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FOOD ADDICTION (E SCHULTE, SECTION EDITOR)

The Reinforcing Natures of Hyper-Palatable Foods: Behavioral Evidence for Their Reinforcing Properties and the Role of the US Food Industry in Promoting Their Availability

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Abstract

Purpose of Review Hyper-palatable foods (HPF) may exploit our neurobiological propensities to seek and consume rewarding foods. The review highlights evidence from basic behavioral and neurobiological studies in humans on the reinforcing properties of HPF and consequences of repeated HPF consumption over time. The review also addresses HPF within the context of the US food environment.

Recent Findings There is reasonably strong behavioral evidence to indicate that HPF may have reinforcing properties that are similar to drugs of abuse. Evidence indicates that healthy individuals may exhibit greater preference for HPF relative to non-HPF and that powerful cues may develop that indicate the presence of HPF as a reward. Preliminary evidence indicates that elevated HPF intake may yield neurobiological consequences for brain reward neurocircuitry. The US food environment provides wide and easy access to HPF. Conceptualized as a substance, HPF exist unregulated in our environment, similar to the tobacco availability in the 1940s. Parallels have been drawn between food and tobacco company practices; a review of industry documents indicates that tobacco companies owned major US food companies since the 1980s, possibly leading the development and proliferation of HPF.

Summary There is reasonably strong evidence to indicate that HPF may have powerful reinforcing properties similar to drugs of abuse; however, more longitudinal work is needed.

Critical attention to the factors and drivers of HPF proliferation in the US food system is paramount to conceptualizing the presence of HPF in our food environment and in considering strategies to protect the US population's health.

Keywords Food reward · Food addiction · Reinforcement · Food environment · Policy

Introduction

Food as a Reinforcer and Hyper-Palatable Foods

Food consumption promotes our survival. Accordingly, consumption of food is an inherently rewarding process that serves to increase the likelihood that we consume more food in the future [1, 2]. There are multiple mechanisms through which food serves to reinforce our ingestive behavior, which consist of behavioral learning processes and internal physiological processes. The role of physiological processes, such as those related to gut-brain signaling, have recently been reviewed [3, 4]. Behavioral reinforcement processes, which may have important influences on neurobiological and physiological processes, will be the focus of this review.

Many foods are palatable in their originally occurring forms in nature. For example, fresh fruits such as an apple are typically sweet and pleasant to ingest. Many vegetables, such as zucchini or broccoli, have savory flavors and may also be pleasant to ingest. The appealing properties of foods elicit our approach toward and consumption of food, thereby promoting our survival [5]. Neurobiologically, consumption of a food (upon initial exposure) activates brain reward neurocircuitry, which provides a pleasant eating experience

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and serves to promote further ingestion [6, 7]. With repeated consumption of a food, cues indicating the presence of a food (e.g., sweet scent of an apple) activate brain reward pathways, promoting approach and consumption of the food [8]. Along with the pleasant and rewarding characteristics of food, foods that occur in nature typically contain one primary palatability-inducing nutrient that occurs in combination with nutrients that promote satiety. For example, sugar in an apple is accompanied by both fiber and water, which promote satiety and slow the process through which sugar may be absorbed and digested. Thus overall, a set of evolutionarily important processes exist that reinforce our approach toward and consumption of food, and these occur alongside nutritional safeguards, such as satiety-promoting nutrients, that together influence our consumption behavior [9].

