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Is Food Addictive? A Review of the Science

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Abstract

As ultraprocessed foods (i.e., foods composed of mostly cheap industrial sources of dietary energy and nutrients plus additives) have become more abundant in our food supply, rates of obesity and diet-related disease have increased simultaneously. Food addiction has emerged as a phenotype of significant empirical interest within the past decade, conceptualized most commonly as a substance-based addiction to ultraprocessed foods. We detail (*a*) how approaches used to understand substance-use disorders may be applicable for operationalizing food addiction, (*b*) evidence for the reinforcing potential of ingredients in ultraprocessed foods that may drive compulsive consumptions, (*c*) the utility of conceptualizing food addiction as a substance-use disorder versus a behavioral addiction, and (*d*) clinical and policy implications that may follow if ultraprocessed foods exhibit an addictive potential. Broadly, the existing literature suggests biological and behavioral parallels between food addiction and substance addictions, with ultraprocessed foods high in both added fat and refined carbohydrates being most implicated in addictive-like eating. Future research priorities are also discussed, including the need for longitudinal studies and the potential negative impact of addictive ultraprocessed foods on children.

Contents

INTRODUCTION

The modern food environment has changed rapidly in the last 50 years, with ultraprocessed foods becoming the dominant source of calories in the Western world (15, 116). Ultraprocessed foods are defined as industrial formulations made entirely or mostly from substances extracted from foods (oils, fats, sugar, starch, and proteins), derived from food constituents (hydrogenated fats and modified starch), or synthesized in laboratories from food substrates or other organic sources (flavor enhancers, colors, and several food additives used to make the product hyperpalatable) (83). Ultraprocessed foods include carbonated soft drinks, ice cream, chocolate, and potato chips (83). Although controversy exists over inconsistencies in the application of the ultraprocessed label (52), foods typically labeled as ultraprocessed are the main sources of refined carbohydrates (e.g., added sugar) and added fats in the modern diet (83, 116). Compared with minimally processed foods (e.g., fruits, vegetables, legumes), these ultraprocessed foods are also cheap, convenient, and heavily marketed. A significant increase in obesity and diet-related disease has accompanied the rising availability of ultraprocessed foods (95, 125). Diets that are marked by high levels of ultraprocessed food consumption are independently associated with a number of negative consequences, including accelerated heart aging, type 2 diabetes, and increased mortality (1). Thus, ultraprocessed foods have been a key factor in the rising global rates of obesity, diet-related disease, and poor health.

The ability to reduce obesity and diet-related disease in the face of this changing food environment has proven remarkably challenging. Public health campaigns that aim to educate the public about the negative health outcomes associated with ultraprocessed foods and to encourage consumption of minimally processed foods (such as fruits and vegetables) have failed to shift eating patterns in a meaningful way (67, 68). On an individual level, people express a strong desire to eat healthier and lose weight. Almost half of people in the United States attempt to lose weight in any

given year, often by trying to shift their consumption from ultraprocessed to minimally processed foods (78). The weight-loss industry capitalizes on this desire, and in the United States the weight-loss market is now worth $72 billion (76). Despite high levels of personal desire and available services from the weight-loss industry, the ability to make sustained improvements in dietary intake and weight loss can be very challenging (130). Over the long term, many individuals who have initial success will return to prior patterns of dietary intake and regain lost weight (84). Thus, in a modern food environment dominated by ultraprocessed food, overeating is extremely common, efforts to lose weight are challenging, and even successful attempts frequently result in relapse.

A Dietary Evolutionary Mismatch

An evolutionary mismatch between human biology and the modern food environment is a likely contributor to difficulties with long-term weight management (see **Figure 1** for a conceptual model). For the vast majority of human existence, one of the biggest threats to survival was famine. Calorie-dense foods (like fruits, nuts, and meats) were generally not abundantly available and were prone to conditions that would result in scarcity (e.g., weather changes, animal migration) (32). The reward and motivation systems of the brain appeared to evolve, in part, to optimize the likelihood that humans would obtain sufficient calories (64, 143). Intake of calorie-dense ingredients (e.g., sugar, fat) is effective in activating reward/motivation systems, including the endogenous opioid and mesolimbic dopaminergic pathways (20). This enhances the subjective pleasure of consuming foods that are calorie dense and enhances motivation to seek them out. The memory system (e.g., hippocampus) appears to more effectively encode and retain experiences associated with high-calorie foods and the mesolimbic dopaminergic system is more likely to activate at cues (e.g., smells, sights, locations) that have previously been paired with higher (relative to lower) calorie foods, which enhances motivation to seek out these foods (3, 22). Over time, the habit system can become engaged to automatically trigger the tendency to seek out these foods in response to cue exposure, which further streamlines efforts to pursue high-calorie foods (114). Gut hormones that signal caloric need and hunger (e.g., ghrelin, orexin) prime reward/motivation systems in the brain to be more reactive to high-calorie foods and related cues, which can motivate the intake

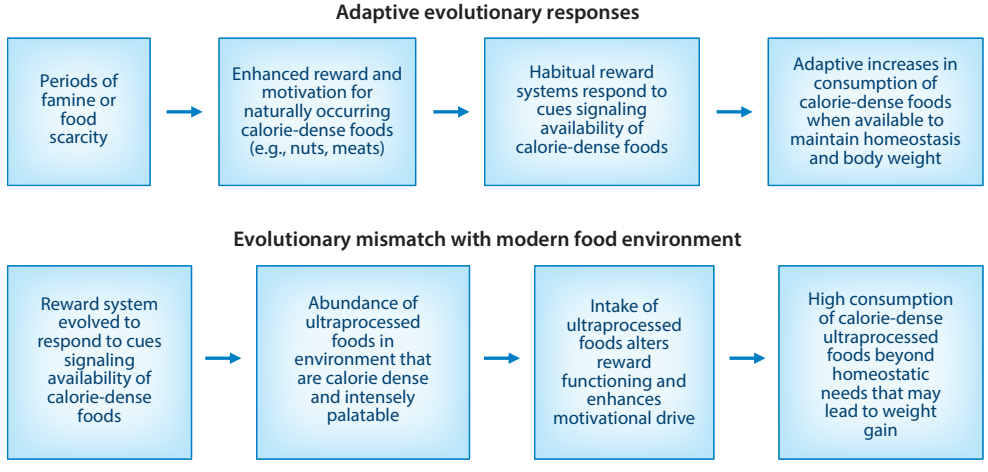

Figure 1

Conceptual model of the dietary evolutionary mismatch: adaptive evolutionary responses and evolutionary mismatch with modern food environment.

of sufficient calories during times of physiological need (13, 28). Stress hormones (e.g., cortisol) can also enhance the reactivity of reward/motivation systems to enhance desire for high-calorie foods, perhaps as a way to buffer against the threat of famine (112).

