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The concept of "food addiction" helps inform the understanding of overeating and obesity: YES

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ABSTRACT

Addictive substances such as opiates and other drugs are highly reinforcing and some (but not all) individuals consume them compulsively. Highly processed (HP) foods have unnaturally high concentrations of refined carbohydrates and fat. These foods are highly reinforcing and some (but not all) individuals consume them compulsively. HP foods, like addictive substances, are more effective in activating reward-related neural systems than minimally processed foods. More importantly, HP foods are associated with the behavioral indicators of addiction: diminished control over consumption, strong craving, continued use despite negative consequences, and repeated failed attempts to reduce or eliminate intake. Thus, HP foods are key in addictive patterns of food intake. Like addictive drugs, HP foods are complex, human-made substances designed to effectively deliver reinforcing ingredients (e.g., refined carbohydrates, fat). Withdrawal and tolerance are not necessary for an addiction classification; however, HP foods can trigger both these processes. On a public health level, the negative consequences of HP foods are high, even for those without clinically relevant levels of addictive eating. The recognition that some foods can be addictive will inform clinical obesity treatment and underscore the importance of environmentally focused policy interventions. *Am J Clin Nutr* 2021;113:263–267.

Keywords: processed food, addiction, withdrawal, tolerance, policy, industry, food environment

Main Argument (Gearhardt)

Highly processed (HP) foods, such as pizza, ice cream, white bread, cookies, and potato chips, now compose the majority of the US food supply (1). HP foods are created by combining refined carbohydrates and fat (often along with sodium and food additives) at concentrations that surpass naturally occurring foods that are minimally processed (MP), such as fruits, vegetables, and legumes (1, 2). National surveys confirm that most adults and even young children consume excessive amounts of HP foods, despite widespread knowledge of potential health consequences (1, 3). Most attempts to reduce HP intake fail, and the majority of those that initially succeed eventually result in relapse (4). This chronically relapsing pattern of excessive HP food intake despite clear adverse health consequences bears a striking resemblance to the intake of addictive substances, which begs the question of whether HP foods can be addictive and whether the concept of food addiction has clinical or policy relevance in combatting obesity.

What indicates a substance is addictive?

Addictive substances are characteristically created by processing naturally occurring substances (e.g., plants for opiates, cocaine, and tobacco; fruits, grains, or sugar for alcoholic beverages) into those with unnaturally high concentrations of reinforcing ingredients (e.g., nicotine, ethanol). There is significant heterogeneity among addictive substances. Some substances are intoxicating (e.g., opiates, alcohol), but others are not (e.g., tobacco). Some have deadly somatic withdrawal symptoms (e.g., seizures in alcohol withdrawal), but others do not (e.g., cannabis, tobacco) (5). Some are commonly consumed in discrete binges (e.g., alcohol), but others are consumed in a controlled, consistent pattern of use (e.g., tobacco). Some are legal (e.g., alcohol, tobacco), whereas others are not (e.g., cocaine). Given this heterogeneity, it is important to evaluate the commonalities across addictive substances to guide the evaluation of the addictiveness of HP foods.

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Abbreviations used: DSM-5, Diagnostic and Statistical Manual, 5th edition; HP, highly processed; MP, minimally processed; NAcc, nucleus accumbens; YFAS, Yale Food Addiction Scale.

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All addictive substances are highly reinforcing and mood altering (i.e., can increase positive affect or reduce negative affect), and some (but not all) individuals who consume them will do so compulsively (i.e., continue use despite negative consequences or a desire to stop) (6, 7). Addictive substances engage the reward system and lead to dopamine release in the nucleus accumbens (NAcc) (6). However, the addictiveness of a substance cannot be determined by a specific biological marker (e.g., magnitude of dopamine release), but rather by the ability of the substance to trigger a core set of behavioral indicators: diminished control over consumption, strong urges or cravings for the substance, continued use despite negative consequences, and repeated failed attempts to cut down or quit (6, 7).