Hyper-Palatable foods

In the current US food system, many available foods have been modified from their originally occurring forms in nature. Food modification is not inherently problematic. However, some foods have been altered in ways that may serve to exploit our neurobiology and behavioral propensity to approach and consume palatable foods. For example, hyper-palatable foods (HPF) are foods that have substantial deviations from their naturally occurring forms, and these deviations are designed to maximize a food's rewarding properties during consumption [10•]. In contrast to naturally occurring foods that contain one primary palatability inducing nutrient, HPF contain combinations of nutrients that are not typically found in nature (e.g., high quantities of both sugar and fat) [11]. The combinations of nutrients are present at thresholds that artificially enhance a food's palatability and provide a highly rewarding eating experience, eliciting strong activation in brain reward regions [3, 12]. Furthermore, HPF may delay engagement of physiological satiety mechanisms and extend eating occasions involving HPF. Sensory specific satiety is an exposure-based mechanism that serves to bridge food intake with gastro-intestinal reception and processing of food [13]. Within a meal, each bite of a food will be experienced as less enjoyable or pleasant than the prior one, which is a learning-based habituation process that leads to the cessation of consumption [13]. However, sensory-specific satiety is a process that is subject to variability based on characteristics of a food, and evidence indicates that HPF may garner weaker sensory specific satiety responses compared to non-HPF, which may facilitate extended eating occasions [14]. Taken together, HPF may create a highly rewarding eating experience and may delay engagement of physiological satiety mechanisms, leading to excess energy intake.

Many different types of foods may be hyper-palatable. Our analysis of HPF in the US food system revealed that HPF are widely available and comprise many different types of foods and products [10•]. For example, some HPF may be fast foods and desserts, which may be expected to be hyper-palatable. However, HPF may also many meal-based items, frozen foods sold in grocery stores, snacks, and even some foods marketed as healthy due to having reduced calories or fat [10•]. Furthermore, foods identified as related constructs in the literature, such as ultra-processed foods and high energy density foods, may also be hyper-palatable. For example, ultra-processed foods undergo extensive processing and often contain industrial additives that are designed to enhance a food's palatability (e.g., high fructose corn syrup as a sweetener); thus, many ultra-processed foods may be hyper-palatable [15]. In addition, foods with elevated energy density may contain nutrients such as fat and/or sugar that increase both the caloric density of the food and a food's palatability [16, 17]. Thus, foods with high energy density may also have enhanced palatability. To this point, our prior work has indicated that approximately 50% of foods that are HPF also have high energy density and that > 60% of HPF may also be classified as ultra-processed [10•, 18]. Thus, foods that have enhanced palatability are extremely widespread in our food system and are present across many types and categories of foods. In this review, the term HPF will be used to describe foods that may have artificially enhanced palatability; however, the foods may be described using other terminology (e.g., high energy density) in their original articles.

Hyper-Palatable Food as a Substance

HPF may serve as potent rewards and may yield reinforcement processes that drive habitual intake of HPF, similar to the reinforcement processes observed with other powerful reinforcers, such as drugs of abuse. In this regard, Gearhardt and colleagues have suggested that HPF may be considered the target substance of food addiction [19•, 20]. If viewed as a substance, HPF may be expected to induce a series of behavioral and neurobiological changes from repeated consumption over time, which would serve to drive and maintain habitual HPF intake. For example, with drugs of abuse (alcohol, nicotine, opioids, etc.), ample evidence has indicated that repeated consumption of a substance yields a series of neurobiological changes that drive continued use [21, 22]. For example, with repeated substance use, neuroadaptive changes occur from repeated and excessive activation of brain reward neurocircuitry, such that individuals experience blunted pleasure or reward during use, but also experience greater motivation or drive to use as they become hyper-sensitive to substance-related cues in the environment (sensitization) [22]. As a result, a behavioral pattern occurs

in which individuals exhibit strong motivation to use and engage in compulsive use despite consequences, often while experiencing less pleasure or rewarding effects while using [21, 23]. Importantly, the effects are on a continuum and in a dose–response relationship. Thus, effects may be relevant for those who engage in a variety of use patterns, from occasional to excessive or daily use [24]. Applied to HPF, similar behavioral, motivational, and neurobiological changes may be expected to occur as repeated HPF intake may serve to dysregulate neurobiological reinforcement mechanisms [25]. Effects may be expected to occur most prominently among individuals who engage in excess HPF intake and/or who have individual risk factors or vulnerabilities to the rewarding effects of HPF [26–28]. However, the reinforcing properties of HFP would also be expected to be observed on a continuum, with most of the population exhibiting some level of approach toward and preferential consumption of HPF, which may yield passive overconsumption and/or reward-driven eating behavior. A smaller segment of the population would be expected to develop compulsive use of HPF due to preexisting vulnerabilities to the reinforcing properties of HPF [29]. The evidence for HPF as a potent reinforcer with similar properties as other substances of abuse is reviewed herein, with a summary of behavioral evidence first, followed by neurobiological evidence in humans. It should be noted that the foods evaluated in the studies described below aligned with the premise of hyper-palatability; however, many used other terms to describe and identify foods.

