0), the YFAS 2.0 (updated to reflect changes in the fifth edition of the DSM), the modified YFAS (mYFAS) 2.0, and the YFAS for children (YFAS-C). Note that the YFAS-C was adapted from the original YFAS

Symptom	YFAS 1.0	YFAS 2.0	mYFAS 2.0	YFAS-C
Substance taken in larger amount or over a longer period than was originally intended	• I eat to the point where I feel physically ill	• I ate to the point where I felt physically ill	• I ate to the point where I felt physically ill	• I eat until my stomach hurts or I feel sick
	• I find that when I start eating certain foods, I end up eating much more than planned	• When I started to eat certain foods, I ate much more than planned		• When I start eating, I find it hard to stop
	• I find myself continuing to consume certain foods even though I am no longer hungry	• I continued to eat certain foods even though I was no longer hungry		• I eat food even when I am not hungry
Persistent desire to cut down or regulate substance use; may report multiple unsuccessful efforts to decrease or discontinue use	• How many times in the past year did you try to cut down or stop eating certain foods? (1, 2, 3, 4, 5+)	• I tried and failed to cut down on or stop eating certain foods	• I tried and failed to cut down on or stop eating certain foods	• How often do you try to cut down on certain foods?
	• Not eating certain types of food or cutting down on certain types of food is something I worry about	• I worried a lot about cutting down on certain types of food, but I ate them anyways		• I worry about eating too much food
	• I want to cut down or stop eating certain kinds of food (Y/N)	• I really wanted to cut down on or stop eating certain kinds of foods, but I just could not		• I want to cut down or stop eating certain foods
	• I have been successful at cutting down or not eating these kinds of food (Y/N)	• I tried to cut down on or not eat certain kinds of food, but I wasn't successful		• I am able to cut down on certain foods
Great deal of time spent obtaining the substance, using the substance, or recovering from its effects	• I spend a lot of time feeling sluggish or fatigued from overeating	• I spent a lot of time feeling sluggish or tired from overeating	• I spent a lot of time feeling sluggish or tired from overeating	• I feel tired a lot because I eat too much
	• I find myself constantly eating certain foods throughout the day	• I spent a lot of time eating certain foods throughout the day		• I eat food all day long
	• I find that when certain foods are not available, I will go out of my way to obtain them. For example, I will drive to the store to purchase certain foods even though I have other options available to me at home	• When certain foods were not available, I went out of my way to get them. For example, I went to the store to get certain foods even though I had other things to eat at home		• If I cannot find a food I want, I will try hard to get it (ex. ask a friend to get it for me, find a vending machine, sneak food when people are not looking)
Important social, occupational, or recreational activities given up or reduced because of substance use	• There have been times when I avoided professional or social situations where certain foods were available, because I was afraid I would overeat	• I avoided work, school, or social activities because I was afraid I would overeat there	• I avoided work, school or social activities because I was afraid I would overeat there	• I avoid places that might have a lot of food, because I might eat too much
	• There have been times when I consumed certain foods so often or in such large quantities that I started to eat food instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy	• I ate certain foods so often or in such large amounts that I stopped doing other important things. These things may have been working or spending time with family or friends		• I eat food rather than do other things I like (ex. play, hang out with friends)
	• There have been times when I consumed certain foods so often or in such large	• I felt so bad about overeating that I did not do other important things. These		• I eat so much that I feel bad afterwards. I feel so bad that I

Table 1 (continued)

Symptom	YFAS 1.0	YFAS 2.0	mYFAS 2.0	YFAS-C
	quantities that I spent time dealing with negative feelings from overeating instead of working, spending time with my family or friends, or engaging in other important activities or recreational activities I enjoy	things may have been working or spending time with family or friends		do not do things I like (ex. play, hang out with friends)
	• There have been times when I avoided professional or social situations because I was not able to consume certain foods there	• I avoided work, school or social functions because I could not eat certain foods there		• I avoid places where I cannot eat the food I want
Continued substance use despite knowledge of persistent or recurrent physical or psychological consequences	• I kept consuming the same types of food or the same amount of food even though I was having emotional and/or physical problems (Y/N)	• I kept eating in the same way even though my eating caused emotional problems	• I kept eating in the same way even though my eating caused emotional problems	• I eat in the same way even though it is causing problems
		• I kept eating the same way even though my eating caused physical problems		
Tolerance (markedly increased dose of substance required to achieve the desired effect, or markedly reduced effect when the usual dose is consumed)	• Over time, I have found that I need to eat more and more to get the feeling I want, such as reduced negative emotions or increased pleasure (Y/N)	• Eating the same amount of food did not give me as much enjoyment as it used to	• Eating the same amount of food did not give me as much enjoyment as it used to	• When I eat the same amount of food, I do not feel good the way I used to (ex. feel happy, calm, relaxed)
	• I have found that eating the same amount of food does not reduce my negative emotions or increase pleasurable feelings the way it used to (Y/N)	• I needed to eat more and more to get the feelings I wanted from eating. This included reducing negative emotions like sadness or increasing pleasure		• I need to eat more to get the good feelings I want (ex. feel happy, calm, relaxed)
Withdrawal (syndrome occurring when blood or tissue concentrations of a substance decline in an individual who had maintained prolonged heavy use of a substance; Individual likely to consume the substance to relieve withdrawal symptoms)	• I have had certain withdrawal symptoms such as agitation, anxiety, or other physical symptoms when I cut down or stopped eating certain foods	• When I cut down on or stopped eating certain foods, I felt irritable, nervous, or sad	• If I had emotional problems because I had not eaten certain foods, I would eat those foods to feel better	• I eat certain foods to stop from feeling upset or sick
	• I have consumed certain foods to prevent feelings of anxiety, agitation, or other physical symptoms that were developing	• When I cut down on or stopped eating certain foods, I had physical symptoms. For example, I had headaches or fatigue		• When I do not eat certain foods, I feel upset or sick
	• I have found that I have elevated desire for or urges to consume certain foods when I cut down or stopped eating certain foods	• If I had physical symptoms because I had not eaten certain foods, I would eat those foods to feel better		• When I cut down or stop eating certain foods, I crave them a lot more
		• If I had emotional problems because I had not eaten certain foods, I would eat those foods to feel better		
		• When I cut down or stopped eating certain foods, I had strong cravings for them		
Continued use despite persistent or recurrent social or interpersonal problems	N/A	• I had problems with my family or friends because of how much I overate	My friends or family were worried about how much I overate	N/A
		• I avoided social situations because people would not approve of how much I ate		

Table 1 (continued)

