Pharmacological Research 224 (2026) 108097

Contents lists available at ScienceDirect

Pharmacological Research

journal homepage: www.elsevier.com/locate/yphrs

The addicted brain: How processed foods hijack reward pathways

Kaylee Hough [a], Marzia Friuli [b], Nicole M. Avena [a,*], Adele Romano [c]

[a] Icahn School of Medicine at Mount Sinai, New York, NY, United States
[b] Department of Life Sciences, Health and Health Professions, Link Campus University, Rome, Italy
[c] Department of Physiology and Pharmacology "V. Erspamer", Sapienza University of Rome, Italy

ARTICLE INFO

Keywords:
Food addiction
Sugar addiction
Dopamine
Mesolimbic reward system
Substance abuse

ABSTRACT

The concept of addiction, traditionally confined to substances such as drugs and alcohol, has expanded to encompass behavioral patterns such as compulsive eating. Emerging evidence suggests that ultra-processed foods (UPFs), particularly those high in refined sugars and saturated fats, may elicit neurobiological responses akin to those observed in substance use disorders. This review explores the hypothesis that food addiction shares common clinical and neurochemical mechanisms with traditional forms of addiction, drawing from DSM-5 diagnostic criteria and recent findings in neuropharmacology. Animal and human studies have demonstrated that excessive consumption of palatable foods can induce behaviors characteristic of addiction—bingeing, craving, tolerance, and withdrawal—accompanied by significant dopaminergic alterations within the mesolimbic reward circuitry. Neuroimaging and molecular studies further reveal that chronic overconsumption of UPFs alters dopaminergic tone, disrupts prefrontal control, and activates stress pathways, thereby reinforcing compulsive intake. The Yale Food Addiction Scale (YFAS) and its pediatric adaptations provide structured tools for identifying food addiction phenotypes in clinical and research settings. Moreover, parallels between binge eating disorder and substance dependence highlight overlapping neurobehavioral mechanisms. As the obesity epidemic intensifies, particularly among populations with limited access to nutritious foods, understanding the pharmacological underpinnings of food addiction becomes critical. This review underscores the need to reframe UPFs as potentially addictive agents and calls for integrative therapeutic strategies and policy-driven reforms aimed at mitigating their impact on public health.

1. Defining addiction: clinical and diagnostic frameworks

In clinical applications, there is a lack of consensus on the formal definition of addiction. The American Society of Addiction Medicine (ASAM) defines addiction as a "chronic medical disease involving complex interactions among brain, circuits, genetics, the environment, and the individual's life experiences" [1]. Addiction arises when a person becomes mentally and/or physically reliant on or compelled to consume a certain substance [2].

The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) serves as a key reference for healthcare professionals in the identification and understanding of mental health conditions and their associated complications [3].

To address substance use disorder, the DSM-5 outlines comprehensive diagnostic criteria that help differentiate between various forms of substance-related problems. Notably, the criteria were revised to more clearly distinguish between substance abuse and dependence, addressing the concerns from the earlier classification systems that overly emphasized severity [4].

The DSM-5 defines eleven criteria for diagnosing substance use disorder, all grounded in evidence-based clinical research [3]. These criteria are organized into four main categories (see Fig. 1) and specifically assess substance use behavior within the previous 12 months.

The DSM-5 criteria provide an informative foundation of the characteristics of substance use disorder and how healthcare practitioners can clinically diagnose it. The DSM-5 categorizes various classes of substances, including alcohol, caffeine, cannabis, hallucinogens, inhalants, opioids, sedatives, hypnotics or anxiolytics, stimulants, tobacco, and others (including unknown substances) [3].

Historically, addiction has been primarily associated with drugs and alcohol; however, scientific perspectives on addiction have evolved, expanding beyond substances to include certain compulsive behaviors [5]. These behavioral (or process) addictions have gained recognition from the American Psychological Association (APA), with gambling

* Corresponding author.
E-mail address: nicole.avena@mssm.edu (N.M. Avena).

https://doi.org/10.1016/j.phrs.2026.108097
Received 11 July 2025; Received in revised form 7 December 2025; Accepted 9 January 2026
Available online 10 January 2026

disorder being the first non-substance-related addiction officially included in the DSM-5 in 2013 [6].

As research in this field advances, growing evidence suggests that compulsive behaviors—beyond the use of drugs or alcohol—can meet the diagnostic criteria within the DSM-5 for addiction. Although food is not currently listed as an addictive substance in the DSM-5, studies over the past two decades indicate that highly palatable, ultra-processed foods (UPFs) may trigger neurobiological responses similar to those induced by addictive substances like drugs and alcohol [6]. Importantly, food is a biological necessity sustaining metabolic homeostasis, and its intake is fundamentally regulated by hunger signals, nutrient sensing, and gut-brain communication. This homeostatic regulation contrasts with the consumption of ultra-processed, hyper-palatable products that are specifically designed to enhance hedonic value. These formulations combine energy-dense macronutrient profiles with additives and sensory cues that disproportionately activate central pathways, increasing incentive salience and promoting intake beyond physiological requirements [7]. Thus, when referring to food-related addictive-like behaviors in this review, the focus is on these engineered high-reward foods rather than food as an essential substrate for survival. Technological advancements have accelerated the development and widespread availability of these products, transforming the modern food supply. As UPFs have become increasingly accessible worldwide, the role of eating appears to have shifted from sustaining survival to fostering patterns of overconsumption and compulsive eating.