Behavioral Evidence (Humans)

If HPF serve as potent reinforcers and drive HPF intake behavior, evidence from basic behavioral studies should indicate that healthy individuals exhibit preferential valuation and approach toward HPF, relative to foods that are not HPF (non-HPF). Evidence should also indicate that with repeated consumption, individuals experience greater motivation to consume HPF, becoming hyper-sensitive to HPF cues (sensitization). Overall, there is reasonably strong evidence to suggest that HPF may be valued and preferred over non-HPF, and there is preliminary evidence indicating that repeated HPF intake may elicit enhanced motivation to consume HPF (sensitization) over time. However, given the substantial body of literature focused on consequences of HPF intake (e.g., obesity and food addiction), far fewer basic behavioral studies have been conducted to establish these fundamental aspects of HPF as reinforcers among healthy individuals.

Evidence indicates that healthy individuals may on average exhibit greater valuation of and preference for HPF compared to non-HPF. Studies in the literature have examined preferences for HPF using behavioral tasks that measure valuation of HPF and non-HPF based on (1) how much individuals are willing to pay to access an HPF or non-HPF or (2) how much effort individuals exhibit (e.g., mouse clicks) to access an HPF or non-HPF. Overall, evidence has indicated that on average, healthy individuals may be willing to pay more for or exhibit greater behavioral effort to obtain an HPF vs a non-HPF, indicating greater valuation and preference for HPF vs non-HPF among healthy samples of adults [30–34], adolescents [35–37], and children [38]. In addition, one study examined preference and choice impulsivity for HPF vs non-HPF among healthy adults and used a standardized definition of HPF to select food items [39]. Similar to the studies of HPF valuation, results indicated that individuals exhibited greater choice impulsivity that was specific to HPF, compared to non-HPF [39]. Thus overall, evidence suggests that healthy individuals on average exhibit preferential valuation of HPF relative to non-HPF, likely due to their stronger reinforcing properties.

Evidence also supports the premise that individuals seek out/approach HPF for their rewarding effects. First, a large body of evidence has suggested that cues that indicate the presence of HPF, such as food logos, signs, and related paraphernalia, may become conditioned stimuli that predict the availability of HPF [40–42]. We navigate our environments by learning about cues that maximize our chances of obtaining evolutionarily meaningful rewards (e.g., food) [40]. Thus, the development of conditioned cues for HPF speaks to the strength of HPF as a reinforcer and parallels the cue development processes that occur with substances [43]. In addition to eliciting approach to HPF cues, preliminary evidence also indicates that HPF may be sought and consumed for their rewarding effects. For example, researchers have found that one common motive that individuals seek out HPF is for their rewarding effects, termed enhancement motive in the literature [44, 45]. Consuming HPF for their rewarding effects has been specifically identified as an important motive in binge eating when conceptualized on a continuum from passive overconsumption to loss of control eating [46]. Furthermore, preliminary evidence indicates that HPF, when defined using the standardized definition, comprised the vast majority of calories consumed during binge eating episodes among a clinical eating disorders sample, thus indicating that HPF may be primarily targeted for consumption during clinically significant binge eating episodes [47]. Overall, evidence indicates that HPF may be sought out for their rewarding effects, consistent with other substances of abuse.