The ability of a substance to trigger the behavioral indicators of addiction increases when the reinforcing ingredient (e.g., nicotine, ethanol) is at a high dose and is rapidly absorbed (8). Delivery mechanisms that increase absorption rate markedly increase addictive potential (8). For example, smoking tobacco leads to a rapid increase of nicotine and is highly addictive. In contrast, a nicotine patch that slowly releases nicotine has minimal addictive potential. Addictive substances characteristically are not simply an individual reinforcing ingredient, but rather a complex mixture of many ingredients that increases the addictive potential of the reinforcing ingredient. For example, cigarettes contain hundreds of ingredients in addition to nicotine, including cocoa, which dilates airways and increases nicotine absorption, and flavor enhancers (e.g., menthol) that mask aversive tastes. The ethanol in alcohol beverages (which typically ranges from ~5% for beer to ≤60% for liquor) is often combined with sugar and other flavor enhancers. Pure grain alcohol (which can approach 100% ethanol) is rarely consumed on its own due both to its aversive taste and the speed with which it triggers aversive symptoms (e.g., dizziness, vomiting). Some addictive substances can be made at home, but industrial options are typically cheaper, more convenient, and are engineered to optimize reward. In sum, complex substances that optimize the positive effects of a rapidly absorbed, reinforcing ingredient are most likely to be addictive.

Despite the addictive nature of common substances of abuse, the majority of individuals who use them do not become addicted. For example, only 20.9% of cocaine users become addicted (9). Individual factors, such as a family history of addiction, mood disorders, trauma exposure, and inhibitory control difficulties importantly modulate risk (9). Situational factors are also important. Substance use in response to negative affect increases addictive potential (10). Cues commonly associated with the addictive substance become powerful motivators of use, and intermittent binge patterns of intake enhance the incentive power of substance-related cues (11). Environmental factors are important in determining the harms associated with addictive substances. Epidemics often occur when addictive substances are inexpensive, easily accessible, socially acceptable, and heavily marketed (9).

Applying an addiction framework to HP foods

At first glance, HP foods appear to differ from traditionally addictive substances. Food is essential for survival, unlike substances like nicotine and alcohol. However, beyond providing calories, HP foods provide few health benefits. As with other addictive substances, HP foods are highly reinforcing, mood altering, and some (but not all) individuals will consume them compulsively (2, 6). HP foods are more effective than MP foods at activating dopamine release in the NAcc (12, 13), but most importantly HP (but not MP) foods are associated with the behavioral indicators of addiction: diminished control over consumption, strong urges or cravings for the substance, continued use despite negative consequences, and repeated failed attempts to cut down or quit (2). Thus, HP foods can be viewed as having addictive potential.

High doses of refined carbohydrates and fat underlie the reinforcing potential of HP foods. Humans are evolutionarily designed to find carbohydrates and fat rewarding due to their energetic value (12, 13). As with traditionally addictive substances, HP foods have been processed to have unnaturally high doses of these reinforcing ingredients (2, 12). HP foods (and the refined carbohydrates and fats in these foods) are effective in activating rapid dopamine release in the NAcc through their pleasant oral somatosensory properties (e.g., sweet taste, mouth feel) and postingestive effects (12, 13). The ability of refined carbohydrates to rapidly increase blood glucose, and of fat to activate the vagus nerve appears important in triggering dopamine release (12, 13). Foods with high concentrations of both refined carbohydrates and fat (e.g., chocolate, ice cream, French fries, pizza) are mostly strongly associated with addictive behaviors (2, 14). However, individuals will addictively consume foods that are high in carbohydrates but contain little or no fat (e.g., sugar-sweetened cereals, gummy candies, white bread) (2, 14). The ability of these foods to rapidly spike blood glucose likely contributes to their addictive potential (2, 15). High-fat foods with little or no carbohydrates (e.g., steak, bacon) have a lower addictive potential (14). Thus, rapidly absorbed refined carbohydrates are more strongly implicated in the addictive potential of HP foods than fat.