2. Early preclinical evidence and the development of diagnostic tools for food addiction

Evidence of food addiction dates back to the early 2000s with various publications focused on the impact of excess sugar intake in rats. One study in 2006 looked at the bingeing behavior of Sprague-Dawley rats, demonstrating their dependence on sugar. The study also noted that whenever sugar was removed from the rat's diet, they exhibited signs of withdrawal—similar to the clinical manifestations of withdrawal in drug and alcohol addiction [8]. Examining the core components of addiction—bingeing, withdrawal, and craving—a study conducted in 2008 provided a comprehensive review of the existing literature on sugar as an addictive substance in animal models. The review concluded that, under certain conditions, Sprague-Dawley rats can develop a dependency on sugar, a phenomenon that may offer insights into human conditions such as eating disorders and obesity [9]. One particular study highlighted that repeated, excessive sugar consumption can induce neurobiological and behavioral changes that closely resemble the effects of substance abuse. Whenever Sprague-Dawley rats binged on a 10 % sucrose solution repeatedly, dopamine was released in the nucleus accumbens (NAc). This finding suggested that repeated bingeing on sugar releases opioids, which leads to the brain compensating by expressing less of these opioid peptides in certain regions. Whenever the rats in this study were deprived of food, they displayed anxiety and depression, contributing to withdrawal-like behaviors upon the cessation of sugar availability. These findings suggest that, in specific contexts like bingeing or excessive overconsumption, sugar has the potential to be addictive [10]. Another study conducted in Sprague-Dawley rats aimed to investigate whether sugar-dependent rats would exhibit increased consumption of unsweetened ethanol, and conversely, whether access to ethanol would escalate sugar intake [11]. Remarkably, the results revealed that the rats who were dependent on sugar consumed the greatest amounts of ethanol, thereby supporting the hypothesis that sugar dependence can influence an animal's propensity to ingest ethanol [11]. The authors suggested that these findings may offer valuable insights into the comorbidities often observed between binge-eating disorders and alcohol consumption [11].

While a large body of existing literature on food addiction explores the potential addictive nature of food within animal models, these studies provide an insightful foundation for understanding potential parallels in humans. As evidence of food addiction continues to materialize and the body of research develops, scientists have begun to explore ways to quantify these eating behaviors. The Yale Food Addiction Scale (YFAS) was initially developed to provide a validated tool for identifying individuals who exhibit addiction-like eating behaviors, particularly in response to highly palatable foods rich in sugar and fat [12]. It was the first instrument specifically designed to translate the DSM-4 substance dependence criteria into the context of food consumption [12]. To extend its applicability to younger populations, a child version (YFAS-C) was introduced, using simplified language and age-appropriate examples while preserving the structure and scoring logic of the original tool [13]. Following the release of the DSM-5, the YFAS 2.0 was introduced to incorporate the updated eleven diagnostic criteria for substance use disorders, enhancing diagnostic alignment and

Categories of Substance Use Disorder Symptoms

Category	Description
Impaired Control	- Using more of a substance more often than intended - Wanting to cut down or stop using, but not being able - Spending a significant amount of time obtaining, using, or recovering from the effects of the substance - Cravings and urges to use the substance
Social Problems	- Neglecting responsibilities and relationships - Giving up activities they used to care about because of their substance - Inability to complete tasks at home, school, or work
Risky Use	- Using in risky settings - Continued use despite known problems
Physical Dependence	- Needing more of a substance to get the same effect (tolerance) - Having withdrawal symptoms when a substance is not used

Fig. 1. The four DSM-5 categories of substance use disorder. Adapted from the Alcohol, Drug, Addiction, and Mental Health Services.

psychometric performance [13]. A corresponding pediatric version, the YFAS-C 2.0, was also created and validated, offering a comprehensive and developmentally appropriate instrument for assessing clinically significant food addiction in adolescents [14]. The YFAS includes both a symptom count and a dichotomous diagnostic classification, allowing for the assessment of both the presence and severity of food addiction. Three or more symptoms recorded on the scale, combined with clinically significant impairment or distress, are utilized to assign a food addiction diagnosis [13].

3. Neurocircuitry of addiction: from reward to compulsion

Addiction, commonly referred to as substance use disorder, is a persistent and recurring condition characterized by compulsive, self-directed utilization of a substance [15,16]. Addiction is considered a chronic relapsing disorder that presents with specific neuroadaptations [16,17]. Early research on addiction focused on the rewarding effects of drug abuse [15]. Modern discussions of addiction have shifted to studying how long-term substance use alters the brain in a way that lowers the threshold for relapse [17]. Addiction has traditionally been viewed solely through the lens of alcohol and drug abuse; however, as clinical research has advanced, our understanding of addiction has broadened, revealing that a wide range of substances and behaviors—beyond just drugs and alcohol—can also possess addictive potential.

Whenever a substance is used, it can interfere with the brain's communication system. Drugs of abuse either influence the brains reward pathway by impacting dopamine or altering the way in which the brain sends, receives, and processes communication [18,19]. Chronic substance use can result in long-term changes in the neurotransmitter systems, such as those involving dopamine, GABA, and glutamate [15]. This adjustment within the brain can disrupt communication between areas involved in judgment, emotional balance, and self-control—particularly the prefrontal cortex [15]. With this, it can become difficult for individuals to think clearly, manage impulses, and understand the depth of their behaviors around addiction [15]. Neuroimaging research in individuals struggling with addiction consistently demonstrates decreased activity in the prefrontal cortex, reinforcing the idea that addiction impacts higher-order cognitive functioning [15].