Preliminary behavioral evidence from several studies has suggested that repeated HPF consumption over time may increase motivation to seek out HPF as individuals become hyper-sensitive to HPF cues; however, observed effects have varied across studies and groups examined. For example, two experimental studies among healthy adults used 14-day exposure protocols in which participants consumed the same

HPF snack daily. Both studies reported that motivation to consume the HPF, measured behaviorally, increased over the exposure period for individuals with obesity; however, motivation to consume HPF decreased among individuals without obesity [48, 49]. In a longer trial among healthy adults, findings revealed that motivation to consume HPF did not change following daily HPF snack intake over 12 weeks [50]. The retention of motivation to consume the HPF snack despite repeated exposure over time was interpreted to indicate that HPF may circumvent habituation processes that would be expected to occur with repeated exposure over time [50]. Finally, in another 14-day consumption trial among adolescents, researchers reported the sample on average exhibited decreases in motivation to consume HPF following daily HPF intake [37]. However, the small proportion of adolescents in the sample who experienced increased motivation to consume HPF had greater weight gain at 2-year follow-up [37].

Overall, a reasonably strong body of basic behavioral evidence suggests that on average, healthy children, adolescents, and adults may exhibit greater valuation of HPF relative to non-HPF and may seek out HPF in response to cues in the environment. Longitudinal evidence of the effects of HPF from repeated consumption over time is much more limited and has indicated that in response to repeated HPF intake over 14–21 days, some individuals may experience enhanced motivation to consume HPF (sensitization); however, it is unclear whether the effects may be observed overall or individuals who may be particularly sensitive to the rewarding effects of HPF. Thus, more work is needed, particularly longitudinal consumption studies to characterize the effects of HPF exposure on potential change in motivation to consume HPF over time, which may lead to habitual HPF intake.

Neurobiological Evidence (Humans)

A limited number of studies have examined the basic effects of HPF consumption on brain reward neurocircuitry (examined cross-sectionally) and neurobiological changes (examined longitudinally). Regarding cross-sectional evidence of HPF intake patterns and brain reward responsivity, most of the evidence has been indirect. For example, substantial evidence from cross-sectional studies has identified differences in brain reward responsivity by weight class or BMI [51]. In these studies, BMI or obesity status has been used as a proxy to indicate repeated/habitual HPF intake; however, most studies have not studied the association between HPF intake and brain reward responsivity explicitly. In this regard, in a recent meta-analytic review of the literature, researchers reported that individuals with obesity were found to exhibit greater responsivity to food cues in brain regions that govern reward and motivation, suggesting that individuals with obesity may exhibit sensitization to HPF cues [51]. Only three prior cross-sectional studies directly examined the association between HPF intake and brain reward responsivity, all of which were conducted among healthy samples of adolescents or adults. For example, the studies used fMRI paradigms and found that greater dietary intake of HPF or sugar was associated with greater responsivity to HPF cues in brain reward regions (dorsal and ventral striatum), suggesting sensitization to HPF [52], and reduced responsivity in striatal regions when consuming an HPF (e.g., a milkshake) [53, 54], thus indicating blunted reward responding during consumption. Overall, more work is needed to directly examine cross-sectional associations between HPF consumption and differences in HPF motivation and reward responsivity.

A small body of evidence from longitudinal studies suggests that neurobiological changes may occur among adolescents and adults from HPF or sugar intake over time. However, only one prior study measured intake, and the remainder assumed HPF intake through observed weight gain. Specifically, Burger et al. [55] provided experimental evidence that indicated that after consuming a sugar-sweetened beverage for 21 days, adolescents exhibited marked increases in motivation to consume the beverage when exposed to cues and decreases in response in reward regions (e.g., dorsal striatum) during beverage consumption in fMRI. The large effects sizes suggested sensitization to the sugar-sweetened beverage cues over time [55]. In addition, two repeated measures studies examined the association between weight or body fat gain, which was assumed to be a result of repeated HPF intake, and responsivity to HPF cues and consumption [56, 57]. Findings indicated that adolescents who gained body fat over a 3-year period exhibited increased striatal responsivity to HPF cues, suggesting increased motivation to consume HPF [56]. Furthermore, in a sample of adult women, individuals who gained weight over a 6-month period were found to have reductions in striatal responsivity to consumption of an HPF during an fMRI paradigm [57], suggesting blunted responsivity to HPF reward during consumption. In addition to these studies, three studies with healthy children [58] and adolescents [59, 60] identified greater responsivity to HPF cues in brain reward regions at baseline to be prospectively associated with weight gain, with the assumption that greater HPF intake yielded may have contributed to changes in weight observed longitudinally.