Thus, reward/motivation, memory, and habit systems of the brain were selected by evolutionary pressures to support the receipt of sufficient calories as efficiently as possible through high-calorie foods (64, 143). In contrast, excessive caloric intake was less of an evolutionary threat to survival, and systems designed to signal sufficient or excessive caloric intake were less essential. Gut hormones that signal higher levels of energy reserve (e.g., leptin) may diminish reward/motivation responses to high-calorie foods (36, 38), but these systems tend to be slower and weaker than those that motivate calorie seeking (143). Further, with repeated high levels of exposure, bodily systems can become resistant (e.g., leptin resistant) to the dampening effects of these hormones (86). Thus, the strong drive to attend to and consume high-calorie foods is coupled with significantly weaker signals to prevent excessive caloric intake.

Although human neural systems are primed to protect against caloric scarcity, the food environment has flipped these evolutionary advantages into risk factors. The most prevalent calorie-dense food options have changed from those such as fruits, nuts, and meats to ultraprocessed foods that have high levels of industrially sourced calorie-dense ingredients (e.g., high-fructose corn syrup, trans fats) (131). Interestingly, naturally occurring foods that are more calorically dense are typically either high in sugar (e.g., fruit) or high in fat (e.g., meat, nuts), but rarely are they high in both. In contrast, the ultraprocessed foods in the modern food environment are often composed of combinations of ingredients (e.g., white flour, sugar, fat) at levels typically not seen in nature (83). These high levels of refined carbohydrates and fats trigger metabolic signals (i.e., glucose oxidation and PPARα activation), which send reinforcing signals to the brain that this food item is highly rewarding (113). This potent combination is further amplified by the addition of unnaturally high levels of sodium and other flavor enhancers and preservatives (39, 85). Compared with naturally occurring foods, ultraprocessed foods can have fiber, protein, and water removed during processing and texturizers can be used to soften the food (making it more likely to melt in the mouth and require less chewing) (25). This allows ultraprocessed foods to be consumed more rapidly and increases the speed with which highly rewarding ingredients, such as refined carbohydrates, are absorbed into the system. Thus, ultraprocessed foods are designed to optimize not only the magnitude of the reward signal in the brain through high doses of calorie-dense ingredients and additives but also the speed with which that reward is delivered (103).

Parallels with Addictive Drugs

Table 1 describes the parallels between drugs of abuse and ultraprocessed foods.

The creation of addictive substances. Our food supply has undergone massive changes in the past 50 years so that we now have a food supply dominated by highly rewarding, ultraprocessed foods (1, 15). The consequences of these changes are not fully understood, but considering parallels with addictive substances (e.g., cigarettes, cannabis, alcoholic beverages, illicit drugs) may be informative. Humans create addictive substances by processing naturally occurring substances (e.g., fermenting fruits into wine, drying tobacco and cannabis leaves for cigarettes) into products with unnaturally high doses of reinforcing ingredients (e.g., ethanol, nicotine, tetrahydrocannabinol) that are effective at activating reward/motivation systems (133). These processed products are typically combined with other additives that further enhance their rewarding effects (e.g., added sugars in alcoholic beverages, menthol in cigarettes) and addictive potential (5). Similarly, ultraprocessed foods are created by combining processed ingredients (e.g., high-fructose corn syrup,

Table 1 Parallels between substance-use disorders and food addiction

Topic	Evidence of parallel between substance-use disorders and food addiction
Substance	Drugs of abuse and ultraprocessed foods have been modified from their natural states to be highly reinforcing by increasing the dose of rewarding ingredients (e.g., nicotine, refined carbohydrates) and the rapidity by which they are delivered to the bodily systems.
	Like addictive drugs, ultraprocessed foods do not exist in nature and are not necessary for survival.
Individual risk factors	Personal risk factors for substance-use disorders and food addiction include a family history of addiction, cognitive control deficits, trauma exposure, and depression.
Environmental risk factors	Environmental risk factors that have increased negative public health consequences related to addictive substances are also driving excessive intake of ultraprocessed foods, such as low cost, high availability, and frequent marketing.
Behavioral symptoms	Shared symptoms have been observed across substance-use disorders and food addiction, including continued use despite negative consequences, cravings, and repeated unsuccessful attempts to cut down.
Neurobiological underpinnings	Neuroimaging studies have demonstrated similar patterns of reward dysfunction and inhibitory control deficits for those with symptoms of food addiction and substance-use disorders.

white flour) and additives (e.g., flavor enhancers) into novel products with unnaturally high levels of rewarding ingredients, such as refined carbohydrates and fat.

One of the most important factors in determining addictive potential is the speed with which the substance is absorbed by the body (14). Delivery mechanisms that lead to rapid absorption of the addictive ingredient, like smoking a cigarette, snorting cocaine, or quickly consuming a shot of liquor, all increase addictive potential (51). In contrast, slowing the absorption rate of an addictive substance can transform an addictive drug into a therapeutic medication, as is the case for slow-release nicotine patches that aid attempts to quit smoking and slow-release stimulant medications used to treat attention-deficit, hyperactivity disorder (71, 138). In parallel, the creation of ultraprocessed foods often includes the removal of ingredients, such as fiber, water, and protein, that slow the rate of absorption of rewarding ingredients (like sugar) into the system and the addition of ingredients (like texturizers) that increase how quickly the food can be consumed (25). Thus, as with addictive drugs, the speed with which rewarding ingredients are delivered and impact the body is increased in ultraprocessed foods.

In sum, the creation of addictive substances bears a striking resemblance to how ultraprocessed foods are created (103). As with addictive drugs, ultraprocessed foods are the result of processing naturally occurring substances (e.g., corn, animal fat) and refining them into evolutionarily novel substances with unnaturally high levels of rewarding ingredients. They are then combined with additives that further amplify their effects and are quickly consumed into the body in a way that increases their ability to rapidly and effectively activate reward/motivation systems in the brain (103).

Necessity for survival. Although ingredients in addictive drugs can have beneficial effects (e.g., increased energy, pain relief, reduced tension), consuming addictive drugs is not essential for survival. In other words, if one never consumes an addictive drug, survival would be possible. The reinforcing and compulsive nature of addictive drugs comes from their ability to activate to an unnaturally high degree the reward/motivation, memory, and habit systems that were optimized to enhance human survival (e.g., caloric intake, social affiliation, sexual intercourse) (132). The ability of addictive drugs to potently activate these systems can shift attention and drive away from life-sustaining behaviors and instead drive forward compulsive drug-seeking and drug-taking behavior that is detrimental to health and survival. Although ultraprocessed food provide calories, which are necessary for survival, they are typically calorie dense and nutrient poor (83). High levels of

ultraprocessed food intake are more strongly associate with obesity, diet-related disease, and early mortality than good health (1, 95). Thus, as with addictive drugs, ultraprocessed foods are not necessary for survival and excess consumption is implicated in poor health and preventable death. Of note, ultraprocessed foods may not need to be as potently rewarding as addictive drugs to begin to highjack reward/motivation systems and encourage excess consumption because these systems are already designed to encourage the intake of high-calorie foods (but not addictive drugs).