With chronic substance use, natural rewards become less pleasurable, and the stress response can become heightened [15,20]. At the same time, regions of the brain that are involved in stress and emotion—like the extended amygdala—become more reactive, leading to heightened feelings of stress or discomfort during withdrawal [15,20]. These changes can make even minor stressors or reminders of past drug use powerful triggers for relapse. Addiction disrupts the prefrontal cortex, the area of the brain responsible for judgment, impulse control, and decision-making [15,19]. As a result, even mild stress or exposure to drug-related cues can quickly reignite drug-seeking behavior [15,20].

Compulsive behaviors are linked to the dysfunction of the brain regions that involve reward and emotional processing as well as inhibitory control [21]. Repetitive, chronic compulsions occur whenever a behavior that was originally goal-oriented transitions into an impulsive habit [22]. The basal ganglia include the NAc and the dorsolateral striatum (DLS). The NAc is well known for its role in reward and reinforcement, while the DLS plays a role in habit formation [22]. Repeated stimulation of the dopaminergic system in the NAc by a stimulus (drugs, alcohol, or highly palatable, ultra-processed food items) shifts signaling to the dorso-striatal dopaminergic pathways—resulting in habit formation [23].

Whenever a stimulus is repeatedly engaged with to relieve negative feelings or emotions, such as stress or anxiety, it is thought to cause neuroadaptations that result in the desensitization of the mesolimbic dopaminergic system [22]. Overtime, habitual use of a substance to combat an emotional state can lead to withdrawal-induced side effects. In situations where an individual loses control over their ability to engage with a substance, neuroadaptations of dopaminergic signaling are thought to underlie the loss of control [22].

Together, these mechanisms help explain how repeated exposure to highly palatable, ultra-processed foods may engage addiction-related neurocircuitry—shifting neural signaling from reward-driven to habit-driven pathways, reducing top-down inhibitory control, and promoting compulsive patterns of intake that parallel those observed in substance use disorders.

4. Dopamine signaling and reward dysregulation in addictive behaviors

The mesolimbic dopaminergic (ML-DA) system is the primary neural circuit in the body that plays a role in motivation, reward-centered behaviors, and behavior reinforcement. The ML-DA system is most widely known for its role in the reward pathway. Electric brain stimulation studies have conveyed that the ML-DA is activated in states of positive reward as well as behaviors that are appetite-motivated. Copious hypotheses exist that recognize different interpretations of the ML-DA functions [24]. The ML-DA system is characterized by VTA dopaminergic neurons that project to various brain areas, including the NAc and other limbic structures [25]. Dopamine neurons typically fire in a steady, low-frequency mode whenever they are at rest; however, whenever they have exposure to a stimulant, dopamine neurons fire with high frequency—producing high extracellular dopamine concentrations.

Whenever the ML-DA system is activated, it generates a physiological reaction that is both rewarding and reinforcing [25]. The structure of the ML-DA system plays a central role in signaling to the brain how rewarding a behavior may be. Dopamine is a neurotransmitter and hormone that is widely known as the "feel-good" neurotransmitter due to its role in connecting pleasurable feelings to behaviors. Dopamine has proven in countless studies to be involved in the hedonic component of reward [26]. Reward initiates an increase in dopamine activity within the brain. Whenever humans engage in healthy rewards, such as listening to music, interacting with loved ones, or enjoying a home-cooked meal, small bursts of neurotransmitters are released, resulting in pleasurable feelings to reinforce those behaviors. This neurochemical reinforcement helps the brain identify such behaviors as valuable and promotes their repetition, thereby contributing to habit formation. In contrast, the use of drugs and alcohol trigger abnormally high dopamine release, producing exaggerated sensations of pleasure [18]. Complications, including the development of addiction, arise when substances produce intense feelings of elation due to large bursts of dopamine release—further reinforcing maladaptive behaviors [16].

5. Stages of the addiction cycle: binge, withdrawal, and anticipation

Evidence supports the idea that addiction to a substance involves a three-stage cycle: binge/intoxication, withdrawal/negative affect, and preoccupation/anticipation [27]. As the cycle of addiction progresses and continues, it can increase in severity and leave those affected feeling as if they lack control of their behaviors [27]. The first stage of the addiction cycle, binge/intoxication, occurs whenever a person experiences the rewarding side effects of consuming a substance—such as euphoria [28]. Whenever these behaviors are repetitive and the basal ganglia's reward system—including NAc shell and core—is continually activated, the behavior of using a substance is reinforced [29]. The basal ganglia have a role in motivation and habit formation, and when it is continually activated, it has the ability to change behaviors in response to stimuli. In this stage, the overactivation of the basal ganglia can contribute to compulsive behaviors regarding the substance—leading to bingeing [28].

The second stage, withdrawal/negative affect, occurs next in the cycle. The extended amygdala may serve as a crucial neuroanatomical substrate that integrates brain arousal and stress systems with hedonic processing circuits. This integration contributes to the generation of negative emotional states that underlie negative reinforcement mechanisms driving the development of addiction [29]. Whenever use of a substance abruptly ceases, undesirable side effects and symptoms occur—contributing to withdrawal [28]. Side effects likely to occur within this stage include pain, dysphoria, irritability, anxiety, disturbed sleep, and a change in emotions. When substance use ceases, the brain's reward systems are no longer continually activated as they were before, leading to a reward deficit [28].