Overall very limited evidence has directly connected HPF intake to cross-sectional differences or longitudinal changes in HPF reward responsivity and motivation. Most research has used BMI or weight gain and assumed that HPF consumption was the underlying driver. More research is needed to directly examine the association between HPF intake and neurobiological changes. While not reviewed herein, the

animal literature presents compelling evidence that HPF and sugar intake may yield substantial neurobiological changes that are consistent with neurobiological changes observed with substances of abuse [61–64]. Thus, evidence in the human literature is needed to examine the basic connections between HPF intake and changes in reward neurocircuitry among healthy samples of children, adolescents, and adults, information that is key to understand potential consequences related to habitual/compulsive HPF intake, obesity, and food addiction.

In addition to a limited body of evidence, the existing literature is limited due to inconsistencies in terminology and definitions used to describe highly reinforcing foods. Prior studies in the behavioral and neuroimaging literature used a variety of descriptive definitions (e.g., fast foods) or terms (e.g., high energy density foods) when referencing foods that may be particularly reinforcing. Future work using a standardize definition of foods that have enhanced reinforcing properties is needed to bring concordance to the literature. Recently, Fazzino et al. [39] provided a standardized definition of HPF, which specifies quantitative thresholds for combinations of nutrients (fat, sugar, carbohydrates, and/or sodium) that are hypothesized to induce hyper-palatability and as a result may be highly rewarding to consume. The provision of this definition is a first step in facilitating research with a standardized definition, which may strengthen scientific inquiry and policy guidance. In addition, given that definitions of energy density and ultra-processed foods do not directly address a food's palatability (but do address other important food characteristics that may increase their reinforcing properties), it is important for future research to elucidate the degree to which the definitions identify foods that may be consistent with or may be vary in their reinforcing properties compared to HPF. Overall, the field has substantial work to do to better define and identify the specific characteristics of foods (e.g., ingredient combinations and food processing characteristics) that yield their excessive reinforcing properties, which may strongly influence our food approach and intake behavior.

Distinction Between HPF as Substance vs Individual Differences in Vulnerability to HPF

The present review focuses on the reinforcing properties of HPF, conceptualized as a substance. This premise is distinct from most of the literature, which has focused on identifying individual-level differences in vulnerabilities to the effects of HPF. Most research has focused on identifying subgroups or characteristics of individuals who may be vulnerable to HPF effects and may be at risk for developing obesity or food addiction. For example, ample work from Epstein and colleagues has established that individuals differ in the degree to which they value food as a reward and that individuals with high food reinforcement may be at greater risk for obesity [65–67]. In a similar lens of identifying individual-level vulnerabilities to HPF, Stice and Burger [68] presented a theoretical framework for obesity that identified individual-level differences in sensitivity to HPF cues and neurobiological changes from repeated HPF intake that may combine to increase obesity risk. Finally, Gearhardt and colleagues identified food addiction as a clinical phenomenon that is exhibited among a subset of individuals who may be particularly susceptible to the rewarding effects of HPF and who demonstrate clinically significant symptoms and impairment from HPF consumption [69, 70].