Symptom	YFAS 1.0	YFAS 2.0	mYFAS 2.0	YFAS-C
		• My friends or family were worried about how much I overate		
Failure to fulfill major role obligations at work, school, or home	N/A	• My overeating got in the way of me taking care of my family or doing household chores • I did not do well at work or school because I was eating too much	• My overeating got in the way of me taking care of my family or doing household chores	N/A
Recurrent substance use in situations in which it is physically hazardous	N/A	• I kept eating certain foods even though I knew it was physically dangerous. For example, I kept eating sweets even though I had diabetes. Or I kept eating fatty foods despite having heart disease • I was so distracted by eating that I could have been hurt (e.g., when driving a car, crossing the street, operating machinery) • I was so distracted by thinking about food that I could have been hurt (e.g., when driving a car, crossing the street, operating machinery)	• I was so distracted by eating that I could have been hurt (e.g., when driving a car, crossing the street, operating machinery)	N/A
Craving (intense desire or urge for the drug that can occur at any time)	N/A	• I had such strong urges to eat certain foods that I could not think of anything else • I had such intense cravings for certain foods that I felt like I had to eat them right away	• I had such strong urges to eat certain foods that I could not think of anything else	N/A
Use causes clinically significant impairment or distress	• My behavior with respect to food and eating causes significant distress	• My eating behavior caused me a lot of distress	• My eating behavior caused me a lot of distress.	• The way I eat makes me really unhappy.
	• I experience significant problems in my ability to function effectively (daily routine, job/school, social activities, family activities, health difficulties) because of food and eating	• I had significant problems in my life because of food and eating. These may have been problems with my daily routine, work, school, friends, family, or health	• I had significant problems in my life because of food and eating. These may have been problems with my daily routine, work, school, friends, family, or health	• The way I eat causes me problems (ex. problems at school, with my parents, with my friends)

The first column shows the symptom that the items relate to; here, we show the wording from the DSM-5 which relates to substance use disorders. Unless otherwise stated, the response options for each YFAS version are: *YFAS 1.0:* "Never," "Once a month," "2–4 times a month," "2–3 times a week," "4 or more times or daily." *YFAS/mYFAS 2.0:* "Never," "Less than monthly," "Once a month," "2–3 times a month," "Once a week," "2–3 times a week," "4–6 times a week," "Every day." *YFAS-C:* "Never," "Rarely," "Sometimes," "Very often," "Always"

individuals who meet the criteria for food addiction [30, 35, 36]. Finally, food addiction is associated with higher BMI, higher cholesterol, a history of smoking, and lower physical activity [13]. More generally, food addiction in treatment-seeking individuals is associated with a significantly lower health-related and psychosocial quality of life [17, 37].

Some studies have found food addiction in treatment-seeking individuals to be associated with less weight loss following treatment [38–40], although Koball and colleagues [19] found that food addiction was *not* predictive of weight loss, rehospitalization, or follow-up attendance in a sample of patients who underwent bariatric surgery. Together, these behavioral and clinical outcome characteristics suggest that food addiction is an important factor for consideration in treatment-seeking populations, but further research is needed to examine other factors that could explain the discrepancies between studies.

Support for the Food Addiction Model

Food as an Addictive Substance

Foods that are high in sugar or fat tend to be more palatable than others [41], leading to these foods being commonly overconsumed in both healthy individuals [42] and those with food addiction [21]. The observation that an animal will expend effort, and even endure pain [43, 44], to gain access to a palatable food demonstrates the inherent reward value of food [45]. Indeed, the most commonly craved food in humans is chocolate (e.g., [46, 47])—a food high in both fat and sugar [48]. Animals exposed to a high-sugar diet increase their intake of these foods after only a short period of exposure [49]. In addition, chocolate itself induces a conditioned and persistent preference for the location where the food was delivered, in a similar way to drugs of abuse (e.g., [50]). In some cases, motivation for food surpasses motivation for other substances commonly thought of as "addictive;" Lenoir and colleagues [51] found that rats preferred heavily saccharin-sweetened water over cocaine, regardless of cocaine dose, and even in animals that had been previously conditioned to prefer cocaine over the sweetened water. These results suggest that intense sweetness, or supranormal stimulation of sweet receptors, can be more rewarding than drugs of abuse. These findings also have implications for the highly sugar-enriched food environment in modern society.

Neurophysiological and neuroimaging studies demonstrate that palatable food intake can elicit a neural response similar to that associated with addictive drugs such as cocaine. Specifically, food and drug rewards are associated with striatal dopamine release in both humans [52] and animals (e.g., [53]). In humans, this response is positively associated with the subjective experience of hunger and food enjoyment [52, 54]. Similarly, frontostriatal dopamine release relates to craving in cocaine use disorder [55]. A growing body of neuroimaging work now demonstrates substantial overlap between palatable foods and addictive substances, with common patterns of brain activation associated with exposure to food and drug cues [56, 57]. This overlap could reflect a shared mechanism by which individuals develop dependence on palatable food or drugs.

Prolonged exposure to high-fat or high-sugar foods is also associated with neural changes similar to those associated with chronic substance use. In rats, the expression of striatal dopamine receptors is affected by long-term intake of a high-fat [58], high-sugar [49], or "cafeteria" style [43] diet. Similar alterations in dopamine receptor availability have been observed in substance use disorders [59]. Although the direct relationship between dietary intake and long-term alterations in dopamine has not been examined in humans, alterations in dopamine receptor availability have been found in individuals who are obese or overweight [60, 61] and those who engage in binge eating [62]. Preclinical studies are beginning to provide mechanistic insights: in rats, exposure to high-fat foods combined with food deprivation increases the expression of fumarate hydratase, ATP synthase subunit alpha (ATP5a1), and transketolase [63]—proteins that have been identified as possible biomarkers of vulnerability to cocaine addiction [64]. Thus, both the acute and long-term effects of exposure to certain food types bear resemblance to those associated with drug use, supporting the idea that food could act as an addictive substance.

Addictive Behavior in Animals Exposed to High-Fat and High-Sugar Foods

In further support of the food addiction model, preclinical studies show that animals exposed to high-sugar, high-fat, and other highly palatable, high-energy diets display some of the behavioral characteristics associated with substance use disorders (for a review, see [65]). These behaviors map onto many of the clinical criteria for substance use disorder. Rats given restricted access to a sweet or high-fat food persist in pressing a lever associated with reward for a longer period (when the reward was no longer being delivered) than animals given no or unrestricted access to the food [66, 67], exhibiting attenuated extinction learning. The extended and perseverative responding to obtain the rewarding food in these animals could be analogous to "a great deal of time spent obtaining, using, or recovering from substance use" in humans—one of the core DSM-5 criteria for substance use disorders [8, 65]. Demonstrating continued use despite negative consequences (as well as use in a physically hazardous environment), Johnson and Kenny [43] found that rats exposed to the cafeteria diet—a varied diet comprising high-fat and high-sugar foods—continued to access the food when doing so was

associated with a conditioned foot shock (see also [44]). In contrast, animals with no or restricted access to this diet reduced their food intake when the foot shock was introduced [43]. In another study, animals continued to seek the food when it had been altered to taste unpleasant [68].

Signs of withdrawal and tolerance have also been observed in animals exposed to high-fat or -sugar diets. Animals given intermittent access to sugar and chow exhibit withdrawal symptoms, such as anxiety, aggression, and teeth-chattering, when access to sugar is subsequently removed (for a review, see [1]). Suggestive of tolerance, animals given either restricted or unrestricted access to high-sugar foods progressively increase their intake after only a short period of exposure [49]. A possible physiological marker of tolerance was reported by Johnson and Kenny [43], who found that animals given extended access to the cafeteria diet exhibit decreased sensitivity to reward. Reward sensitivity was assessed using the brain stimulation reward paradigm, in which reward threshold is reflected in the intensity of electrical stimulation required to elicit self-stimulation in an animal. Extended access (18–23 h daily for 40 days) to the cafeteria diet was associated with weight gain and a progressive increase in reward threshold compared to animals given restricted (1 h per day) or no access to the cafeteria diet [43].