Individual risk factors. It is important to note that not all people who consume addictive drugs become addicted to them. For example, 90% of people consume alcohol over their lifetime, but only 14% develop an alcohol-use disorder (55). Even with illicit drugs like cocaine, a relatively small subset of users (20.9%) go on to become addicted (74). Thus, individual risk factors interact with the addictive potential of a substance to determine the likelihood that a specific individual will become addicted. Individual risk factors that increase a propensity for addiction include a family history of addiction, cognitive control difficulties, trauma exposure, and depression (74). As with addictive drugs, not all people who consume ultraprocessed foods struggle to control their intake. Many of the same individual risk factors that increase the risk for drug addiction (e.g., family history of addiction, cognitive control deficits, trauma exposure, depression) also increase the likelihood of excessive ultraprocessed food intake (81).

Environmental contributors to harm. Environmental factors associated with addictive substances also play an essential role. Addiction epidemics are driven not by drastic changes in individual risk factors but by changes in the environment (23). When addictive substances become cheap, easily accessible, heavily marketed, and socially acceptable to use, the prevalence of addictive responses to that substance will increase (23). It is clear that the same environmental factors that increase the public health consequences associated with addictive drugs are also contributing to excessive intake of ultraprocessed foods, including low cost, high availability, and frequent marketing (23).

In sum, the striking parallels between addictive drugs and ultraprocessed foods raise the provocative question of whether we have altered the foods that dominate our modern food environment in such a manner that they are capable of triggering addictive processes. If the addictive nature of ultraprocessed foods is an overlooked contributor to the rising rates of obesity, diet-related disease, and the difficulty of achieving long-term weight loss, then important implications must be considered for both treatment and policy. In the following sections, we (*a*) discuss approaches to identifying an addictive phenotype in eating behavior and the associated clinically relevant outcomes of this phenotype, (*b*) consider the current evidence for which food characteristics are most likely contributing to an addictive response, (*c*) evaluate the implications of applying a substance versus a behavioral addiction framework to addictive eating, and (*d*) discuss clinical and policy implications.

IDENTIFYING AN ADDICTIVE PHENOTYPE IN EATING BEHAVIOR

Conceptualization of Addictive Disorders

It is worth briefly considering the evolving definition of how addictive disorders are conceptualized prior to considering how this framework may apply to problematic food intake. Historically, the addiction label was mostly applied to substances (e.g., alcohol, heroin) that clearly caused mind-altering intoxication and resulted in aversive physical symptoms when the drug was withdrawn (88). However, tobacco presented a challenge to this conceptualization of addiction, which resulted in considerable controversy up until the 2000s (120). The ingestion of tobacco results in

no apparent intoxication syndrome. Individuals can effectively go about their day fulfilling necessary role obligations (e.g., driving a car, participating in child care) while having nicotine delivered rapidly to the brain through tobacco products. Although physical symptoms of withdrawal are relatively mild when tobacco intake is reduced, aversive psychological symptoms (e.g., irritability, anhedonia) often occur and can present a significant obstacle to quit attempts (6). Despite the differences between tobacco and other addictive drugs (e.g., lack of intoxication syndrome, mild physical withdrawal), it is clear that people exhibit addictive patterns of use (i.e., a diminished ability to control intake even in the face of significant consequences), and there is now scientific consensus that tobacco is a highly addictive substance. Like tobacco, ultraprocessed foods do not trigger intoxication and do not cause life-threatening physical withdrawal symptoms, but people are prone to compulsively consume them even in the face of significant negative consequences. Thus, the reconceptualization of addiction triggered by tobacco paves the way for the evaluation of the addictive potential of ultraprocessed foods.

Conceptualization and Assessment of Food Addiction

Initial research into the conceptualization of food addiction relied on self-identification as being a chocoholic/carbohydrate craver or on weight status (e.g., obesity) (46). Both of these approaches have significant limitations. Self-identification with addiction-related terms may not map onto a clinically relevant addiction phenotype but may instead reflect the casual use of addiction terminology in society (e.g., shopaholic). Further, weight status likely results in both over- and underidentification of an addictive eating phenotype. Obesity is a heterogeneous condition that can result from a number of different pathways, including genetic disorders, medication side effects, and physical inactivity, that are not the result of addictive processes. Further, addictive eating may be present but not result in a higher body mass index (BMI) due to compensatory behaviors (e.g., purging, fasting, excessive exercise), which may lead to underidentification of addictive eating in normal weight samples.

In 2009, the Yale Food Addiction Scale (YFAS) was developed to move beyond these limitations by providing a validated tool to operationalize addictive eating through the application of the diagnostic criteria for substance-related addictive disorders to excessive food intake (46). As with other mental health conditions, there is not currently a valid or reliable biomarker of addiction. Instead, the diagnosis of substance-related addictive disorders relies on the presence of behavioral indicators that are associated with diminished control over consumption, continued use despite negative consequences, tolerance/withdrawal, craving, and impairment/distress (see **Table 2**) (6). The YFAS asks individuals to report on the presence of these diagnostic indicators in the context of their consumption of ultraprocessed foods, including sweets, salty snacks, and sugary drinks. The YFAS provides two scoring options: a continuous symptom count (based on summing the number of symptoms endorsed) and a so-called food addiction diagnosis, which is based on the diagnostic threshold for substance-related addictive disorders. Specifically, this food addiction diagnosis occurs on a continuum of mild (two to three symptoms), moderate (four to five symptoms), and severe (six or more symptoms). No singular symptom is required, but clinically significant impairment or distress should be present for this diagnosis (6).

The YFAS has gone through rigorous psychometric validation in a number of samples and has been found to have strong internal consistency, test-rest reliability, and convergent/discriminant/incremental validity (80, 81). Measurement invariance testing has also found that the YFAS is psychometrically sound across different genders and races/ethnicities (24). Abbreviated versions of the YFAS (modified-YFAS and modified-YFAS 2.0) have also been developed and validated to provide briefer assessment options (42, 104). Developmentally appropriate

Table 2 Descriptions of criteria for substance-use disorders in the *Diagnostic and Statistical Manual of Mental Disorders* (6)

Criteria number	Diagnostic criteria for substance-use disorders
1.	Substance taken in greater quantities and over a longer period of time than intended
2.	Persistent desire yet repeated unsuccessful attempts to quit using the substance
3.	Significant time spent obtaining the substance, using, and/or recovering from the effects of substance use
4.	Important social, occupational, or recreational activities given up or reduced due to substance use
5.	Use of substance despite knowledge of adverse physical/emotional consequences
6.	Tolerance (increase over time in the amount of substance use; decrease over time in desired affective experiences)
7.	Withdrawal symptoms when cutting down or abstaining from the substance and use of substance to relieve withdrawal symptoms
8.	Cravings for the substance
9.	Failure to fulfill role obligations due to substance use
10.	Substance use despite interpersonal or social consequences
11.	Substance use in physically hazardous situations (e.g., while operating vehicle)

versions have also been validated for assessing addictive eating in children (YFAS-Children) (49) and adolescents (dimensional YFAS 2.0-C) (102). The YFAS has been translated and validated in more than a dozen languages (e.g., Spanish, Chinese, Korean, Persian) to provide a foundation for global research on addictive eating (80, 81).