The third and final stage in the addiction cycle involves preoccupation/anticipation. This stage is characterized by impulsive behaviors, intense cravings, and impairments to executive function and involves different neurocircuits depending on the trigger (e.g., drugs, cues, and stressors) [29]. This stage in the cycle can result in an individual becoming hyper-focused on a substance following a period of restraint or avoidance of the substance. Chronic substance use impairs cognitive functions linked to the medial and orbital prefrontal cortices and the hippocampus, further increasing relapse vulnerability [29]. From this stage, the cycle then repeats itself once the individual chooses to again indulge in the substance.

6. Ultra-processed foods: classification, composition, and public health implications

When food is processed, it undergoes changes that alter it from its natural form. Processed foods can range from relatively simple items like hummus, spice mixes, and dark chocolate to more heavily altered products that may harm our health. Although there is no universally agreed-upon definition of ultra-processed foods, they can be defined as industrial products that are derived from foods or created from other sources. UPFs contain minimal whole foods and are generally ready-to-eat food choices, such as packaged chips, cookies, donuts, and frozen pizza—items that are often consumed daily [30]. Children, in particular, are consuming UPFs at increasingly high rates, often replacing more nutritious choices like whole grains, fruits, and vegetables. When UPFs are eaten frequently and in large quantities, they can lead to serious health issues over time, including obesity, compulsive eating behaviors, and chronic diseases.

UPFs can be diverse in their formulation, which can make it difficult for scientists to organize these foods into distinct categories. The NOVA system of food classification was generated based on the “nature, extent, and purpose of food processing.” [30] NOVA categorizes all foods and food products into four distinct groups. The first group is the unprocessed or minimally processed foods, which include foods that are naturally occurring, such as seeds, fruits, roots, eggs, and milk. The group two classification consists of processed culinary ingredients, such as oil, butter, sugar, and salt. Processed foods make up group three, which include canned goods, cheeses, and freshly baked breads. According to NOVA, most of the foods within the “processed” category consist of two to three ingredients and are modified versions of the food’s natural state. The fourth and final group within the NOVA classification system is the UPF group, which include soft drinks, packaged snacks, and frozen meals. NOVA highlights that various processes are utilized along with many ingredients in order to create UPF items. The NOVA classification system can be utilized to assess the impact of UPF on human health as well as act as a tool to identify and address public health concerns [30].

7. When ultra-processed foods become addictive: neurobiological and behavioral evidence

Addiction was once considered a term relevant only to drug and alcohol use; however, findings suggest that food can be considered a substance and prompt addiction in specific situations. The food items that are most likely to result in addictive properties include UPFs that are high in added sugar and saturated fat. The idea of food functioning as an addictive substance remains controversial; however, a growing body of evidence suggests that certain foods—such as sugar—may possess addictive qualities. One study compared the brain chemistry of Sprague-Dawley rats in two groups—one group had intermittent, excessive sugar intake, while the other group was provided with a standard diet without excess sugar. Particularly, rats were exposed to a 25 % glucose solution under a 12-hour access / 12-hour deprivation schedule for 30 days, leading to escalating intake concentrated in the first hour of access. The findings of the study suggested that exposure to a diet rich in highly palatable foods can result in an increased activation of dopamine in the brain [31]. Specifically, dopamine D1 receptor binding increased in the nucleus accumbens core and shell, D2 receptor binding decreased in the dorsal striatum, dopamine transporter binding increased in the midbrain, and μ-opioid receptor binding increased in the accumbens shell, cingulate cortex, hippocampus, and locus coeruleus.

Subsequent work using related intermittent-access paradigms demonstrated behavioral signatures consistent with addiction-like responses to sugar. In one study, female rats received daily limited access to sugar followed by periods of deprivation, leading to increased operant responding for sucrose following abstinence, a phenomenon referred to as the “sugar deprivation effect,” indicating heightened motivation and craving after withdrawal [32]. Related experiments further showed behavioral cross-sensitization to psychostimulants: female rats with a history of intermittent sucrose access exhibited increased locomotor activation in response to a low dose of amphetamine compared to continuous-access or chow-only controls, suggesting long-lasting sensitization of dopaminergic pathways [33]. Together, these findings indicate that intermittent sugar exposure induces both increased motivation for sucrose and enhanced responsiveness to drugs of abuse, supporting a convergence between reward pathways engaged by palatable foods and those activated by addictive substances. Another study focused on food addiction in the human population by including one hundred and twenty adults (82 women and 38 men) between the ages of 25 and 47 years. The study found that individuals who met the Yale Food Addiction Scale (YFAS) for clinically significant food addiction experienced a higher multi-locus genetic profile score associated with increased dopamine signaling [34]. In this study, the relation between the multi-locus genetic profile scores and food addiction was driven by reward-based eating [34]. This association suggests that genetic predispositions modulating dopaminergic function may contribute to vulnerability to addictive-like eating [34].

A well-known hallmark of drug and alcohol addiction is the ability of a substance to promote extracellular increases of dopamine in the brain. As discussed in the studies aforementioned, growing evidence suggests that consuming highly palatable UPFs containing sugar triggers a similar neurobiological response in the brain—specifically, a surge in dopamine release—which may contribute to the addictive nature of these foods [32]. However, food is not presently recognized by the DSM-5.

To establish substance use disorder, the DSM-5 utilizes an 11-item questionnaire that measures the degree to which the diagnostic criteria are met. The questions included in the DSM-5 are highlighted in Table 1. The severity coding for the criteria includes 1) Mild; 2–3 symptoms endorsed, 2) Moderate; 4–5 symptoms endorsed, and 3) Severe; 6 or more symptoms endorsed [35].