Addictive Behavior in Humans Who Exhibit Compulsive-Eating Tendencies

In humans, behaviors resembling characteristics of substance use and addictive disorders are noted in some individuals who are overweight and those who report compulsive eating. People often eat to excess despite the known negative consequences (e.g., weight gain, metabolic syndrome), and cravings are reported by both healthy individuals (e.g., [69]) and those with eating pathologies ([35, 70]. Eating can also preclude social, occupational, and/or recreational activities: according to a study by Lent and Swencionis [71], 60% of bariatric patients report that they sometimes forego other activities in favor of eating. The same study found that a significant amount of variance in social isolation was accounted for by scores on a putative measure of "addictive personality" (comprising items from the Eysenck Personality Questionnaire) that were previously found to discriminate individuals with a substance use disorder from those without [72], possibly suggesting that social activities were given up. The progression of symptoms in bulimia nervosa could also point to aspects of tolerance in humans; tentative evidence suggests that longer duration of the illness is associated with increased frequency, duration, and severity of binge eating episodes (data cited in [3]). Finally, the presence of self-help groups akin to Alcoholics Anonymous to help people moderate their eating [73] demonstrates that many people eat larger amounts or for longer periods than intended, and engage in (often unsuccessful) attempts to control their food intake. Indeed, the presence of these grass-roots mutual support organizations that adapt addiction-related perspectives reveals the extent to which individuals in the community resonate with the parallels between food addiction and drug addiction.

Another line of evidence in support of the food addiction model is that individuals who engage in pathological overeating display some of the same characteristics as individuals with substance use disorders. Specifically, individuals with BED show higher levels of impulsivity, anxiety, and depression compared to individuals without eating or addictive disorders (for a review, see [74]). Importantly, impulsivity is increasingly understood as a multidimensional construct (e.g., [75]) and food addiction has exhibited particularly notable associations with two forms of impulsivity: negative urgency (e.g., [76, 77]) and delay discounting [78]. In the first case, negative urgency refers to a tendency to act out in response to negative affect and it is an impulsive personality trait that has been robustly associated with substance use disorders in numerous previous studies (for meta-analyses, see [79–81]). In the second case, delay discounting is a behavioral economic index of impulsivity that reflects how much a person values smaller immediate rewards relative to larger delayed rewards. Steep discounting of future rewards has been associated with substance use disorder [82, 83], ADHD [84], and obesity [85] in recent meta-analyses, and, interestingly, food addiction symptom score mediated the link between delay discounting and obesity in a recent study [78].

Finally, environmental antecedents of consumption appear to be the same for food addiction and drug addiction. In the same way that individuals with substance use disorders display attentional biases towards drug-relevant stimuli [86], individuals with BED show an attentional bias towards food (e.g., [87]). In addition, high-palatability food cues elicit craving and other appetitive responses (e.g., [88]), akin to drug cues [89]. Substance use disorders and BED also show similar epidemiological features: chronic or early life stress are strongly associated with the development of both BED [90] and substance use disorders [91], and acute stress is a common antecedent of binge eating episodes and substance use [92, 93]. This shared risk factor could suggest a common mechanism contributing to the development of BED and substance use disorders whereby the experience of early/chronic stress or trauma can alter subsequent reward responses in these individuals.

Neurobiology of Food Addiction in Humans

To date, few studies have examined the relationship between neural activity and food addiction symptoms as measured by the YFAS; however, those that have support the notion of distinct characteristics associated with food addiction.

Gearhardt and colleagues [94] found that when anticipating receiving a palatable milkshake, food addiction severity was positively related with left hemisphere activity in the medial orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), and amygdala. Individuals with high YFAS scores (three or more symptoms) also showed greater milkshake anticipatory activity in the left dorsolateral prefrontal cortex (DLPFC)—a region implicated in inhibitory control and reward expectancy [95]. Heightened amygdala activity during food cue exposure is associated with higher self-rated hunger and desire to eat [96], while medial OFC and ACC activity is associated with higher trait reward sensitivity [97]. These findings point to the particular importance of anticipatory processing and food reward expectations in food addiction. Heightened responsivity of these regions during *anticipation* of palatable food intake could thus contribute to the difficulty in controlling food intake that is characteristic of food addiction. Neural responses during actual receipt of the milkshake was not associated with YFAS scores, although individuals who endorsed three or more food addiction symptoms showed lower activity in the left lateral OFC compared to individuals who endorsed one or no symptoms [94]. Interestingly, *increased* activity in the lateral OFC was associated with increasing satiety in a study of chocolate consumption in healthy participants [98], so it is possible that the relatively low lateral OFC activity during milkshake receipt in individuals with food addiction reflects a weaker satiety response.

There is also emerging evidence for altered neural activity in food addiction, independent of cue reactivity. Similar to Gearhardt and colleagues [94], de Ridder and colleagues [99] reported a positive correlation between YFAS scores and activity in the ACC, but this time in a study of resting-state brain activity. In their study, rostral ACC gamma band activity was positively associated with self-rated hunger in individuals with high YFAS scores (6.8 symptoms on average). This is also one of the first studies to include a control comparison group with substance use disorder and found that self-rated *craving* also related to rostral ACC gamma-band activity in individuals with alcohol use disorder. By contrast, individuals with obesity who had low YFAS scores (3.5 symptoms on average) showed no correlation between hunger ratings and gamma-band activity in the rACC, but a *negative* correlation between hunger and beta-band activity in the same area. Conjunction analyses showed common areas of activity for individuals with high YFAS scores and those with alcohol use disorder, but no significant overlap between the low YFAS and alcohol use disorder group. These findings suggest common resting-state activity in individuals with higher food addiction symptoms and those with alcohol dependence with similar relationships with hunger and drug craving [99].

Food addiction is also associated with greater functional connectivity within frontoparietal brain regions [100], which is similar to what has been observed in individuals with substance use disorders (e.g., [101, 102]). Finally, a neuroimaging study of adolescents with BMIs in the overweight or obese range found no association between neural activation during gustatory cue exposure and YFAS scores [103]. Although the lack of association reported in this study is at odds with the results of the other studies [94, 99, 100], it is important to note that the average YFAS score was lower in Feldstein Ewing and colleagues' participants compared to previous samples. It is possible that the range of food addiction severity was not large enough to detect neural differences in this sample. It is also possible that neurodevelopmental factors affected the relationship between food addiction and brain activity in this sample of adolescents.

Differences in the dopaminergic system have also been linked to food addiction [35, 36]. Food addiction was positively associated with a genetic marker of heightened dopamine signaling, which also correlated with binge eating, food cravings, and emotional eating [35]. Alterations in dopamine transmission in the reward system have been observed in individuals with BED [62] and those with substance use disorders [59]; these findings support the notion of common mechanisms underlying compulsive behavior in these conditions. Along the same lines, Davis et al. [36] found that individuals who met the criteria for food addiction showed no change in appetite when they received the dopamine agonist methylphenidate, which is typically associated with appetite suppression (e.g., [104, 105]). These findings suggest unique food intake patterns following a pharmacologic challenge as well as alterations in dopamine signaling strength in food addiction—the latter being similarly characteristic in substance use disorders.