In the United States, the prevalence of food addiction based on the YFAS is akin to that of other legal drugs [15% for food addiction (81), 14% for alcohol-use disorders (55)]. There is mixed evidence on whether differences in YFAS food addiction based on gender and race/ethnicity exist (80, 81, 105). However, there is consistent evidence that food addiction is higher for individuals with obesity, with a 4.54 greater likelihood of meeting the YFAS food addiction cutoff (47). However, it is important to note that not all individuals with obesity exhibit an addictive eating phenotype; thus, elevated BMI is not a sufficient proxy for food addiction.

Food addiction is highest for individuals with binge-type eating disorders, with estimates being as high as 97% for those with bulimia nervosa (81). Despite these high endorsements rates, YFAS food addiction scores are still associated with a more severe clinical presentation in the context of other eating disorders, including greater impulsivity, emotion dysregulation, and more frequent binge-eating episodes (80, 81). Surprisingly, endorsement rates are also elevated in anorexia nervosa, which is indicated by excessive underconsumption of food. Food addiction prevalence in anorexia nervosa differs by anorexia subtype, with the restrictive subtype having lower levels (47%) than the binge-purge subtype (74%) (129). Thus, just as there is a subjective experience of binge eating in anorexia, there also appears to be a subjective experience of food addiction despite an objectively small amount of food consumption. However, food addiction (like subjective binge eating) appears to map onto a more severe disordered eating pathology in anorexia and may be clinically important. Further, approximately 50% of individuals who exhibit behaviors that reflect the YFAS food addiction diagnostic criteria do not have an existing eating disorder, yet they appear to be as clinically impaired as individuals with an existing eating disorder diagnosis (e.g., binge eating disorder) (45). Thus, YFAS food addiction may be capturing a phenotype of problematic eating that is not currently captured by existing eating disorder diagnoses and (with additional evaluation) could be considered for inclusion in the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders* (DSM) as a recognized mental disorder.

There is also evidence that factors implicated in addictive disorders are associated with YFAS food addiction (see **Table 1**). Greater cognitive control and emotion regulation difficulties, more intense cravings, higher levels of depression, and a greater likelihood of experiencing trauma are all associated with the presence of addictive disorders and YFAS food addiction (80, 81). Neuroimaging studies have also linked reward-related patterns associated with addictive disorders with YFAS scores. Even when controlling for BMI, YFAS scores in women are associated with greater reward-related activity (e.g., caudate, anterior cingulate cortex) in response to a highly palatable foods cue (e.g., picture of a milkshake) relative to a picture of a water glass (50). In a sample of women with obesity, those who met the criteria for YFAS food addiction (relative to those who did not) exhibited elevated responses in a region associated with cue-induced craving in substance-use disorder (i.e., superior frontal gyrus) for ultraprocessed food images and more robust, decreased activations for minimally processed food cues (110). YFAS scores are also associated with heighted connectivity in reward-related regions (i.e., ventral striatum, basolateral amygdala) when in a fasted state, which suggests that caloric deprivation may enhance a propensity toward addictive eating (29, 92). Differences in inhibitory control neural systems are also associated with YFAS food addiction. Higher YFAS scores in adult women are associated with lower activation in a neural region associated with inhibition (i.e., lateral orbitofrontal cortex) during consumption of a chocolate milkshake in the scanner (relative to a tasteless solution) (50). In adolescents, food addiction scores are associated with hypoactivation in the left middle temporal gyrus and left precuneus/left calcarine sulcus during an inhibitory control task (i.e., go/no-go task) (57). Thus, as with addictive disorders, YFAS food addiction appears to be associated with neural differences in reward and inhibitory control systems.

YFAS scores also appear to be clinically relevant. Although some studies have not found a link between YFAS scores and response to weight-loss treatments (80), a recent large-scale weight-loss treatment trial found that YFAS scores were the strongest psychosocial predictor of attrition and failure to lose weight (40). The literature is mixed regarding the predictive utility of presurgery YFAS scores for response to weight-loss surgery. However, studies have repeatedly found that weight-loss surgery does predict a reduction in YFAS food addiction symptoms (65). For some individuals, this reduction in food addiction symptoms may not be retained at later follow-up points (12-months postsurgery), and the presence of food addiction postsurgery is associated with poorer weight-loss, eating, and lifestyle behaviors (18). There has been less research on the predictive utility of YFAS scores in the treatment of disorder eating. One small study does suggest that pretreatment YFAS scores in bulimia nervosa do predict poorer response to a psychosocial treatment but are not related to attrition (59). An important future direction is the continued investigation of the clinical implications of food addiction and the possibility of developing tailored treatment to address this phenotype.

WHICH FOODS MAY BE ADDICTIVE?

Although the term food addiction is used to reflect YFAS scores, it is clear that not all foods are equally likely to trigger an addictive response. Thus, to deepen our understanding of food addiction and inform clinical and policy implication, it is important to investigate what foods (and aspects of these foods) may be most addictive. Further, the YFAS conceptualizes food addiction as a substance-use disorder, which theorizes that an addictive agent in certain foods directly leads to the development of addictive-like eating behavior. With substance-use disorders, addiction develops when an individual with a predisposition uses an addictive substance. Importantly, if this susceptible person never interacts with an addictive drug, he or she would not develop a substance-use disorder. In parallel, while behavioral characteristics that may predispose an individual to food

addiction have been explored (e.g., impulsivity, emotion dysregulation), the identification of which ingredient or combination of ingredients in foods may be addictive is an essential component for the validity of the food addiction framework. This is also particularly important for addressing early critics of the food addiction construct who posit that it is inappropriate to call overeating an addiction since humans require food for survival (144).

In the past 5 years, a number of studies have used the YFAS or the concept of food addiction to evaluate which foods may exhibit an addictive potential. Schulte and colleagues (103) conducted the first systematic examination that identified the foods most closely associated with YFAS indicators of food addiction. Participants in the study first filled out the YFAS and then rated how likely they were to experience the types of eating problems described by the YFAS with 35 nutritionally diverse food items ranging in processing, caloric density, fat, sodium, carbohydrates, sugar, fiber, and protein (103). Ultraprocessed foods, which were defined as foods with added amounts of fat and/or refined carbohydrates (e.g., chocolate, chips, pizza) [also identified as group 4 in the NOVA classification system (83)], were consistently more associated with YFAS indicators than were naturally occurring, minimally processed foods (e.g., fruits, vegetables, lean protein). Notably, ultraprocessed foods were significantly more problematic for individuals who endorsed experiencing elevated YFAS symptoms of addictive-like eating, providing further support for the role of ultraprocessed foods in food addiction (103).