Substituting the terms “drug” and “alcohol” in the DSM-5 questionnaire with “ultra-processed foods (UPFs)” or “sugar” illustrates that the diagnostic criteria outlined in the DSM-5 can also apply to the excessive consumption of certain food items. While the DSM-5 does not officially classify food as an addictive substance, emerging research and its alignment with the existing diagnostic criteria indicate that food may have the potential to be addictive and could lead to dependence in certain individuals.

Table 1
DSM-5 criteria for substance use disorder.

1	In the past 12 months, I often used alcohol or drugs in large amounts over longer periods of time than I intended
2	In the past 12 months, I often wanted or tried to cut down or control my alcohol or drug use
3	In the past 12 months, I spent a lot of time either (a) using alcohol or drugs, (b) in activities trying to obtain alcohol or drugs, or (c) recovering from the effects of my drinking or drug use
4	In the past 12 months, I gave up or reduced by involvement in important social, occupational, or recreational activities because of my alcohol or drug use
5	In the past 12 months, I continued to use alcohol or drugs despite knowing that it likely caused or made worse psychological or physical problems I had (for example, continued drinking or drug use knowing it was making my ulcer or depression worse)
6	In the past 12 months, I found I needed greater amounts of alcohol or drugs than I use to in order to feel intoxicated or get a desired effect OR I got much less of an effect by using the same amount of alcohol or drugs in the past
7	In the past 12 months, I experienced withdrawal symptoms when I tried to cut down or stop my drinking or drug use OR I drank alcohol or used drugs to relieve or avoid withdrawal symptoms
8	In the past 12 months, my continued alcohol or drug use resulted in my not fulfilling major obligations at work, school, or home (for example, repeated absences of poor performances at work or school; neglecting my children or home)
9	In the past 12 months, I have repeatedly used alcohol or drugs in situations that were physically hazardous (for example, driving a car or operating machinery)
10	In the past 12 months, I have experienced strong desires, urges, or cravings to use alcohol or drugs
11	In the past 12 months, I continued to use alcohol or drugs despite having persistent or recurrent social or interpersonal problems caused or made worse by the effects of my drinking or drug use (e.g., arguments with friends or family about my drinking or drug use or physical fights)

8. Binge eating disorder and food addiction: shared mechanisms and distinct features

Bingeing occurs whenever an individual undergoes a period of uncontrolled eating behaviors. For individuals who experience continual patterns of bingeing behaviors, binge-eating disorder (BED) is a recognized clinical diagnosis. BED is characterized by bingeing episodes that occur regularly where those impacted experience a loss of control over their behaviors [36]. In 2013, binge-eating disorder was recognized by the DSM-5. Unlike binge eating disorders, the concept of food addiction has not been formally recognized as a clinical eating pathology or psychiatric condition. However, food addiction shares behavioral and psychological features with medically defined eating disorders.

In bingeing episodes, individuals typically indulge in ultra-processed, highly palatable food items. Binge eating disorder and food addiction share overlapping neurobiological substrates, particularly in brain regions involved in reward processing, impulse control, and homeostatic regulation. Both conditions are characterized by dysregulation in the mesolimbic dopamine system, including heightened activity in the NAc and altered signaling in the prefrontal cortex, which may contribute to impaired inhibitory control and compulsive eating behaviors [37,38]. In both addiction and binge-eating disorder, those impacted experience diminished control over their eating behaviors and repetitive, excessive intake of highly palatable foods. In both cases, individuals persist in these behaviors even when faced with potentially harmful consequences [39]. Functional imaging studies in both male and female subjects have shown that individuals with BED and those exhibiting food addiction symptoms demonstrate increased neural responsivity to palatable food cues in areas such as the orbitofrontal cortex and amygdala, suggesting a shared vulnerability to reward-driven eating [40,41]. These findings support a dimensional view of disordered eating, wherein BED and food addiction may represent overlapping phenotypes with common neurocognitive impairments.

In both binge eating behaviors and substance abuse, similar patterns of increased dopamine activity are evident [39]. Dopamine plays a critical role in the neurobiological mechanisms influencing food cravings, decision-making, executive function, and impulsivity, all factors associated with binge eating behaviors [42]. A review looking at 31 studies (25 human and 6 animal) found that in most of the studies, eating was associated with either hyperdopaminergic or hypodopaminergic states, with evidence pointing to increased dopamine release in response to food cues in binge eaters, particularly within the dorsal and ventral striatum [42]. These findings align with data from food addiction models, where similar patterns of dopaminergic activation have been observed in response to highly palatable foods [39]. This overlap supports the conceptualization of binge eating disorder and food addiction as distinct but neurobiologically and behaviorally overlapping conditions. Due to patterns such as compulsive eating behaviors, overindulgence, persistence in harmful behaviors, and diminished self-control, there appears to be a potential link between food addiction and binge-eating disorder [39]. However, further research is necessary to clarify the nature and extent of this relationship.