Validity and Usefulness of the Food Addiction Model

Although there is clear overlap between neural activity associated with anticipation/intake of addictive substance and palatable food, there are also clear distinctions between food and drugs which bring into question the validity of the food addiction model (e.g., [4]). A complication for the food addiction model, which does not typically apply for drug addiction, is that food is essential for life. It is unclear where the line between use and misuse of certain foods lies, making it difficult to advocate for any treatment involving abstinence or reduction of intake. In defense of the food addiction model, the YFAS asks respondents about their eating behavior with regard to "certain foods," rather than food in general [3]. Therefore, the fact that food (unlike addictive drugs) is consumed ubiquitously is in some ways irrelevant to the validity of the food addiction model. Indeed, a food addiction model does not propose that compulsive behavior pertains to all foods, but rather those high in fat, sugar, or salt, which pharmacodynamically act like addictive drugs in the brain (i.e.,

provoking acute increases in dopaminergic and opioidergic release, and other changes in neural activity). If food addiction is considered in terms of dependence on specific food types—for example, chocolate, sweets, fast-food—then distinguishing "use" from "misuse" is more feasible and useful.

With a strong focus on the overlap between neural processing of addictive drugs and palatable food, the relative *magnitude* of these responses is often overlooked; addictive drugs are typically more potent in evoking reward-related activity compared to food rewards [106, 107]. This does not preclude the possibility that individuals can become addicted to food in a similar way to drugs of abuse, but could lead to a different presentation of symptoms in each. For example, it is possible that symptoms such as withdrawal and tolerance are present but much less discernible in food addiction compared to drug addiction, simply because the neural responses to food are smaller than those to drugs. If this is the case, we might expect higher reward sensitivity to be associated with stronger signs of withdrawal and tolerance in an individual with food addiction.

Another point of discussion is whether it is meaningful to calculate a continuous symptom score in individuals who do not meet "diagnostic" criteria for food addiction [4]. In some cases, participants have been classified as being "high" or "low" in food addiction symptomology (e.g., [94, 99]), and behavioral or neural variables compared between the two groups. If we assume that food addiction and its classification using the DSM's criteria for substance use disorders is valid, then it could be argued that the only meaningful comparison is between individuals who meet criteria for food addiction and those who do not. That is, there is no theoretical basis on which to assume fundamental differences between individuals with different *subclinical* levels of food addiction. Furthermore, the number of symptoms present can vary considerably across samples, meaning that high and low food addiction can look very different in different studies. For example, the average symptom count of the "high food addiction" group in one neuroimaging study [94] was closer to the symptom count of the "low food addiction" than the high food addiction group in another study [99]. It is also not always clear how many of the individuals endorse significant impairment or distress associated with their symptoms; since this symptom is necessary to meet criteria for food addiction, it should be at least reported in any study using the term "addiction." To facilitate comparisons across studies, it would be useful to standardize the definitions of high and low food addiction, or to compare only those who endorse clinically significant symptoms. Given the varying symptoms of food addiction assessed by the YFAS, examining neural or behavioral correlates of specific symptoms might be most valuable in cases where not all symptoms are endorsed.

Behavioral or Chemical Addiction?

Although certain combinations of food nutrients and sensory properties are associated with reward-related neural activity, there has been debate over whether food addiction would be better conceptualized as "eating behaviour," putting it more akin to behavioral addictions such as gambling disorder [108, 109]. In favor of the "eating addiction" model, Hebebrand and colleagues state that there is little evidence of any one food or food component being addictive, and that individuals who overeat typically eat a wide range of different foods and have diverse food preferences [108]. However, Schulte and colleagues [109] argue that in order to be considered a behavioral addiction, the type of food eaten should have no effect on whether or not addictive-like eating develops—a feature that is refuted by findings that foods high in fat and sugar are most closely associated with addictive-like eating [109]. According to Schulte and colleagues, gambling disorder is characterized by compulsive engagement in a behavior (gambling) that in itself is rewarding. Although money (typically the object of gambling) is a rewarding stimulus, it is the behavior of gambling—with its intermittent rewards and inherent risk of losses—that becomes reinforcing [109].

It is possible that gambling increases the reward value of money [109]; in the same way, food might become more rewarding with certain patterns of intake (e.g., binge eating, stress-induced eating). However, an important difference between food and money is that food is considered a primary reinforcer (i.e., inherently rewarding), whereas money is a secondary reinforcer since it is conditioned to be rewarding (at least in many societies). Compulsive sexual behaviors might be more akin to addictive-like eating, since sex—like food—is considered a primary reinforcer. Indeed, compulsive sexual behavior (also referred to as "hypersexuality," "sex addiction," and "excessive sexual drive") has been compared to substance use and addictive disorders [110]. While more systematic studies are necessary, early findings demonstrate neurobiological similarities across addictions (both substance-based and non-substance-based), hypersexuality, and food addiction (Table 2).

Summary and Future Directions

At this point, the evidence for addictive properties of certain foods and overlap between compulsive eating behavior and substance use disorders is compelling. Preclinical and human studies alike demonstrate that foods high in fat and sugar elicit neural responses that are qualitatively similar to those associated with addictive drugs, suggesting that exposure to these foods could produce a cluster of symptoms similar to those seen in substance use disorders. Neuroimaging and pharmacological studies show that individuals who meet the YFAS

Table 2 Key criteria across substance use disorders, gambling disorder, hypersexuality, and food addiction and neurobiological similarities and differences across the four conditions in cue-reactivity studies, effect of opioid antagonists, and striatal activation and dopaminergic systems

	Substance use disorder	Gambling disorder	Hypersexuality	Food addiction
Key criteria				
Status in DSM/ICD	In DSM-5 [8] and ICD-11	In DSM-5 [8] and ICD-11	In ICD-11 as compulsive sexual behavior disorder	Not included in current versions of DSM or ICD
Compulsive, recurrent engagement in behavior	Substance taken in larger amount or for a longer period than intended; persistent desire and multiple unsuccessful attempts to decrease or discontinue use; great deal of time spent obtaining, using, or recovering from effects of substance use [8]	Repeated unsuccessful efforts to control, cut back, or stop gambling; preoccupation with gambling (e.g. persistent thoughts of reliving past gambling experiences, handicapping or planning the next venture, thinking of ways to get money with which to gamble; [8])	Persistent pattern of failure to control intense, repetitive sexual impulses or urges resulting in repetitive sexual behavior; Numerous unsuccessful efforts to significantly reduce repetitive sexual behavior	Eating more or for longer than intended (e.g., to the point of feeling sick); repeated, unsuccessful attempts to reduce or control food intake; considerable time spent thinking about food, eating, or recovering from effects (e.g., feeling tired or sluggish; [9])
Consequences of behavior (e.g., social, occupational, recreational)	Social, occupational, or recreational activities given up or reduced; continued use despite knowledge of persistent or recurrent physical or psychological consequences; continued use despite social or interpersonal problems; failure to fulfill major role obligations at work, school, or home [8]	Lying to conceal the extent of involvement with gambling; relationships, jobs, and other opportunities jeopardized or lost due to gambling; reliance on other people for money to relieve desperate financial situations caused by gambling [8]	Neglect of health and personal care or other interests, activities and responsibilities; Impairment in personal family, social, educational, occupational, or other areas of functioning	Avoiding work or social events due to fear of overeating; eating has negative effects on performance at work, or causes problems in social relationships [9]
Risky use/engagement	Recurrent substance use in situations in which it is physically hazardous; continued use despite potential negative consequences [8]	Persistent gambling despite negative social and financial problems [8]	Continued behavior despite adverse consequences (e.g., social problems, sexually transmitted diseases, unsafe environments)	Eating certain foods despite health risks; eating despite psychological and physical problems, and concerns of family and friends [9]
Tolerance	Markedly increased dose of substance required to achieve the desired effect, or markedly reduced effect when the usual dose is consumed [8]	Needs to gamble with increasing amounts of money in order to achieve the desired excitement [8]	Escalation of sexual activity as the disorder progresses; increasing amount of time spent seeking out potential partners and engaging in sexual activities [110]	More food needed to obtain same psychological or physical effects; eating the same amount of food does not have the same effect as it used to [9]
Withdrawal	Syndrome occurring when blood or tissue concentrations of a substance decline in an individual who had maintained prolonged heavy use of a substance; individual likely to consume the substance to relieve withdrawal symptoms [8]	Is restless or irritable when attempting to cut down or stop gambling [8]	Negative mood states (dysphoria, depression, guilt) when individual reduces sexual activities [110]	Cutting down or stopping intake causes negative psychological or physical effects (e.g., anxiety, irritability; headache or fatigue; craving for food; [9])
Neurobiological similarities and differences				
Neural circuitry associated with cue exposure	Heightened activity in areas including the ventral striatum, amygdala, anterior cingulate cortex, and prefrontal cortex in	Increased activity in medial prefrontal cortex and ventral striatum in response to gambling-related cues in	Heightened activity in ACC, ventral striatum, and amygdala during exposure to sexually explicit videos in individuals with compulsive sexual	Food addiction associated with increased activity in medial orbitofrontal cortex, ACC, and amygdala during anticipation of