Paralleling findings from Schulte and colleagues (103), ultraprocessed foods have been identified as most implicated in YFAS indicators or perceived experiences of addictive-like eating by a number of self-report studies (34, 75, 91). Additionally, the consumption of ultraprocessed foods has been associated with subjective experiences of reward that have predicted the abuse liability of addictive substances, such as elevated craving, enjoyment, and satisfaction (108, 109). Relatedly, ultraprocessed foods are more commonly linked to behavioral features of addiction, such as increased loss of control eating and binge consumption (4, 128). These foods also appear to engage brain regions related to reward/motivation (e.g., dorsal striatum) in a similar manner as drugs of abuse (134–136). In summary, studies using self-report, behavioral, and neuroimaging methods have consistently concluded that ultraprocessed foods are the most likely to be associated with features of addiction. This is consistent with the parallels between the creation of ultraprocessed foods and addictive drugs reviewed above.

Thus, parallels exist between ultraprocessed foods and addictive drugs, which may explain why these foods are consistently more associated with addictive responses. However, one point of controversy in the literature is that ultraprocessed foods represent a class of foods and the specific ingredient or combination of ingredients that can be identified as the addictive agent has not yet been identified (58, 144). Comparing this with substance-use disorders, it would not be sufficient to say that alcoholic drinks are more likely than water to be associated with addictive responses; rather, studies revealed that ethanol is the addictive agent in alcoholic beverages that leads to problematic use. While no studies have yet systematically investigated the addictive properties of specific macronutrients, research in preclinical models and recent studies in humans may provide preliminary evidence into which ingredient(s) in ultraprocessed foods may be the addictive agent(s).

Refined Carbohydrates

Refined carbohydrates include sugar and white flour, which are metabolized similarly in the body (63). The amount of refined carbohydrates in a food can be reflected in the food's glycemic index, which is a measure of the magnitude of postprandial glucose release (82). The rewarding properties of refined carbohydrates have been linked to variability in glucose following consumption, characterized by an acute postprandial spike followed by a delayed drop in blood glucose

below one's fasted level, which can trigger cravings to maintain consumption of these foods (122). This hypoglycemic state has been associated with greater activation in reward-related regions that encode the hedonic value of foods and motivate cravings, such as the striatum (73, 90). Further, the release of insulin that follows the consumption of refined carbohydrates has been found to interact with striatal dopamine in a way that heightens the rewarding nature of these foods and influences subsequent consumption (121). Thus, refined carbohydrates appear to alter reward-related physiological and metabolic responses in a manner that may directly perpetuate problematic overconsumption.

In preclinical studies, prolonged, intermittent consumption of sugar, a refined carbohydrate, has been associated with behavioral features of addiction, such as binge eating, increased intake over time suggestive of tolerance, and opiate-like withdrawal when removed from the diet (10, 11). Rats also exhibit downregulation in dopamine responsivity after a period of prolonged sugar consumption, suggestive of sensitization, and locomotor and/or consummatory cross-sensitization to amphetamine, cocaine, and alcohol (8, 9, 53). Neuroimaging studies in humans have observed that intake of ultraprocessed foods high in only refined carbohydrates seems to engage classic dopaminergic reward-related regions (e.g., striatum, insula) (118, 119) that are also responsive to substance use. Broadly, refined carbohydrates appear to activate neural circuitry that may directly motivate cravings and perpetuate consumption, and these foods have been associated with core features of addiction.

Fat

While refined carbohydrates appear to modulate responses in dopaminergic reward regions (e.g., striatum) based on fluctuations in glucose, dietary fat has been more associated with rewarding somatosensory properties, such as texture, flavor, and taste (54, 139). Notably, the types of dietary fat most often found in ultraprocessed foods (e.g., saturated or trans fat) yield elevated reward responses compared with dietary fat most commonly present in naturally occurring foods (e.g., unsaturated) (16). Neuroimaging studies have found that ultraprocessed foods high in fat, but low in sugar, engage regions implicated in oral somatosensory reward (e.g., postcentral gyrus, Rolandic operculum) and encoding the hedonic properties of a reward (e.g., midorbitofrontal cortex) (54, 118, 119). Preference for and increased intake of high-fat foods have also been closely linked to stimulation of the μ-opioid receptors within the ventral striatum, a region associated with liking for rewards (87, 141, 142). Thus, fat appears to play an important role for enhancing palatability and mouthfeel, especially in ultraprocessed foods.

Fat has also been implicated in behavioral indicators of addiction in preclinical models. Rats given prolonged, intermittent access to ultraprocessed foods high in only fat (e.g., shortening) will exhibit binge consumption and enhanced motivation for the food (7, 11). However, removal of fat from the diet does not produce the withdrawal-like symptoms observed upon removal of sugar (7, 11, 21). Although withdrawal is just one symptom of an addictive disorder and is not required for diagnosis, the present evidence suggests that refined carbohydrates such as sugar may be associated with more numerous indicators of addiction than dietary fat. As such, in ultraprocessed foods high in both fat and refined carbohydrates, dietary fat may serve a more ancillary role of enhancing the addictive potential of the refined carbohydrates by increasing palatability and recruiting a broader array of reward-related responses.

A Case for the Combination

While there are some ultraprocessed foods that contain elevated amounts of only refined carbohydrates (e.g., gummy candy, soda, white bread) or only fat (e.g., cheese, bacon), the majority of

ultraprocessed foods that have been most implicated in addictive-like eating have the combination of being high in both fat and refined carbohydrates, such as pastries, potato chips, pizza, cheeseburgers, French fries, and ice cream (34, 75, 91, 103). This is supported in preclinical models, demonstrating that consumption of ultraprocessed foods high in both fat and refined carbohydrates (e.g., Oreo cookies, M&Ms) leads to downregulation of dopamine receptors, binge eating, and willingness to obtain these foods despite negative consequences (e.g., foot shock) (62, 89). Interestingly, the combination of fat and refined carbohydrates does not exist in any naturally occurring foods, and thus these foods may have the highest possible dose of rewarding ingredients. In support of this hypothesis, a recent study by DiFeliceantonio and colleagues (35) examined neural responses to ultraprocessed food cues that were high in fat only, refined carbohydrates only, or both. Reward-related neural activation to the ultraprocessed food cues high in both fat and refined carbohydrates produced a supra-additive response that was greater than the sum of the activation for the ultraprocessed food cues high in only fat or only refined carbohydrates (35). Thus, the combination of refined carbohydrates and fat may be key to determining the addictive potential of ultraprocessed foods.