Overall, extensive work has been conducted in the field to identify individual-level vulnerabilities to the rewarding effects of HPF that may occur among a subset of individuals in the population. Importantly, HPF may be the substance that directly facilitates the observed effects and consequences. Thus, it is critical to gain a more thorough scientific understanding of HPF, defined using a standard definition, as a substance, in a similar manner as the field of addiction, which has conducted extensive research to understand the basic neurobiological and behavioral impacts of substance use (e.g., alcohol, nicotine, and opioids) on healthy individuals. Thoroughly understanding the neurobiological and behavioral changes that may occur from the intake of HPF is critical to developing prevention and treatment approaches that recognize the potency of the substance (HPF) and best address the target mechanism (HPF intake) that may yield clinical consequences such as obesity and food addiction among vulnerable individuals.

Hyper-Palatable Foods in the US Food Environment

Behavior Doesn't Occur in a Vacuum: The Food Environment

HPF are powerful reinforcers; however, use of a substance, or HPF in this case, is largely influenced by the surrounding environment. Choice is relative and depends on the availability of HPF, as well as the other alternatives in the environment. In the USA, the food environment is considered obesogenic and is structured in a manner that maximizes access to HPF and provides limited access to alternative foods (e.g., non-HPF such as fresh fruits and vegetables) [71, 72]. This environmental structure may exacerbate approach and consumption of HPF as normative eating behavior (passive overconsumption), as well as potentially pathological behavior (e.g., food addiction). Thus, the food environment is structured to exploit our natural behavioral tendencies to seek out convenience in HPF access, and HPF

themselves are designed to exploit our neurobiological proclivities to seek and consume highly rewarding foods. This picture is grim when considering the degree to which it may be possible to access and eat foods that do not excessively activate our brain reward neurocircuitry and the potential for population-level health consequences, such as obesity and food addiction.

How Did We Get Here? US Tobacco Company Involvement in the US Food System

The US food environment has changed dramatically since the early 1970s and may have been strongly influenced by the entrance of US tobacco companies to the food system. Below, changes in the food system are described first, followed by a review of tobacco company involvement in the food system. Before the 1970s, the food environment in the USA was largely supplied by smaller, local food producers, and regional companies [73]. However, in the 1970s and 1980s, larger food companies developed by absorbing smaller food producers and food companies and centralizing food processing and distribution efforts [74]. As a result, food companies maximized their profits by consolidating production expenses and distribution efforts while substantially increasing sales in the USA and globally [74, 75]. As food companies focused on minimizing costs, the companies also focused on developing food technology and creating less costly versions of ingredients, such as high fructose corn syrup, an inexpensive alternative to sugar [76]. As a result, the US food environment became saturated with inexpensive, easily accessible HPF [75, 77].

The changes observed in the food environment since the 1970s occurred alongside the entrance of a new player in the US food system, US tobacco companies. Leading tobacco companies in the USA, specifically Phillip Morris and RJ Reynolds, began investing in food companies in the 1970s and 1980s, which served to compensate for declining tobacco sales and increased federal regulation of tobacco products in the USA [78, 79]. By the 1990s, tobacco companies led the US food market in sales annually [80]. For example, tobacco company Phillip Morris entered the US food market in the early 1980s and bought two major food brands, Kraft and General Foods. By 1985, Phillip Morris had more than double the US market shares compared to any other food company [81], a position that was maintained through the 1990s [80]. By 1999, Phillip Morris brands led the market in sales of 17 out of 20 food product categories, such as frozen foods [78]. The investment in the food system was extremely profitable for Phillip Morris; sales from food products were approximately equal to sales of tobacco products between 1989 and 2001 [78, 82, 83] and helped buoy the company's profits during a time of intense litigation and profit loss from tobacco. Phillip Morris-developed food products are still very present in the food environment today. Kraft remains a leading food company and is owned by a now-parent company of Phillip Morris, Altria Group [83, 84]. Thus, Phillip Morris's influence and involvement in the food system has been a reliable and stable factor in our food system since the early 1980s.