Table 2 (continued)

	Substance use disorder	Gambling disorder	Hypersexuality	Food addiction
	response to drug cues in individuals with substance use disorders [111, 112]	individuals with gambling disorder [113, 114]	behavior; functional connectivity in these areas correlated with subjective desire [115]	palatable milkshake. Individuals high in food addiction show greater DLPFC activity during anticipatory processing (compared to those low in food addiction; [94])
Effect of opioid antagonists	FDA-approved treatment for alcohol use disorder. Reduces craving and euphoria associated with alcohol intake in individuals with alcohol use disorder [116]	Evidence of effectiveness in treating gambling disorder [117, 118]	Evidence of effectiveness in reducing sexual urges and compulsive sexual behaviors [119, 120]	Suppresses food intake and stress-induced hyperphagia in animals. Reduced binge size in females with bulimia nervosa. Reduces pleasantness ratings of glucose solutions (for a review, see [121]
Striatal activation and dopaminergic systems	Altered dopamine transmission [59] and activity in ventral striatum (e.g., [122, 123]) strongly associated with substance use disorders	Mixed findings on striatal responsivity to monetary cues in individuals with gambling disorder (e.g., [124, 125])	Striatal responses to subliminally-presented sexual stimuli enhanced by dopamine agonist and reduced by dopamine antagonist [126]. Side effect of L-dopa treatment for Parkinson's disease is hypersexuality	Decreased ventral striatal responses to monetary cues in individuals with BED [127]; alterations in dopamine transmission in BED [62] and food addiction [35, 104]

criteria for food addiction show similar patterns of resting and task-related brain activity and similar alterations in dopamine transmission as are seen in substance use disorders. Studies of clinical populations suggest that food addiction has detrimental effects on several aspects of physical and mental health, although causal relationships have not yet been established. To better understand the relationship between the core symptoms of food addiction and other health outcomes, longitudinal studies should be conducted to examine the development and course of food addiction in clinical and non-clinical populations. Given discrepant findings on the clinical implications of food addiction, another priority for this research area is to more closely examine the influence of specific symptoms of food addiction, and their interaction with other behavioral and physiological variables, on treatment outcomes.

Compliance with ethical standards

Conflict of interest KN and IB have no potential conflicts of interest to disclose; JM is a principal in BEAM Diagnostics, Inc.

Human and Animal Rights and Informed Consent This article does not contain any studies with human or animal studies performed by any of the authors.

References

1. Davis C. Compulsive overeating as an addictive behavior: overlap between food addiction and binge eating disorder. Curr Obes Rep. 2013;2(2):171–8.
2. Meule A. Focus: Addiction: back by popular demand: a narrative review on the history of food addiction research. Yale J Biol Med. 2015;88(3):295.
3. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. Appetite. 2009;52(2):430–6.
4. Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? Obes Rev. 2013;14(1):19–28.
5. Weiner B, White W. The journal of inebriety (1876–1914): history, topical analysis, and photographic images. Addiction. 2007;102(1):15–23.
6. Stoltz SG. Recovering from foodaholism. J Spec Gr Work. 1984;9(1):51–61.
7. Association AP, Association AP. Diagnostic and statistical manual of mental disorders (revised 4th ed). Washington, DC Author. 2000.
8. Association AP. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub; 2013.
9. Gearhardt AN, Corbin WR, Brownell KD. Development of the Yale Food Addiction Scale version 2.0. Psychol Addict Behav. 2016;30(1):113–21.
10. Brunault P, Ballon N, Gaillard P, Réveillère C, Courtois R. Validation of the French version of the Yale food addiction scale: an examination of its factor structure, reliability, and construct validity in a nonclinical sample. Can J Psychiatr. 2014;59(5):276–84.
11. Chen G, Tang Z, Guo G, Liu X, Xiao S. The Chinese version of the Yale food addiction scale: an examination of its validation in a sample of female adolescents. Eat Behav. 2015;18:97–102.