Although there is a clearly defined addictive agent in drugs of abuse (e.g., ethanol in alcohol, nicotine in cigarettes), these substances contain numerous ingredients, which also serve to elevate their reinforcing nature (5, 93). This explains why individuals with substance-use disorders do not use pure forms of their substance of choice (e.g., 100% ethanol) but rather formulations of the substance that have been carefully crafted to balance ideal proportions of the addictive agent with ingredients that enhance palatability and reduce adverse side effects (e.g., adding sugar to cigarettes reduces the harsh taste of nicotine) (127). With respect to ultraprocessed foods, it may be that there is a specific ingredient that seems to be the primary driver of the problematic responses, but the addictive potential is further elevated by the presence of other rewarding ingredients. For instance, perhaps refined carbohydrates are the addictive agent in ultraprocessed foods, given that they act on addictive-like dopaminergic systems, but fat enhances somatosensory reward and blunts the negative side effects of consuming large quantities of sugar (e.g., reducing glycemic index, blunting withdrawal responses) to create an ideal combination of these rewarding ingredients. Delineating the roles of various ingredients in ultraprocessed foods is an urgent next step in this line of research that will not only provide insight into the validity for the addictive potential of these foods but also inform targets for intervention and public policy initiatives.

Other Potential Contributors

Salt. Cocores & Gold (26) introduced the salted food addiction hypothesis, which posits that salted foods act as mild opiate agonists in a manner that perpetuates overeating and obesity. Importantly, salt is an ingredient that is often added to ultraprocessed foods, co-occurring with fat and refined carbohydrates, such as pizza, potato chips, and French fries. These foods form the basis of the salted food addiction hypothesis, which makes it challenging to disentangle the role of salt compared with the known rewarding properties of fat and refined carbohydrates. Further, no prior studies have attempted to examine the unique behaviors associated with foods high in only salt, as has been done in preclinical studies isolating responses to fat and sugar. There are no ultraprocessed foods that are high in only salt, which may suggest that salt may contribute in a more complimentary manner to enhance the rewarding properties of ingredients such as fat or refined carbohydrates.

Nonnutritive sweeteners. Nonnutritive sweeteners, such as sucralose and aspartame, have a sweet taste higher than that of pure sucralose (sugar) but do not produce changes in glucose. These sweeteners appear to activate neural systems in a different manner than does sugar. Existing

research suggests that artificial sweeteners produce a partial activation of reward regions compared with sugar, since they tap into sensory and taste rewards associated with sweetness but do not engage postingestive dopaminergic reward processes because they lack the expected glucose (44, 79, 140). Thus, researchers have hypothesized that this incomplete reward response may fuel subsequent cravings for foods that contain added sugars in order to activate the postingestive component of reward (72, 140). In support, nonnutritive sweeteners have been associated with weight gain over time in prospective cohort studies (27, 43, 117), though it remains unknown whether greater consumption of nonnutritive sweeteners is a cause or consequence of weight gain. Further, while preliminary evidence suggests that rats exhibit binge consumption with saccharin, an artificial sweetener (2), to our knowledge no studies have examined addictive-like responses to nonnutritive sweeteners in humans, and this represents an area for further study.

Sensory and cognitive properties of food. There are numerous sensory and cognitive properties of ultraprocessed foods that may enhance their rewarding nature, such as palatability, olfactory cues, behavioral contexts, and learned associations of eating these foods during emotionally salient times (e.g., birthday cake). Studies using percutaneous endoscopic gastrostomy tubes for feeding (food directly put into stomach, bypassing mouth and esophagus) have observed significant weight loss among individuals with obesity (124), which may be partially attributed to reduced exposure to the hedonic properties of food associated with consumption.

Additional research may be beneficial for disentangling the roles of these sensory and cognitive characteristics in elevating the reinforcing nature of ultraprocessed foods. Importantly, the addictive potential of other substances (e.g., nicotine, alcohol) is also enhanced through rewarding sensory experiences, behavioral contexts (e.g., mood enhancement at social events), and learned associations. Thus, the contributions of these characteristics would be expected to interact with the addictive ingredient(s) identified in ultraprocessed foods rather than being the sole driving mechanisms perpetuating addictive-like consumption.

A Note on Terminology

In the current review, we use the term ultraprocessed foods, which is based on the NOVA classification that focuses on foods that are composed of industrial sources of energy (e.g., high-fructose corn syrup, hydrogenated oils) and food additives (83). In our prior work, we have often used the term highly processed foods (103, 108, 109), which was operationalized by the presence of added refined carbohydrates and/or added fats. All the foods we have previously classified as highly processed (e.g., ice cream, cookies, pizza) would also qualify as ultraprocessed foods if they are created by the food industry (83). Yet, the highly processed label also recognizes that homemade versions of these products (e.g., homemade cookies) that are created through the use of processed ingredients (e.g., sugar, white flour) are also prone to be consumed in addictive ways. However, research that links a diet high in ultraprocessed foods with negative health consequences has increased rapidly over the last decade (31, 70, 125, 131). Thus, in the current review, we have chosen to adopt the term ultraprocessed foods for consistency with this growing literature. Other terms have also been used to refer to the types of foods that are more likely to be consumed in an addictive manner, including hyperpalatable (48, 91) or refined foods (61). In 2019, a data-driven approach for identifying hyperpalatable foods was proposed based on the presence of fat, simple sugars, carbohydrates, and sodium (39). The overlap between ultraprocessing based on the NOVA classification and the hyperpalatable classification is still unknown, as well as which term best reflects the types of foods that are consumed in an addictive manner. It is clear that all foods are not equally likely to be consumed addictively, particularly naturally occurring, minimally processed foods (e.g., fruits, vegetables, legumes) (103). Thus, the term food addiction needs further

refinement. It will be important for a consistent term to be chosen to reduce confusion in the literature and to more specifically focus on what types of foods should be the focus of clinical and policy interventions. However, more research is needed to definitively settle on which term may be best (i.e., ultraprocessed food addiction versus highly processed food addiction versus hyperpalatable food addiction).

FOOD ADDICTION: A SUBSTANCE-USE DISORDER OR BEHAVIORAL ADDICTION?

One of the major controversies surrounding the food addiction construct in the past 5 years is whether the framework is best conceptualized as a substance-use disorder or a behavioral addiction (58, 69, 106, 107). The key distinction between these two perspectives is whether there is an addictive agent within certain foods (e.g., ultraprocessed foods) that triggers biological and behavioral responses that directly perpetuate compulsive consumption (106, 107). From a behavioral addictions standpoint, addictive-like responses would be triggered by the act of food consumption, which would theoretically suggest that the characteristics of the food would not play a direct role in maintaining the problematic behavior.