9. Food addiction, obesity, and dopaminergic dysregulation

Obesity is a public health issue and epidemic occurring within the United States in more than 2 in 5 adults. According to the CDC, in the United States, obesity accounts for $173 billion in medical expenditures in 2019 dollars [43]. Despite various contributing factors to the development of obesity, overindulgence in food is one of the leading causes of obesity in the United States [44]. The addiction to food, specifically carbohydrates in the form of sugar, can occur through positive reward, hedonism, and desire [45]. It has been suggested that the brain's reward system may be a contributing factor in the development of obesity—a connection that appears in patterns of sugar consumption, as well as drug and alcohol use [45]. One study found that food addiction can contribute to the severity of obesity and body composition measurements in the general population [46]. Notedly, the study also found that women were at a higher risk of developing food addiction than the male population [46]. Moreover, in individuals with morbid obesity, reduced availability of dopamine D2 receptors and a blunted dopaminergic response to palatable food have been observed, similar to findings in substance addiction [47]. This reduction may drive compulsive overeating as a compensatory response to lower reward sensitivity. Furthermore, chronic overconsumption of highly palatable foods can lead to neuroadaptations in the dopamine system, such as sustained dopamine release and receptor downregulation, reinforcing maladaptive eating behaviors and contributing to obesity [47]. The observed similarities lend support to the hypothesis that, in some instances, obesity and food addiction may be interconnected not just through behavioral patterns, but also through shared neurochemical and neuroanatomical mechanisms rooted in dopaminergic dysfunction. However, further research is necessary to establish the relationship and overlap between obesity and food addiction.

10. Policy, prevention, and treatment strategies for food addiction

Despite arguments that exist on the ability of food to exhibit addictive properties, most professionals agree that the overconsumption of UPFs is a crisis as the obesity epidemic continues to grow. Although there have not been extensive studies linking obesity with food addiction, it is likely that many individuals who are obese may have experienced food addiction. In order to address the public health concern of food addiction in the United States, change must start with closely examining the American food supply and increasing accessibility to fresh, nutritious foods.

More than 53 million Americans (17 %) lived in food deserts where they had little to no access to supermarkets or grocery stores in 2019 per the USDA [48]. Food deserts are often characterized by an abundance of small convenience stores and vendors stocked primarily with ultra-processed foods, while offering little to no access to fresh, nutritious, whole food options. Expanding access to fresh, whole foods and ensuring the availability of well-stocked grocery stores can empower low-income families to maintain a healthier, more nutritious diet. While improving access to fresh foods is essential, such efforts must also consider affordability to ensure that healthier options remain accessible to low-income populations.

Along with addressing the current food deserts and food accessibility in America, it is also important to confront the current American food supply. Presently, more than half of the calories consumed in the average American's household is comprised of UPFs [49]. The United States food system is dominated by UPF items. Developing and enforcing policies aimed at curbing the consumption of ultra-processed foods and enhancing consumer education could prove highly effective in promoting healthier dietary habits. To promote behavior change in individuals struggling with food addiction, providing adequate resources and professional help is vital. It is recommended to support individuals with food addiction by encouraging collaboration with licensed therapists, physicians, and registered dietitians to foster lasting lifestyle modifications—while also expanding access to these essential clinical services for the broader adult population.

11. Conclusions and future directions

A large body of evidence exists supporting the hypothesis that an intricate relationship is present between neural pathways in the brain's reward system and eating behaviors in humans. Current research suggests that food can exhibit addictive properties and result in a dependence that reflects that of drug and alcohol abuse. In particular, ultra-processed highly palatable foods hijack the brain's mesolimbic dopaminergic system, leading to behaviors that prompt the repetitive, excessive consumption of ultra-processed foods. Whenever the chronic consumption of these highly palatable foods persists, it has the ability to alter the brain's reward system and contribute to dependence on food. The current food market that is saturated with UPFs and the behaviors that prompt their consumption have contributed to a rise in obesity and overweight individuals and instances of chronic disease states. Although significant strides have been made to better understand the impact of food addiction, further research is vital to understand the complexity of the condition as well as establish effective solutions to the growing food addiction epidemic. Recently, the general public has started to become more aware of the reality of UPFs and the potential harm they can cause, generating a movement that urges government officials to re-evaluate UPFs in terms of safety and health.

CRediT authorship contribution statement

Nicole M. Avena: Writing – review & editing, Writing – original draft, Supervision, Conceptualization. **Marzia Friuli:** Writing – review & editing, Conceptualization. **Adele Romano:** Writing – review & editing, Conceptualization, Supervision. **Kaylee Hough:** Writing – review & editing, Writing – original draft, Conceptualization.

Declaration of Competing Interest

The authors have nothing to declare.

References

[1] What is the definition of addiction? Asam.org. Updated September 15, 2019. Accessed March 2025. https://www.asam.org/quality-care/definition-of-addiction.

[2] Substance use, abuse, and addiction. Apa.org. Accessed March 2025. https://www.apa.org/topics/substance-use-abuse-addiction#:~:text=Addiction%20is%20a%20stat%20of%20psychological%20and%2For%20physical,or%20behaviors%2C%20such%20as%20sex%2C%20exercise%2C%20and%20gambling. .

[3] American Psychiatric Association. Diagnostic and Statistical Manual of Mental Disorders, 5th ed., American Psychiatric Publishing, Arlington, VA, 2013.

[4] Substance-Related and Addictive Disorders. Psychiatry.org. Published 2013. Accessed March 2025. ⟨https://www.psychiatry.org/File%20Library/Psychiatrists/Practice/DSM/APA_DSM-5-Substance-Use-Disorder.pdf⟩.

[5] A.N. Gearhardt, N.B. Bueno, A.G. DiFeliceantonio, C.A. Roberto, S. Jiménez-Murcia, F. Fernandez-Aranda, Social, clinical, and policy implications of ultra-processed food addiction, BMJ 383 (2023) e075354. Published 2023 Oct 9. doi:10.1136/bmj-2023-075354.

[6] K. Mann, M. Fauth-Bühler, S. Higuchi, M.N. Potenza, J.B. Saunders, Pathological gambling: a behavioral addiction, World Psychiatry 15 (3) (2016) 297–298, https://doi.org/10.1002/wps.20373.