12. Meule A, Müller A, Gearhardt AN, Blechert J. German version of the Yale food addiction scale 2.0: prevalence and correlates of 'food addiction' in students and obese individuals. Appetite. 2017;115:54–61.
13. Flint AJ, Gearhardt AN, Corbin WR, Brownell KD, Field AE, Rimm EB. Food-addiction scale measurement in 2 cohorts of middle-aged and older women. Am J Clin Nutr. 2014;99(3): 578–86.
14. Schulte EM, Gearhardt AN. Development of the modified Yale food addiction scale version 2.0. Eur Eat Disord Rev. 2017;25(4): 302–8.
15. Gearhardt AN, Roberto CA, Seamans MJ, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale for children. Eat Behav. 2013;14(4):508–12.
16. Pursey KM, Stanwell P, Gearhardt AN, Collins CE, Burrows TL. The prevalence of food addiction as assessed by the Yale food addiction scale: a systematic review. Nutrients. 2014;6(10): 4552–90.
17. Brunault P, Ducluzeau P-H, Bourbao-Tournois C, Delbachian I, Couet C, Réveillère C, et al. Food addiction in bariatric surgery candidates: prevalence and risk factors. Obes Surg. 2016;26(7): 1650–3.
18. Holgerson AA, Clark MM, Ames GE, Collazo-Clavell ML, Kellogg TA, Graszer KM, et al. Association of adverse childhood experiences and food addiction to bariatric surgery completion and weight loss outcome. Obes Surg. 2018;28(11):3386–92.
19. Koball AM, Clark MM, Collazo-Clavell M, Kellogg T, Ames G, Ebbert J, et al. The relationship among food addiction, negative mood, and eating-disordered behaviors in patients seeking to have bariatric surgery. Surg Obes Relat Dis. 2016;12(1):165–70.
20. Ouellette A-S, Rodrigue C, Lemieux S, Tchernof A, Biertho L, Bégin C. An examination of the mechanisms and personality traits underlying food addiction among individuals with severe obesity awaiting bariatric surgery. Eat Weight Disord Anorexia, Bulim Obes. 2017;22(4):633–40.
21. Benzerouk F, Gierski F, Ducluzeau P-H, Bourbao-Tournois C, Gaubil-Kaladjian I, Bertin É, et al. Food addiction, in obese patients seeking bariatric surgery, is associated with higher prevalence of current mood and anxiety disorders and past mood disorders. Psychiatry Res. 2018;267:473–9.
22. Gearhardt AN, White MA, Masheb RM, Morgan PT, Crosby RD, Grilo CM. An examination of the food addiction construct in obese patients with binge eating disorder. Int J Eat Disord. 2012;45(5):657–63.
23. Gearhardt AN, White MA, Masheb RM, Grilo CM. An examination of food addiction in a racially diverse sample of obese patients with binge eating disorder in primary care settings. Compr Psychiatry. 2013;54(5):500–5.
24. Gearhardt AN, Boswell RG, White MA. The association of "food addiction" with disordered eating and body mass index. Eat Behav. 2014;15(3):427–33.
25. Ivezaj V, White MA, Grilo CM. Examining binge-eating disorder and food addiction in adults with overweight and obesity. Obesity. 2016;24(10):2064–9.
26. de Vries S, Meule A. Food addiction and bulimia nervosa: new data based on the Yale food addiction scale 2.0. Eur Eat Disord Rev. 2016;24(6):518–22.
27. Hilker I, Sánchez I, Steward T, Jiménez-Murcia S, Granero R, Gearhardt AN, et al. Food addiction in bulimia nervosa: clinical correlates and association with response to a brief psychoeducational intervention. Eur Eat Disord Rev. 2016;24(6): 482–8.
28. Meule A, von Rezori V, Blechert J. Food addiction and bulimia nervosa. Eur Eat Disord Rev. 2014;22(5):331–7.
29. Davis C. A commentary on the associations among 'food addiction', binge eating disorder, and obesity: overlapping conditions with idiosyncratic clinical features. Appetite. 2017;115:3–8.
30. Davis C, Curtis C, Levitan RD, Carter JC, Kaplan AS, Kennedy JL. Evidence that "food addiction" is a valid phenotype of obesity. Appetite. 2011;57(3):711–7.
31. Pedram P, Wadden D, Amini P, Gulliver W, Randell E, Cahill F, et al. Food addiction: its prevalence and significant association with obesity in the general population. PLoS One. 2013;8(9):1–6.
32. Meule A. Food addiction and body-mass-index: a non-linear relationship. Med Hypotheses. 2012;79(4):508–11.
33. Kessler RC, Adler L, Barkley R, Biederman J, Conners CK, Demler O, et al. The prevalence and correlates of adult ADHD in the United States: results from the National Comorbidity Survey Replication. Am J Psychiatry. 2006;163(4):716–23.
34. Bégin C, St-Louis ME, Turmel S, Tousignant B, Marion L-P, Ferland F, et al. Does food addiction distinguish a specific subgroup of overweight/obese overeating women. Health (Irvine Calif). 2012;4(12A):1492–9.
35. Davis C, Loxton NJ, Levitan RD, Kaplan AS, Carter JC, Kennedy JL. "Food addiction" and its association with a dopaminergic multilocus genetic profile. Physiol Behav. 2013;118:63–9.
36. Davis C, Levitan RD, Kaplan AS, Kennedy JL, Carter JC. Food cravings, appetite, and snack-food consumption in response to a psychomotor stimulant drug: the moderating effect of "food-addiction.". Front Psychol. 2014;5:403.
37. Chao AM, Shaw JA, Pearl RL, Alamuddin N, Hopkins CM, Bakizada ZM, et al. Prevalence and psychosocial correlates of food addiction in persons with obesity seeking weight reduction. Compr Psychiatry. 2017;73:97–104.
38. Burmeister JM, Hinman N, Koball A, Hoffmann DA, Carels RA. Food addiction in adults seeking weight loss treatment. Implications for psychosocial health and weight loss. Appetite. 2013;60:103–10.
39. Clark SM, Saules KK. Validation of the Yale food addiction scale among a weight-loss surgery population. Eat Behav. 2013;14(2): 216–9.
40. Miller-Matero LR, Bryce K, Saulino CK, Dykhuis KE, Genaw J, Carlin AM. Problematic eating behaviors predict outcomes after bariatric surgery. Obes Surg. 2018;28(7):1910–5.
41. Drewnowski A, Greenwood MRC. Cream and sugar: human preferences for high-fat foods. Physiol Behav. 1983;30(4):629–33.
42. Drewnowski A. Taste preferences and food intake. Annu Rev Nutr. 1997;17(1):237–53.
43. Johnson PM, Kenny PJ. Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. Nat Neurosci. 2010;13(5):635–41.
44. Oswald KD, Murdaugh DL, King VL, Boggiano MM. Motivation for palatable food despite consequences in an animal model of binge eating. Int J Eat Disord. 2011;44(3):203–11.
45. Saper CB, Chou TC, Elmquist JK. The need to feed: homeostatic and hedonic control of eating. Neuron. 2002;36(2):199–211.
46. Weingarten HP, Elston D. Food cravings in a college population. Appetite. 1991;17(3):167–75.
47. Richard A, Meule A, Reichenberger J, Blechert J. Food cravings in everyday life: an EMA study on snack-related thoughts, cravings, and consumption. Appetite. 2017;113:215–23.
48. Rozin P, Levine E, Stoess C. Chocolate craving and liking. Appetite. 1991;17(3):199–212.
49. Colantuoni C, Schwenker J, McCarthy J, Rada P, Ladenheim B, Cadet J-L, et al. Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain. Neuroreport. 2001;12(16): 3549–52.
50. Duarte RBM, Patrono E, Borges AC, César AAS, Tomaz C, Ventura R, et al. Consumption of a highly palatable food induces