Proponents of the behavioral addiction framework (58, 69) highlight that the conceptualization of food addiction based on the YFAS/YFAS 2.0 is based on observable behaviors (e.g., poor control over consumption, use despite negative consequences) and that addictive-like responses are elevated in specific behavioral contexts (e.g., intermittent access), thus suggesting the central role of the behaviors in the phenotype. However, the state of the literature, as described above, specifically points to ultraprocessed foods (or ingredients within these foods) being uniquely implicated in the biological (e.g., downregulation of dopamine receptors with prolonged consumption) and behavioral (e.g., binge eating, withdrawal) addictive-like responses, whereas minimally processed foods have demonstrated little association with these features (62, 89, 91, 97, 103, 108, 109, 142).

Importantly, a substance-based perspective emphasizing the addictive nature of ultraprocessed foods also acknowledges the importance of behaviors in diagnosing and enhancing addictive responses. In the fifth edition of the DSM, substance-use disorders are all diagnosed using 11 behavioral indicators that can be adapted based on individual features of the substance (e.g., withdrawal symptoms from alcohol differ from those for tobacco) (6). The YFAS 2.0 parallels this approach by using these same 11 behavioral indicators adapted for the unique characteristics of ultraprocessed foods.

Further, it is widely acknowledged that certain behavioral patterns of substance use can elevate the addictive potential of the substance, such as bingeing, intermittent access, and use to reduce negative affective states (19, 60, 66, 98, 111, 133). However, it is the addictive agent that interacts with these high-risk behavioral patterns to produce an addictive phenotype, given that engagement in the behaviors alone would not produce problematic responses in the absence of an addictive substance (e.g., binge drinking water would not promote compulsive use) (107). Similarly, animal models have demonstrated that intermittent access to and binge consumption of ultraprocessed foods promote addictive responses (e.g., enhanced motivation, use despite consequences) but this does not occur with the rats' nutritionally balanced chow (7, 9, 62, 89). Thus, the substance-based hypothesis of ultraprocessed food addiction posits that, like all substance-use disorders, the addictive potential of ultraprocessed foods is directly tied to an ingested addictive agent (e.g., fat, refined carbohydrates) that may be enhanced by high-risk behavioral circumstances.

The existing literature provides more support for a substance-based conceptualization of food addiction than a behavioral addiction perspective, given the specificity of ultraprocessed foods

being uniquely implicated in the phenotype. However, as discussed above, the identification of which ingredient(s) in ultraprocessed foods is the addictive agent is an immediate next step in validating the appropriateness of applying a substance-use disorder framework to food addiction. There are various perspectives about the empirical evidence necessary to convincingly confirm the addictive potential of ultraprocessed foods (or ingredients in them). Some researchers assert the addictive nature of these foods based on their ability to activate reward-related neural circuitry in a similar manner as drugs of abuse (134–136), though this has been criticized by others because natural rewards may also activate these regions (41). This has led to a greater focus on brain neuroplasticity, whereby prolonged consumption of substances has been found to change neural processes in a manner that directly perpetuates the addictive behaviors (94, 123)—a pattern not seen with natural rewards (e.g., listening to music, sex). This perspective not only focuses on the substance's ability to alter reward-related neural processes but also emphasizes the need for a behavioral presentation consistent with the diagnostic indicators of addiction, further differentiating addictive substances from natural rewards.

In many ways, these parallels exist with ultraprocessed foods. Prior studies have observed that compulsive consumption of these foods is associated with a change in neural response to food cues or consumption, shifting from the ventral striatum, associated with liking and enjoyment of a reward, to the dorsal striatum, implicated in cravings and enhanced motivation (30, 101). Notably, this pattern is also observed for studies following individuals who use substances recreationally but go on to develop a substance-use disorder (37, 137). This is coupled with the behavioral symptoms of food addiction operationalized by the YFAS at comparable levels with rates of alcohol- and nicotine-use disorders (105). Thus, prolonged consumption of ultraprocessed foods appears to trigger neuroplastic changes in the reward system observed with addictive drugs, coupled with an addictive-like behavioral phenotype, in some individuals.

Future research could strengthen the evidence for the addictive nature of ingredients within ultraprocessed foods by systematically observing neural changes over time. For instance, a tightly controlled inpatient study would be ideal for assessing reward responses before, during, and after repeated consumption of ultraprocessed foods high in fat, refined carbohydrates, or both. This approach would help differentiate which attributes of ultraprocessed foods may trigger addiction-like neuroplasticity. Behaviorally, a recent inpatient study exposed individuals of normal weight to a 2-week diet of ultraprocessed foods and a 2-week diet of minimally processed foods, matching the presentation of calories, sugar, fat, fiber, and macronutrients at each meal and snack (56). Individuals ate approximately 500 calories more per day and gained weight over 2 weeks when eating the ultraprocessed food diet, compared with the minimally processed diet, providing support for the direct role of ultraprocessed foods in perpetuating overeating (56). However, the authors acknowledge that the findings were unable to parse apart the roles of different ingredients in producing these changes or the biological underpinnings (e.g., changes in gut microbiome, hormones, metabolism), and these represent areas for future study. Further, this study did not investigate how exposure to these different dietary conditions impacted neural functioning, which is another important future direction.

CLINICAL AND POLICY IMPLICATIONS OF FOOD ADDICTION

Clinical Applications

Treatments for substance-use disorders are often conceptualized as helping patients achieve abstinence as the primary goal. However, there are numerous evidence-based treatments for substance-use disorders that are not based on an abstinence model, such as harm reduction, mindfulness, and acceptance-based behavioral treatments (17, 77). These interventions are most commonly

implemented for legal substances that are prevalent in mainstream social settings (e.g., alcohol) or produce limited intoxication effects (e.g., nicotine) (12, 77). Broadly, nonabstinence-oriented treatments for substance-use disorders take a more nuanced lens to understand specific substance and contextual triggers that elevate risk for problematic patterns of use. For example, an approach like harm reduction may be applied to a person with alcohol-use disorder by helping them identify that they are highly likely to overconsume alcohol when they are drinking hard liquor alone after a stressful day (high risk). In contrast, this person may rarely drink more than they intend to when they order a glass of wine at dinner with friends while in a positive mood (low risk). Thus, treatment would emphasize abstaining from high-risk situations, while permitting appropriate engagement with the addictive substance in low-risk contexts.