[7] E.M. LaFata, K.C. Allison, J. Audrain-McGovern, E.M. Forman, Ultra-processed food addiction: a research update, Curr. Obes. Rep. 13 (2) (2024) 214–223, https://doi.org/10.1007/s13679-024-00569-w.

[8] Avena N.M., Rada P., Hoebel B.G. Sugar Bingeing in Rats. Curr Protoc Neurosci. 2006;Chapter 9:Unit9.23C. doi:10.1002/0471142301.ns0923cs36.

[9] N.M. Avena, P. Rada, B.G. Hoebel, Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake, Neurosci. Biobehav Rev. 32 (1) (2008) 20–39, https://doi.org/10.1016/j.neubiorev.2007.04.019.

[10] B.G. Hoebel, N.M. Avena, M.E. Bocarsly, P. Rada, Natural addiction: a behavioral and circuit model based on sugar addiction in rats, J. Addict. Med 3 (1) (2009) 33–41, https://doi.org/10.1097/ADM.0b013e31819aa621.

[11] N.M. Avena, C.A. Carrillo, L. Needham, S.F. Leibowitz, B.G. Hoebel, Sugar-dependent rats show enhanced intake of unsweetened ethanol, Alcohol 34 (2-3) (2004) 203–209, https://doi.org/10.1016/j.alcohol.2004.09.006.

[12] A.N. Gearhardt, W.R. Corbin, K.D. Brownell, Preliminary validation of the Yale Food Addiction Scale, Appetite 52 (2) (2009) 430–436, https://doi.org/10.1016/j.appet.2008.12.003.

[13] A.N. Gearhardt, W.R. Corbin, K.D. Brownell, Development of the Yale food addiction scale version 2.0, Psychol. Addict. Behav. 30 (1) (2016) 113–121, https://doi.org/10.1037/adb0000136.

[14] C. Horsager, E.M. LaFata, Faerk E. Lauritsen, M.B. Østergaard, S.D. Gearhardt, AN. Psychometric validation of the full Yale food addiction scale for children 2.0 among adolescents from the general population and adolescents with a history of mental disorder, Eur. Eat. Disord. Rev. 31 (4) (2023) 474–488, https://doi.org/10.1002/erv.2974.

[15] G.F. Koob, N.D. Volkow, Neurobiology of addiction: a neurocircuitry analysis, Lancet Psychiatry 3 (8) (2016) 760–773, https://doi.org/10.1016/S2215-0366(16)00104-8.

[16] R.A. Wise, M.A. Robble, Dopamine and addiction, Annu Rev. Psychol. 71 (2020) 79–106, https://doi.org/10.1146/annurev-psych-010418-103337.

[17] Semaan A., Khan M.K. Neurobiology of Addiction. In: StatPearls. Treasure Island (FL): StatPearls Publishing; November 2, 2023.

[18] National Institute on Drug Abuse. Drugs, Brains, and Behavior: The Science of Addiction. National Institutes of Health; 2018. Accessed December 30, 2024.

[19] D.M. Tomkins, E.M. Sellers, Addiction and the brain: the role of neurotransmitters in the cause and treatment of drug dependence, CMAJ 164 (6) (2001) 817–821.

[20] N.D. Volkow, G.F. Koob, A.T. McLellan, Neurobiologic advances from the brain disease model of addiction, N. Engl. J. Med 374 (4) (2016) 363–371, https://doi.org/10.1056/NEJMra1511480.

[21] C.F. Moore, V. Sabino, G.F. Koob, P. Cottone, Pathological overeating: emerging evidence for a compulsivity construct, Neuropsychopharmacology 42 (7) (2017) 1375–1389, https://doi.org/10.1038/npp.2016.269.

[22] C.F. Moore, J.I. Panciera, V. Sabino, P. Cottone, Neuropharmacology of compulsive eating, Philos. Trans. R. Soc. Lond. B Biol. Sci. 373 (1742) (2018) 20170024, https://doi.org/10.1098/rstb.2017.0024.

[23] B.J. Everitt, T.W. Robbins, Drug addiction: updating actions to habits to compulsions ten years on, Annu Rev. Psychol. 67 (2016) 23–50, https://doi.org/10.1146/annurev-psych-122414-033457.

[24] A. Alcaro, R. Huber, J. Panksepp, Behavioral functions of the mesolimbic dopaminergic system: an affective neuroethological perspective, Brain Res Rev. 56 (2) (2007) 283–321, https://doi.org/10.1016/j.brainresrev.2007.07.014.

[25] A.I. Dunigan, A.G. Roseberry, Actions of feeding-related peptides on the mesolimbic dopamine system in regulation of natural and drug rewards, Addict. Neurosci. 2 (2022) 100011, https://doi.org/10.1016/j.addicn.2022.100011.

[26] O. Arias-Carrión, M. Stamelou, E. Murillo-Rodríguez, M. Menéndez-González, E. Pöppel, Dopaminergic reward system: a short integrative review, Published 2010 Oct 6. Int Arch. Med 3 (2010) 24, https://doi.org/10.1186/1755-7682-3-24.

[27] Chapter 2 The Neurobiology of Substance Use, Misuse, and Addiction. Ncbi.nlm.gov. Published November 2019. Accessed January 2025. ⟨https://pubmed.ncbi.nlm.nih.gov/17905440/⟩.

[28] The Cycle of Alcohol Addiction. Niaaa.nih.gov. Published 2021. Accessed January 2025. ⟨https://www.niaaa.nih.gov/publications/cycle-alcohol-addiction⟩.