a lasting place-conditioning memory in marmoset monkeys. Behav Process. 2014;107:163–6.
51. Lenoir M, Serre F, Cantin L, Ahmed SH. Intense sweetness surpasses cocaine reward. PLoS One. 2007;2(8):e698.
52. Volkow ND, Wang G, Fowler JS, Logan J, Jayne M, Franceschi D, et al. "Nonhedonic" food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. Synapse. 2002;44(3):175–80.
53. Hernandez L, Hoebel BG. Food reward and cocaine increase extracellular dopamine in the nucleus accumbens as measured by microdialysis. Life Sci. 1988;42(18):1705–12.
54. Small DM, Jones-Gotman M, Dagher A. Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. NeuroImage. 2003;19(4):1709–15.
55. Milella MS, Fotros A, Gravel P, Casey KF, Larcher K, Verhaeghe JAJ, et al. Cocaine cue-induced dopamine release in the human prefrontal cortex. J Psychiatry Neurosci. 2016;41:322–30.
56. Kenny PJ. Reward mechanisms in obesity: new insights and future directions. Neuron. 2011;69(4):664–79.
57. Volkow N, Wang GJ, Fowler JS, Tomasi D, Baler R. Food and drug reward: overlapping circuits in human obesity and addiction. In: Carter C, Dalley J. (eds) Brain Imaging Behav Neurosci. Springer; 2011;1–24.
58. Sharma S, Fernandes MF, Fulton S. Adaptations in brain reward circuitry underlie palatable food cravings and anxiety induced by high-fat diet withdrawal. Int J Obes. 2013;37(9):1183–91.
59. Volkow ND, Fowler JS, Wang G-J, Swanson JM. Dopamine in drug abuse and addiction: results from imaging studies and treatment implications. Mol Psychiatry. 2004;9(6):557–69.
60. de Weijer BA, van de Giessen E, van Amelsvoort TA, Boot E, Braak B, Janssen IM, et al. Lower striatal dopamine D 2/3 receptor availability in obese compared with non-obese subjects. EJNMMI Res. 2011;1(1):37.
61. Volkow ND, Wang G-J, Telang F, Fowler JS, Thanos PK, Logan J, et al. Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: possible contributing factors. NeuroImage. 2008;42(4):1537–43.
62. Wang G, Geliebter A, Volkow ND, Telang FW, Logan J, Jayne MC, et al. Enhanced striatal dopamine release during food stimulation in binge eating disorder. Obesity. 2011;19(8):1601–8.
63. Pérez-Ortiz JM, Galiana-Simal A, Salas E, González-Martín C, García-Rojo M, Alguacil LF. A high-fat diet combined with food deprivation increases food seeking and the expression of candidate biomarkers of addiction. Addict Biol. 2017;22(4):1002–9.
64. del Castillo C, Morales L, Alguacil LF, Salas E, Garrido E, Alonso E, et al. Proteomic analysis of the nucleus accumbens of rats with different vulnerability to cocaine addiction. Neuropharmacology. 2009;57(1):41–8.
65. Gordon EL, Ariel-Donges AH, Bauman V, Merlo LJ. What is the evidence for "food addiction?" a systematic review. Nutrients. 2018;10(4):477.
66. Brown RM, Kupchik YM, Spencer S, Garcia-Keller C, Spanswick DC, Lawrence AJ, et al. Addiction-like synaptic impairments in diet-induced obesity. Biol Psychiatry. 2017;81(9):797–806.
67. Furlong TM, Jayaweera HK, Balleine BW, Corbit LH. Binge-like consumption of a palatable food accelerates habitual control of behavior and is dependent on activation of the dorsolateral striatum. J Neurosci. 2014;34(14):5012–22.
68. Heyne A, Kiesselbach C, Sahún I, McDonald J, Gaiffi M, Dierssen M, et al. Research focus on compulsive behaviour in animals: an animal model of compulsive food-taking behaviour. Addict Biol. 2009;14(4):373–83.
69. Kekic M, McClelland J, Campbell I, Nestler S, Rubia K, David AS, et al. The effects of prefrontal cortex transcranial direct current stimulation (tDCS) on food craving and temporal discounting in women with frequent food cravings. Appetite. 2014;78:55–62.
70. Ng L, Davis C. Cravings and food consumption in binge eating disorder. Eat Behav. 2013;14(4):472–5.
71. Lent MR, Swencionis C. Addictive personality and maladaptive eating behaviors in adults seeking bariatric surgery. Eat Behav. 2012;13(1):67–70.
72. Gossop MR, Eysenck SBG. A further investigation into the personality of drug addicts in treatment. Br J Addict. 1980;75(3):305–11.
73. McKenna RA, Rollo ME, Skinner JA, Burrows TL. Food addiction support: website content analysis. JMIR Cardio. 2018;2(1):e10.
74. Davis C, Mackew L, Levitan RD, Kaplan AS, Carter JC, Kennedy JL. Binge eating disorder (BED) in relation to addictive behaviors and personality risk factors. Front Psychol. 2017;8:579.
75. MacKillop J, Weafer J, Gray JC, Oshri A, Palmer A, de Wit H. The latent structure of impulsivity: impulsive choice, impulsive action, and impulsive personality traits. Psychopharmacology. 2016;233(18):3361–70.
76. Murphy CM, Stojek MK, MacKillop J. Interrelationships among impulsive personality traits, food addiction, and body mass index. Appetite. 2014;73:45–50.
77. Wolz I, Granero R, Fernández-Aranda F. A comprehensive model of food addiction in patients with binge-eating symptomatology: the essential role of negative urgency. Compr Psychiatry. 2017;74:118–24.
78. VanderBroek-Stice L, Stojek MK, Beach SRH, MacKillop J. Multidimensional assessment of impulsivity in relation to obesity and food addiction. Appetite. 2017;112:59–68.
79. Coskunpinar A, Dir AL, Cyders MA. Multidimensionality in impulsivity and alcohol use: a meta-analysis using the UPPS model of impulsivity. Alcohol Clin Exp Res. 2013;37(9):1441–50.
80. Kale D, Stautz K, Cooper A. Impulsivity related personality traits and cigarette smoking in adults: a meta-analysis using the UPPS-P model of impulsivity and reward sensitivity. Drug Alcohol Depend. 2018;185:149–67.
81. MacLaren VV, Fugelsang JA, Harrigan KA, Dixon MJ. The personality of pathological gamblers: a meta-analysis. Clin Psychol Rev. 2011;31(6):1057–67.
82. Amlung M, Vedelago L, Acker J, Balodis I, MacKillop J. Steep delay discounting and addictive behavior: a meta-analysis of continuous associations. Addiction. 2017;112(1):51–62.
83. MacKillop J, Amlung MT, Few LR, Ray LA, Sweet LH, Munafò MR. Delayed reward discounting and addictive behavior: a meta-analysis. Psychopharmacology. 2011;216(3):305–21.
84. Jackson JNS, MacKillop J. Attention-deficit/hyperactivity disorder and monetary delay discounting: a meta-analysis of case-control studies. Biol Psychiatry Cogn Neurosci Neuroimaging. 2016;1(4):316–25.
85. Amlung M, Petker T, Jackson J, Balodis I, MacKillop J. Steep discounting of delayed monetary and food rewards in obesity: a meta-analysis. Psychol Med. 2016;46(11):2423–34.
86. Meng Z, Liu C, Yu C, Ma Y. Transcranial direct current stimulation of the frontal-parietal-temporal area attenuates smoking behavior. J Psychiatr Res. 2014;54:19–25.
87. Schmidt R, Lüthold P, Kittel R, Tetzlaff A, Hilbert A. Visual attentional bias for food in adolescents with binge-eating disorder. J Psychiatr Res. 2016;80:22–9.
88. Stojek MK, Fischer S, MacKillop J. Stress, cues, and eating behavior. Using drug addiction paradigms to understand motivation for food. Appetite. 2015;92:252–60.
89. Carter BL, Tiffany ST. Meta-analysis of cue-reactivity in addiction research. Addiction. 1999;94(3):327–40.