These treatment approaches may be similarly promising for addressing symptoms of ultraprocessed food addiction. Given the ample availability and accessibility of ultraprocessed foods globally, achieving complete abstinence is likely infeasible and unnecessary. Individuals have a wide array of taste preferences (e.g., salty versus sweet) and identified trigger foods and thus may be able to narrow the scope of which foods to reduce or cut out based on their personalized high-risk situations. For instance, paralleling the above example for harm reduction with alcohol, a person with ultraprocessed food addiction may experience addictive-like eating toward sweets (e.g., cookies, chocolate), particularly when eating them alone and/or in a negative affective state (high risk). Yet, this person may rarely experience symptoms of food addiction at a restaurant with friends sharing entrees that are partially composed of ultraprocessed foods (e.g., a sandwich on white bread, pasta with chicken) (low risk). Counseling would then focus on reducing the patient's exposure to high-risk situations and foods while keeping low-risk foods in one's diet to maximize food variety and the flexibility of this way of eating.

Harm reduction and other moderation-based approaches would be highly individualized to account for the specific ultraprocessed foods and environmental triggers that lead to addictive-like eating in each patient, which would circumvent the need to emphasize the extremely unlikely outcome of complete abstinence. Critiques of these treatments' potentials primarily relate to the possible rebound effect that may be observed when specific foods are overly restricted and any lapse triggers significant overconsumption (99). Thus, longitudinal approaches will be essential for systematically evaluating the efficacy of harm reduction compared with existing psychotherapies for overeating that do not restrict consumption of specific food items (e.g., cognitive-behavioral therapy for binge eating) among individuals with ultraprocessed food addiction.

Policy Implications

Addictive substances also do not just negatively impact individuals with clinical levels of impairment; widespread subclinical levels of impairment also contribute significantly to public health concerns (100). Widespread subclinical problems are particularly problematic for public health when the addictive substance is legal, accessible, and socially acceptable (as with alcohol) (100). If ultraprocessed foods are addictive, focusing exclusively on clinical treatments is unlikely to address the widespread public health consequences associated with subclinical patterns of excessive intake. If ultraprocessed foods are capable of triggering enough of an addictive pull to result in excess consumption that is only a few hundred calories beyond daily caloric need, this would be sufficient to increase risk for obesity and related health problems.

One of the most important lessons learned from addiction is that education about the harms of a substance has minimal impact on the ability to reduce widespread risky use on its own (100). Although individuals deserve to be adequately informed about the risk associated with an addictive substance, this is not adequate to address public health concerns. The most effective approach to

reducing problematic intake of addictive substances is policy interventions that aim to alter associated environmental factors. For example, in the context of tobacco, successful policy interventions have focused on raising the price of tobacco products, reducing the marketing of these products (particularly to children), restricting access by phasing out vending machines that administered tobacco products, reducing the ease of use by passing clean air laws that prevent indoor smoking, and restricting the addition of certain flavor enhancers to nicotine products (in the context of vaping) (23). Environmental approaches have been used to a lesser extent in the domain of alcohol, but some states do have rigorous policies that restrict access through zoning requirements to limit bars/liquor stores (particularly around schools), add taxes to increase prices, and limit the sale to certain times of day or certain government-run establishments (100). These types of policies also move the narrative away from a primary focus on the personal responsibility of the individual and target industry practices that facilitate, encourage, and profit from excessive consumption.

Similar environmental-based policies could also be used to reduce the negative public health impact of ultraprocessed foods. In the United States, the early evidence on the impact of taxing sugar-sweetened beverages to reduce intake is promising (96). Other countries are taking a more forceful approach to passing policies to combat obesity. For example, Chile has banned the sale of foods that do not meet strict nutrition requirements in schools and has mandates that prevent child-directed marketing, which have reduced intake of unhealthy foods (at least in the short term) (126). The focus on protecting children with policy is extremely important. Even if ultraprocessed foods have a weaker addictive potential than other addictive drugs, most individuals are not exposed to addictive drugs (such as alcohol, cigarettes, and cannabis) until adolescence/early adulthood. In contrast, frequent exposure to ultraprocessed foods occurs very early in childhood (33). Given the vulnerability of the developing brain, protecting children from the addictive effects of ultraprocessed foods should be a key policy initiative. Thus, while no one policy will be sufficient to address the rising rates of obesity and improve the modern food environment, approaches that aim to increase the price, reduce access, and limit marketing of ultraprocessed foods (particularly to children) are likely part of the solution.

However, the use of policy strategies to protect the public against the addictive potential of ultraprocessed foods may be even more pressing than for other addictive substances. We all have to take in calories to survive. There is no option to completely abstain from food intake. Yet, in under-resourced communities, the vast majority of food options are potentially addictive ultraprocessed foods (31). Outlets that serve ultraprocessed foods (such as fast food restaurants and small convenience stores) swamp the presence of outlets that provide access to more nutritious, minimally processed options in poorer communities and communities of color (31). Companies also stock more ultraprocessed foods in their stores in poorer (relative to more affluent) communities, and promotions for ultraprocessed foods increase on days when individuals who receive government assistance are more likely to shop due to the receipt of benefits (115). Thus, if ultraprocessed foods are addictive, there are major issues of racial and social justice associated with the radically different food environments people operate in based on their socioeconomic status and the color of their skin.

CONCLUSION

In conclusion, ultraprocessed foods now dominate our food environment and are strongly implicated in rising rates of obesity and diet-related disease. Ultraprocessed foods are created in ways that parallel the development of addictive drugs, including the inclusion of an unnaturally high dose of rewarding ingredients that are rapidly absorbed into the system and enhanced through additives. As with addictive drugs, some (but not all) individuals exhibit an addictive pattern of

consumption marked by diminished control over intake, intense cravings, and an inability to cut down despite negative consequences. The YFAS provides a psychometrically sound tool to investigate addictive eating, and higher YFAS scores are associated with mechanisms implicated in addictive disorders and poorer clinical outcomes. Identifying the specific components of foods that most contribute to addictive eating, with high levels of rapidly absorbed carbohydrates, fat, and a combination of the two being a likely factor, is an important area of future study. Novel clinical approaches from the addiction field may be useful in addressing addictive eating, particularly harm-reduction approaches that do not require abstinence. However, considering the public policy initiatives that have been used for addictive drugs, policy approaches that aim to alter environmental factors associated with ultraprocessed foods will be essential in reducing public health consequences.

FUTURE ISSUES

1. Identification of the ingredient(s) in ultraprocessed foods that elevate their addictive potential is an essential next step in establishing the validity of food addiction.
2. Studies are needed to understand how greater food addiction symptoms relate to longitudinal outcomes in existing evidence-based weight-loss treatments.
3. The integration of interventions for substance addictions, such as harm reduction, may warrant investigation for the treatment of food addiction.
4. There is a need to evaluate the interpretation of the Yale Food Addiction Scale in the context of existing eating disorders, in order to elucidate the possibility of subjective versus objective addictive-like eating behavior.
5. Further research is needed on whether ultraprocessed foods are triggering addictive responses in children.
6. If ultraprocessed foods are classified as addictive, it is essential to consider the social justice implications for under-resourced communities and communities of color.
7. The application of public policy initiatives that have been effective in reducing the impact of addictive substances should be evaluated in the context of ultraprocessed foods.

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