The removal of ingredients (like fiber) also increases the addictive potential of HP foods by increasing the rapid ingestion of reinforcing ingredients (2, 15). Protein is not strongly associated with a reward response (16), but low-protein diets can reduce satiety and increase the ability of HP foods to trigger a reward-related neural response (16). Sodium is a common ingredient in HP foods that enhances the reward value of foods with refined carbohydrates and fat (17, 18). Homemade foods that contain high concentrations of rewarding processed ingredients (e.g., sugar, butter) can also be addictive, but industrial versions of these foods contain flavor enhancers (e.g., glutamate) and texturizers (e.g., gelatin) that further amplify the rewarding sensory properties of HP foods (19). As with conventional addictive substances, HP foods are complex substances that rapidly deliver high doses of refined carbohydrates and fat that are often combined with food components (e.g., sodium, flavor enhancers) that enhance their rewarding nature.

Also, similarly to conventional addictive substances, not everyone who consumes HP foods overeats or exhibits behavioral indicators of addiction. Estimates of HP food addiction based on the Yale Food Addiction Scale (YFAS) approximates the same prevalence as other legal addictive substances [i.e., 15% for HP food addiction (20), 14% for alcohol-use disorders (21)]. Individual differences associated with HP food addiction are similar to those associated with other addictions (e.g., trauma history, mood disorders, impulsivity) (20). Situational

factors associated with substance addiction are associated with problematic HP food consumption, including intake in response to negative affect, cue-rich environments, and intermittent binge use (20, 22). Environmental factors that increase the harm of addictive substances are strongly implicated in the widespread public health consequences associated with HP foods, such as affordability, accessibility, and marketing (23).

Withdrawal and tolerance

Although HP foods are clearly associated with the core behavioral indicators of addiction (2), there has been less investigation into tolerance and withdrawal—adaptations that develop in response to substances that perturb homeostasis. These adaptations require higher levels of consumption to achieve the same level of effects (i.e., tolerance) and aversive psychological (and sometimes physical) symptoms that occur when substance use is reduced or discontinued (i.e., withdrawal) (5). Withdrawal and tolerance are diagnostic indicators of addiction, but they are not necessary nor sufficient (5). Indeed, withdrawal and tolerance are common with nonaddictive substances [e.g., antidepressants (6)], and withdrawal does not occur following discontinuation of all additive substances (e.g., phencyclidine). Even so, there is emerging evidence that HP foods can lead to tolerance and withdrawal in humans. For example, striatal activation is diminished after repeated exposure to a sugar-sweetened beverage (24), which is consistent with neural adaptations associated with tolerance. Individuals report symptoms of withdrawal (e.g., irritability, anhedonia) when cutting down on HP foods, which is associated with dietary change failure (25).

Potential costs and benefits of the addiction label

HP foods are highly reinforcing and mood altering, and trigger core behavioral indicators of addiction (2). HP foods are also highly effective in activating neural reward systems (due to high concentrations of refined carbohydrates and/or fat) (12, 26). Thus, HP foods clearly meet the criteria for an addictive substance. However, it is still worth weighing the potential costs and benefits of applying an addiction label to HP foods. One concern is that the term "addiction" as applied to food could increase obesity-related stigma, although evidence in this regard is mixed, with some studies finding reduced weight stigma when HP food addiction is used as an explanation for obesity (27). Another concern is that the concept of addiction could imply the need for abstinence from all HP foods, an approach that could lead to unintended consequences (e.g., excessively restrictive eating patterns). However, many empirically supported, addiction-focused treatments aim to reduce harm while allowing moderate use of the addictive substance (28). Such an approach could be relevant for HP foods.

There are important benefits of applying an addiction label to HP foods. A focus on addictive mechanisms could lead to novel treatment approaches for disorders associated with excess HP food intake (e.g., obesity). However, one lesson learned from addictive substances is the importance of limiting environmental availability (e.g., increasing price through taxation, reducing marketing to children) with policy measures to reduce the public health burden (23). Even individuals without clinically relevant addictions are prone to overconsume cheap and accessible addictive substances in ways that negatively impact health (29). The widespread availability of inexpensive HP food also leads to excess consumption that increases risk for diet-related disease (even in individuals without clinically relevant addictions) (3). Belief that HP foods are addictive is strongly associated with support for policies to improve the food environment (30), which can benefit those with clinical and subclinical responses to the addictive nature of HP foods. Another major implication of the addiction label is food industry responsibility. Food industry engineers design HP foods to surpass natural levels of reward and then target vulnerable populations, like children, through aggressive marketing strategies. The food industry has followed the playbook of the tobacco industry, with an apparent strategy to reduce their culpability for the rising rates of diet-related disease (e.g., intense lobbying, funding favorable research studies) (23). However, the recognition that HP foods are addictive provides not only new clinical tools, but also a clear rationale for proactive public health interventions.