[29] G.F. Koob, N.D. Volkow, Neurocircuitry of addiction, Neuropsychopharmacology 35 (1) (2010) 217–238, https://doi.org/10.1038/npp.2009.110.

[30] C.A. Monteiro, G. Cannon, J.C. Moubarac, R.B. Levy, M.L.C. Louzada, P.C. Jaime, The UN Decade of Nutrition, the NOVA food classification and the trouble with ultra-processing, Public Health Nutr. 21 (1) (2018) 5–17, https://doi.org/10.1017/S1368980017000234.

[31] C. Colantuoni, J. Schwenker, J. McCarthy, et al., Excessive sugar intake alters binding to dopamine and mu-opioid receptors in the brain, Neuroreport 12 (16) (2001) 3549–3552, https://doi.org/10.1097/00001756-200111160-00035.

[32] N.M. Avena, K.A. Long, B.G. Hoebel, Sugar-dependent rats show enhanced responding for sugar after abstinence: evidence of a sugar deprivation effect, Physiol. Behav. 84 (3) (2005) 359–362, https://doi.org/10.1016/j.physbeh.2004.12.016.

[33] N.M. Avena, B.G. Hoebel, A diet promoting sugar dependency causes behavioral cross sensitization to a low dose of amphetamine, Neuroscience 122 (1) (2003) 17–20. DOI: 10.1016/S0306-4522(03)00502-5.

[34] C. Davis, N.J. Loxton, R.D. Levitan, A.S. Kaplan, J.C. Carter, J.L. Kennedy, Food addiction' and its association with a dopaminergic multilocus genetic profile, Physiol. Behav. 118 (2013) 63–69, https://doi.org/10.1016/j.physbeh.2013.05.014.

[35] D.S. Hasin, C.P. O'Brien, M. Auriacombe, et al., DSM-5 criteria for substance use disorders: recommendations and rationale, Am. J. Psychiatry 170 (8) (2013) 834–851, https://doi.org/10.1176/appi.ajp.2013.12060782.

[36] K.E. Giel, C.M. Bulik, F. Fernandez-Aranda, et al., Binge eating disorder, Nat. Rev. Dis. Prim. 8 (1) (2022) 16, https://doi.org/10.1038/s41572-022-00344-y.

[37] N.D. Volkow, G.J. Wang, D. Tomasi, R.D. Baler, The addictive dimensionality of obesity, Biol. Psychiatry 73 (9) (2013) 811–818, https://doi.org/10.1016/j.biopsych.2012.12.020.

[38] A. Schienle, A. Schäfer, A. Hermann, D. Vaitl, Binge-eating disorder: reward sensitivity and brain activation to images of food, Biol. Psychiatry 65 (8) (2009) 654–661, https://doi.org/10.1016/j.biopsych.2008.09.028.

[39] A.N. Gearhardt, M.A. White, M.N. Potenza, Binge eating disorder and food addiction, Curr. Drug Abus. Rev. 4 (3) (2011) 201–207, https://doi.org/10.2174/1874473711104030201.

[40] A.N. Gearhardt, S. Yokum, P.T. Orr, E. Stice, W.R. Corbin, K.D. Brownell, Neural correlates of food addiction, Arch. Gen. Psychiatry 68 (8) (2011) 808–816, https://doi.org/10.1001/archgenpsychiatry.2011.32.

[41] I.M. Balodis, C.M. Grilo, M.N. Potenza, Neurobiology of binge eating disorder: emerging evidence for distinct and shared mechanisms, Curr. Psychiatry Rep. 15 (12) (2013) 396.

[42] Y. Yu, R. Miller, S.W. Groth, A literature review of dopamine in binge eating, Published 2022 Jan 28. J. Eat. Disord. 10 (1) (2022) 11, https://doi.org/10.1186/s40337-022-00531-y.

[43] Adult Obesity Facts. Cdc.gov. Accessed January 2025. ⟨https://www.cdc.gov/obesity/adult-obesity-facts/index.html⟩.

[44] C.R. Ferrario, Food addiction and obesity, Neuropsychopharmacology 42 (1) (2017) 361, https://doi.org/10.1038/npp.2016.221.

[45] T. Zilberter, Food addiction and obesity: do macronutrients matter?, 7. Published 2012 May 30, Front. Neuroenerget. 4 (2012), https://doi.org/10.3389/fnene.2012.00007.

[46] P. Pedram, D. Wadden, P. Amini, et al., Food addiction: its prevalence and significant association with obesity in the general population, Published 2013 Sep 4. PLoS One 8 (9) (2013) e74832 https://doi.org/10.1371/journal.pone.0074832.

[47] B. Campana, P.G. Brasiel, A. Silva de Aguiar, S.C.P. Luquetti Dutra, Obesity and food addiction: similarities to drug addiction, Obes. Med. 16 (2019) 100136, https://doi.org/10.1016/j.obmed.2019.100136.

[48] Which Cities Have the Most People Living in Food Desserts. Usafacts.org. Published June 23, 2021. Accessed January 2025. ⟨https://usafacts.org/articles/which-cities-have-the-most-people-living-in-food-deserts/⟩.

[49] Ultraprocessed Foods Account for More than Half of Calories Consumed at Home. Publichealth.jhu.edu. Published December 10, 2024. Accessed January 2025. ⟨https://publichealth.jhu.edu/2024/ultraprocessed-foods-account-for-more-than-half-of-calories-consumed-at-home⟩.