90. Striegel-Moore RH, Dohm F, Kraemer HC, Schreiber GB, Taylor CB, Daniels SR. Risk factors for binge-eating disorders: an exploratory study. Int J Eat Disord. 2007;40(6):481–7.
91. Sinha R. Chronic stress, drug use, and vulnerability to addiction. Ann N Y Acad Sci. 2008;1141(1):105–30.
92. Grilo CM, Masheb RM, Wilson GT. A comparison of different methods for assessing the features of eating disorders in patients with binge eating disorder. J Consult Clin Psychol. 2001;69(2):317–22.
93. Sinha R. How does stress increase risk of drug abuse and relapse? Psychopharmacology. 2001;158(4):343–59.
94. Gearhardt AN, Yokum S, Orr PT, Stice E, Corbin WR, Brownell KD. Neural correlates of food addiction. Arch Gen Psychiatry. 2011;68(8):808–16.
95. Wallis JD, Miller EK. Neuronal activity in primate dorsolateral and orbital prefrontal cortex during performance of a reward preference task. Eur J Neurosci. 2003;18(7):2069–81.
96. Killgore WDS, Weber M, Schwab ZJ, Kipman M, DelDonno SR, Webb CA, et al. Cortico-limbic responsiveness to high-calorie food images predicts weight status among women. Int J Obes. 2013;37(11):1435–42.
97. Schienle A, Schäfer A, Hermann A, Vaitl D. Binge-eating disorder: reward sensitivity and brain activation to images of food. Biol Psychiatry. 2009;65(8):654–61.
98. Small DM, Zatorre RJ, Dagher A, Evans AC, Jones-Gotman M. Changes in brain activity related to eating chocolate: from pleasure to aversion. Brain. 2001;124(9):1720–33.
99. De Ridder D, Manning P, Leong SL, Ross S, Sutherland W, Horwath C, et al. The brain, obesity and addiction: an EEG neuroimaging study. Sci Rep. 2016;6:34122.
100. Imperatori C, Fabbricatore M, Innamorati M, Farina B, Quintiliani MI, Lamis DA, et al. Modification of EEG functional connectivity and EEG power spectra in overweight and obese patients with food addiction: an eLORETA study. Brain Imaging Behav. 2015;9(4):703–16.
101. Coullaut-Valera R, Arbaiza I, Bajo R, Arrúe R, López ME, Coullaut-Valera J, et al. Drug polyconsumption is associated with increased synchronization of brain electrical-activity at rest and in a counting task. Int J Neural Syst. 2014;24(01):1450005.
102. Franken IHA, Stam CJ, Hendriks VM, van den Brink W. Electroencephalographic power and coherence analyses suggest altered brain function in abstinent male heroin-dependent patients. Neuropsychobiology. 2004;49(2):105–10.
103. Ewing SWF, Claus ED, Hudson KA, Filbey FM, Jimenez EY, Lisdahl KM, et al. Overweight adolescents' brain response to sweetened beverages mirrors addiction pathways. Brain Imaging Behav. 2017;11(4):925–35.
104. Davis C, Fattore L, Kaplan AS, Carter JC, Levitan RD, Kennedy JL. The suppression of appetite and food consumption by methylphenidate: the moderating effects of gender and weight status in healthy adults. Int J Neuropsychopharmacol. 2012;15(2):181–7.
105. Leddy JJ, Epstein LH, Jaroni JL, Roemmich JN, Paluch RA, Goldfield GS, et al. Influence of methylphenidate on eating in obese men. Obes Res. 2004;12(2):224–32.
106. Deadwyler SA. Electrophysiological correlates of abused drugs: relation to natural rewards. Ann N Y Acad Sci. 2010;1187(1):140–7.
107. Opris I, Hampson RE, Deadwyler SA. The encoding of cocaine vs. natural rewards in the striatum of nonhuman primates: categories with different activations. Neuroscience. 2009;163(1):40–54.
108. Hebebrand J, Albayrak Ö, Adan R, Antel J, Dieguez C, de Jong J, et al. "Eating addiction", rather than "food addiction", better captures addictive-like eating behavior. Neurosci Biobehav Rev. 2014;47:295–306.
109. Schulte EM, Potenza MN, Gearhardt AN. A commentary on the "eating addiction" versus "food addiction" perspectives on addictive-like food consumption. Appetite. 2017;115:9–15.
110. Garcia FD, Thibaut F. Sexual addictions. Am J Drug Alcohol Abuse. 2010;36(5):254–60.
111. Courtney KE, Schacht JP, Hutchison K, Roche DJO, Ray LA. Neural substrates of cue reactivity: association with treatment outcomes and relapse. Addict Biol. 2016;21(1):3–22.
112. Jasinska AJ, Stein EA, Kaiser J, Naumer MJ, Yalachkov Y. Factors modulating neural reactivity to drug cues in addiction: a survey of human neuroimaging studies. Neurosci Biobehav Rev. 2014;38:1–16.
113. Clark L, Boileau I, Zack M. Neuroimaging of reward mechanisms in gambling disorder: an integrative review. Mol Psychiatry. Nature Publishing Group. 2018;1.
114. Van Holst RJ, van den Brink W, Veltman DJ, Goudriaan AE. Brain imaging studies in pathological gambling. Curr Psychiatry Rep. 2010;12(5):418–25.
115. Voon V, Mole TB, Banca P, Porter L, Morris L, Mitchell S, et al. Neural correlates of sexual cue reactivity in individuals with and without compulsive sexual behaviours. PLoS One. 2014;9(7):e102419.
116. Volpicelli JR, Alterman AI, Hayashida M, O'Brien CP. Naltrexone in the treatment of alcohol dependence. Arch Gen Psychiatry. 1992;49(11):876–80.
117. Kim SW, Grant JE, Adson DE, Shin YC. Double-blind naltrexone and placebo comparison study in the treatment of pathological gambling. Biol Psychiatry. 2001;49(11):914–21.
118. Ward S, Smith N, Bowden-Jones H. The use of naltrexone in pathological and problem gambling: a UK case series. J Behav Addict. 2018;7(3):827–33.
119. Bostwick JM, Bucci JA. Internet sex addiction treated with naltrexone. In: Mayo Clinic proceedings. Elsevier; 2008. pp 226–30.
120. Kraus SW, Meshberg-Cohen S, Martino S, Quinones LJ, Potenza MN. Treatment of compulsive pornography use with naltrexone: a case report. Am J Psychiatry. 2015;172(12):1260–1.
121. Drewnowski A, Krahn DD, Demitrack MA, Nairn K, Gosnell BA. Taste responses and preferences for sweet high-fat foods: evidence for opioid involvement. Physiol Behav. 1992;51(2):371–9.
122. Wrase J, Schlagenhauf F, Kienast T, Wüstenberg T, Bermpohl F, Kahnt T, et al. Dysfunction of reward processing correlates with alcohol craving in detoxified alcoholics. NeuroImage. 2007;35(2):787–94.
123. Peters J, Bromberg U, Schneider S, Brassen S, Menz M, Banaschewski T, et al. Lower ventral striatal activation during reward anticipation in adolescent smokers. Am J Psychiatry. 2011;168(5):540–9.
124. Balodis IM, Kober H, Worhunsky PD, Stevens MC, Pearlson GD, Potenza MN. Diminished frontostriatal activity during processing of monetary rewards and losses in pathological gambling. Biol Psychiatry. 2012;71(8):749–57.
125. van Holst RJ, Veltman DJ, Büchel C, van den Brink W, Goudriaan AE. Distorted expectancy coding in problem gambling: is the addictive in the anticipation? Biol Psychiatry. 2012;71(8):741–8.
126. Oei NYL, Rombouts SARB, Soeter RP, Van Gerven JM, Both S. Dopamine modulates reward system activity during subconscious processing of sexual stimuli. Neuropsychopharmacology. 2012;37(7):1729–37.
127. Balodis IM, Kober H, Worhunsky PD, White MA, Stevens MC, Pearlson GD, et al. Monetary reward processing in obese individuals with and without binge eating disorder. Biol Psychiatry. 2013;73(9):877–86.