Refutation (Hebebrand)

According to Gearhardt, food addiction is a valid concept because of the similarities between the addictive-like consumption of both substances/drugs and HP foods. However, the nature—or more precisely the chemical structure—of the underlying nutrient(s) remains elusive. Only a single sentence in her main argument addresses the crucial prerequisite for the categorization of food addiction as a substance use disorder: "high doses of refined carbohydrates and fat underlie the reinforcing potential of HP foods." What is the evidence that only high doses exert a reinforcing potential? How many substance use disorders result from which kind of refined carbohydrates and/or fat in any given person and overall? How can food additives, spices, and other nutrients be excluded as also causing food addiction?

A Dutch study pinpointed the types of foods underlying YFAS-defined food addiction (31). Just 5% of 1495 young adults reported food addiction for specifically sugar-sweetened foods. A stronger association was observed for combined high-fat sweet and/or high-fat savory foods. The investigators deemed food energy density and the individual experience of eating, not food addiction, as the major determinants of the reward value of food.

Indeed, not everyone who consumes alcohol (or any drug) develops a substance use disorder, as Gearhardt argues. Nevertheless, *all* people who drink alcoholic beverages at even moderate amounts experience an altered state of mind (not just an altered mood), and at high amounts intoxication, one of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) Substance-Induced Disorders (5). As stated in DSM-5, "instead of achieving reward system activation through adaptive behaviors, drugs of abuse directly activate the reward pathways" (5). Susceptibility to develop a substance use disorder hinges on the rewarding experience of such an altered state of mind (32). Tellingly, HP foods ingested daily by the majority of the general population (3) do not result in a "high" or upon overconsumption in substance-induced disorders.

A high glycemic index is discussed as one of several potential mechanisms for eliciting an addictive-like overconsumption of carbohydrates. Indeed, high–glycemic index foods can elicit a rapid shift in blood glucose and insulin concentrations thereby

modifying mesolimbic dopamine concentrations (33). However, such an effect does not readily classify as a "direct" (5) activation of the reward system. Because some unprocessed foods also have a high glycemic index (34), the potential to induce an addictive-like eating behavior would not be specific to HP foods as suggested by Gearhardt. In historical terms, obesity and food craving had already occurred prior to the advent of HP foods (35).

Gearhardt states that "pleasant oral somatosensory properties (e.g., sweet taste, mouth feel)", "postingestive effects," and "the removal of ingredients (such as fiber)" can further increase "the addictive potential of HP foods." Alcoholic beverages are indeed also processed in many ways, which affects individual preferences. However, if withdrawal symptoms occur, a person with an alcohol use disorder will ingest any alcoholic beverage available. Intravenous application of ethanol prevents an alcohol withdrawal syndrome (36). Of course, this model cannot be applied to HP foods: intravenous administration of refined carbohydrate (dextrose) or fat does not elicit addiction despite rapid availability to the central nervous system.

Importantly, the link between "food addiction" and overeating and/or obesity is not addressed by Gearhardt. Ultraprocessed foods are ubiquitous and comprise ∼60% of the total energy intake in the United States (3). Many people experience cravings for particular HP foods. Notably, the majority of people with obesity do not fulfill the criteria for "food addiction" and, vice versa, many without obesity fulfill criteria for YFAS-defined food addiction (37). Nevertheless, laypersons can wrongly equate food addiction with obesity. Accordingly, the societal implications of the broad scaled medicalization inherent to "food addiction" need to be recognized; people with obesity can feel doubly stigmatized. Importantly, the term addiction "was omitted from the official DSM-5 substance use disorder diagnostic terminology because of its uncertain definition and its potentially negative connotation" (5).