The RJ Reynolds tobacco company had a similar trajectory of involvement in the food system, although their food products were specialized to specific market segments. RJ Reynolds initially bought into the US food system via the sugar-sweetened beverage market in the 1960s and expanded their ownership to include Nabisco, a major snack food company, in the 1970s [85, 86]. By 1986, sales of food and tobacco products for RJ Reynolds were approximately equal in the company's portfolio [79], suggesting food sales were a major contributor to RJ Reynolds revenue in the tobacco litigation era. By 1990, Nabisco was the largest manufacturer and marketer in cookie and cracker industry in USA, with market shares that were 2–3 times larger than their closest competitors [79]. Thus, RJ Reynolds also had a major impact in leading the US food system through the 1990s, with a specific focus on specialty snacks and sweetened drinks. However, by the late 1990s, RJ Reynold's market shares from food products dropped, and by the early 2000s, RJ Reynolds divested from Nabisco ownership, thus relinquishing its broader influence in the food system [87].

The influence of US tobacco companies in the food system has been extensive, and tobacco company leadership in the food system likely played a major role in shaping the current US food environment. However, the scientific evidence identifying the ways in which the tobacco companies influenced the food system is extremely limited. In two foundational studies that leveraged primary source documents provided by the University of California, San Francisco Industry Documents Library, Nguyen and colleagues revealed that both RJ Reynolds and Phillip Morris used the techniques they developed in tobacco product development, sales, and marketing to develop and market unhealthy food and beverage products to vulnerable populations in the USA, specifically children [88•], and racial and ethnic minority groups [89]. Thus, Nguyen and colleagues have provided the first pieces of evidence to suggest that tobacco ownership of food companies was likely problematic for public health. A missing piece of the puzzle is whether tobacco companies applied their techniques for creating addictive tobacco products to develop foods that may be difficult to stop eating and have addictive properties (i.e., HPF). Thus, another key piece of evidence needed to expand our understanding of the impact of tobacco company involvement in the US food system is a potential connection between tobacco ownership and HPF proliferation in the food environment.

This information is needed to fully conceptualize our food system, to consider ways in which HPF may be designed to exploit our neurobiology, and implications for addressing clinically relevant conditions, including obesity and food addiction.

Conclusion

HPF differ in key ways from naturally occurring foods and may exploit our neurobiological propensities to seek and consume rewarding foods. There is reasonably strong behavioral evidence to indicate that HPF may have reinforcing properties that are similar to drugs of abuse. Strong evidence indicates that healthy individuals may exhibit greater preference for and valuation of HPF relative to non-HPF, that individuals may seek out HPF for their rewarding effects, and that cues may develop that indicate the presence of HPF. All of these factors speak to the reinforcing properties of HPF. More limited evidence indicates that HPF intake may be associated with greater motivation to consume HPF (sensitization) and blunted experience of reward during HPF intake, which may be observed neurobiologically. More research is needed to understand and characterize longitudinal neurobiological and behavioral changes that may occur from repeated HPF intake over time among healthy human samples.

The US food environment has shifted dramatically since the 1970s and currently provides extensive access to HPF. The major changes in the US food environment may have been driven by US tobacco companies, which invested heavily in the US food system in the early 1980s. Thus, the leader of the US food system over the past 30 years has been US tobacco companies, which have specialized in creating addictive tobacco products, and applied their product development and marketing efforts to develop and sell food in the US food system [88•]. At this juncture, it is critical for researchers to investigate whether tobacco companies may have driven the design and proliferation of HPF in the food environment, which have marked similarities to other addictive substances. If this connection is established, it would provide a strong justification for the regulation of HPF at the federal level. Substances, such as alcohol and nicotine, are regulated in the US environment because it is recognized that they may have substantial detrimental effects on vulnerable groups in the population, including children and those with risk factors for substance use disorder. This same premise should be considered for HPF, which also have established harmful effects on individuals who are vulnerable to their rewarding properties (e.g., food addiction and obesity), and who may suffer an outsized health burden as HPF are allowed to proliferate the US food environment unregulated.

Compliance with Ethical Standards

Competing Interests The author declare no competing interests.

Human and Animal Rights and Informed Consent No human or animal subject data were used in the manuscript.

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