The overconsumption of "addictive" foods is individually influenced by visual, auditory, textural, olfactory, gustatory, temporal, social, physiological, and psychological cues, which interact to stimulate the reward system. This is not to say that this complex psychobiological system controlling food intake in humans does not include direct links between nutrients and the reward system. For example, the mesolimbic dopamine neurons targeted by peripheral hormones controlling appetite and energy expenditure can sense fatty acids, conferring rewarding properties (38, 39). These neurons respond to variation in the extracellular concentrations of glucose, fatty acids, and ketone bodies, monitoring the availability of nutrients. HP foods likely contain >1 nutrient eliciting such a rewarding effect. However, even their combined effect size for reward would appear to be orders of magnitudes smaller than for substances considered classically addictive. Evidence that specific food ingredients are key determinants of addictive-like eating behavior is lacking.

This refutation does not negate the potential negative implications of HP foods for physical and mental (40) health. The food industry has undoubtedly been extremely successful in designing and mass producing a huge variety of novel foods. Humans are not safeguarded from overeating in an environment with virtually limitless availability and variety of rewarding foods, and cues inherent to them. Structural prevention, including regulation of the food industry, is warranted. Policies need to be pursued to limit the continuous marketing of novel HP foods, especially to youngsters, and to restrict access to HP energy-dense and palatable foods, especially in schools. However, these preventive efforts should not be based upon a scientifically problematic concept. We need to focus on remedies, not weak conceptualizations.

Rebuttal (Gearhardt)

The main aim of this debate has been to evaluate the evidence regarding whether HP foods are addictive. It is also important to consider the strength of the evidence that HP foods are *not* addictive and to consider the public health consequences of potentially misclassifying HP foods as nonaddictive. Repeatedly, we have misclassified addictive substances as nonaddictive, which misinforms the public about risks and contributes to the narrative that excessive users just lack willpower. The addictive nature of tobacco was denied for decades, in large part because tobacco does not produce a traditional "high" and triggers a markedly weaker neural reward response relative to other drugs (like stimulants) (41). This misclassification allowed the tobacco industry to refine their products to become more addictive, aggressively market them to vulnerable populations (like children), all with little regulatory oversight (23). Recently, prescription opioids (like OxyContin) were labeled as having little addictive potential and were then aggressively promoted to prescribers and patients. As opioid addiction increased, the industry labeled addictive individuals as "reckless criminals" to shift the culpability from their product (42). These addiction misclassifications have contributed to immense levels of human death and suffering.

There are striking parallels between the history of misclassifying other addictive substances and the current debate about the addictiveness of HP foods. The domination of the food environment by HP foods has occurred over the past 50 y and has been accompanied by a notable increase in obesity, diet-related disease, and preventable death (1). The behavioral indicators of addiction—intense cravings, loss of control over consumption, continued use despite negative consequences, and high rates of relapse—are clearly associated with the intake of HP foods (2, 14). Hebebrand calls into question the addictive nature of HP foods because they do not cause a "high" and have relatively weaker neural reward activation relative to some drugs, which are the same arguments that contributed to the misclassification of tobacco (41). Hebebrand also states that HP foods do not directly activate the neural reward system, and that the chemical structure of the addictive nutrient has not been identified. Yet, direct activation of the reward system is not viewed as necessary (nor sufficient) for addiction (6). Gambling is clearly addictive despite no direct pharmacological activation of the reward system (5). However, there is clear evidence that HP foods do activate the reward system through oral-sensory and gut-to-brain pathways (12, 13, 43). Addiction classification is also not based on identifying the underlying chemical structure of a substance or identification of a specific neural marker, but the ability of the substance to trigger the behavioral indicators of addiction (particularly in high-risk individuals). HP foods clearly meet this qualification. As we did with cigarettes, misclassifying HP foods as nonaddictive would allow the industry to continue to create new addictive HP foods, market these products to vulnerable populations, and blame those who overconsume them

as lacking personal responsibility. If history is repeating itself with regard to HP foods, our health and well-being will again suffer.

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