EATING DISORDERS (C GRILO, SECTION EDITOR)

Current Considerations Regarding Food Addiction

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Abstract "Food addiction" is an emerging area, and behavioral and biological overlaps have been observed between eating and addictive disorders. Potential misconceptions about applying an addiction framework to problematic eating behavior may inhibit scientific progress. Critiques of "food addiction" that focus on descriptive differences between overeating and illicit drugs are similar to early criticisms of the addictiveness of tobacco. Although food is necessary for survival, the highly processed foods associated with addictive-like eating may provide little health benefit. Individual differences are important in determining who develops an addiction. If certain foods are addictive, the identification of possible risk factors for "food addiction" is an important next step. Not all treatments for ad-

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diction require abstinence. Addiction interventions that focus on moderation or controlled use may lead to novel approaches to treating eating-related problems. Finally, addiction-related policies that focus on environmental (instead of educational) targets may have a larger public health impact in reducing overeating.

Keywords Food addiction \cdot Obesity \cdot Addiction \cdot Substance dependence

Introduction: Misconceptions About Food Addiction

The concept of "food addiction" is an area of growing scientific interest and ongoing debate. Animal models suggest that rats given highly palatable foods (e.g., icing, bacon, cheesecake) or intermittent access to sugar will display neurobiological and behavioral indicators similar to those observed in models of drug addiction [1, 2]. In humans, overlapping neurobiological systems (e.g., mesolimbic dopamine system) appear activated by both drugs of abuse and highly palatable foods [3•]. Individuals with addiction or eating-related problems (e.g., obesity, binge eating disorder) exhibit similar patterns of neural reactivity to drug or food cues, respectively [4•, 5]. Higher scores on the Yale Food Addiction Scale (YFAS) [6], which applies the diagnostic criteria for substance dependence to the consumption of highly palatable foods, are associated with increased body mass index (BMI), more frequent binge eating, greater impulsivity, and stronger cravings for fatty, processed foods [7]. Patterns of neural response and genetic profiles implicated in addiction have also been related to higher YFAS scores [8, 9]. Although these findings are important and provide support for food addiction as a clinically relevant concept, more scientific inquiry is needed before the "food addiction" concept can be either accepted or rejected and applied routinely in clinical settings. A major obstacle to

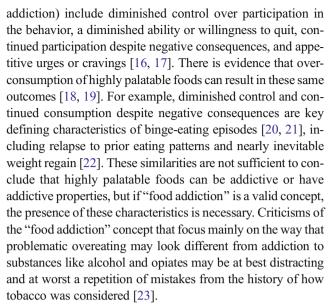


evaluating whether an addictive process contributes to problematic eating may involve potential misconceptions about what an addiction perspective means when applied to eating behavior. In the current paper, we will address many of these notions and highlight important gaps that should be addressed to empirically evaluate whether an addictive process contributes to problematic eating behavior.

Lessons Learned From Tobacco

The term addiction may raise the image of a person "addicted" to an illicit drug who is experiencing intoxication or withdrawal, contributing little to society, engaging in illegal activities, and at-risk of overdose. Yet, this view of addiction does not accurately reflect most addictions nor does it resonate with arguably the most common and deadly addiction in our society—tobacco addiction. Cigarette smoking is a leading cause of preventable death in the USA [10]. Yet, tobacco smoking does not lead to noticeable intoxication, has a mild physical (although not psychological) withdrawal, is legal, readily accessible, and causes little impairment in one's daily life (e.g., work, caring for oneself, raising children). For decades, critics of the addictiveness of nicotine (the ingredient identified as habit-forming or addictive in tobacco) pointed to these differences between tobacco and other drugs (e.g., alcohol, opiates) as evidence that cigarettes could not be addictive [11]. For example, the British opiate-addiction researcher, Sir Humphrey Rolleston was quoted as saying, "That smoking produces a craving for more when an attempt is made to give it up... is undoubted, but it can seldom be accurately described as overpowering, and the effects of its withdrawal, though there may be definite restlessness and instability, cannot be compared with the physical distress caused by withdrawal in morphine addicts. To regard tobacco as a drug of addiction is all very well in a humorous sense, but it is hardly accurate [12]." Critics also suggested that applying the term addiction to a legal substance that had little risk of severe intoxication, withdrawal, or overdose would reduce the meaningfulness of the addiction label [13]. Such perspectives delayed the recognition of tobacco as an addictive substance, which hindered the development of more effective treatment and prevention approaches.

Many critiques of the concept of "food addiction" highlight descriptive differences as the case with tobacco and addiction noted above [14, 15]. Overconsumption of highly processed foods does not lead to intoxication, a life-threatening withdrawal syndrome, or the threat of overdose. Food is easily accessible, socially permissible, and legal. However, we have learned from tobacco that millions can become "addicted" to a substance with these characteristics. Core components of addiction across substances and highly reinforcing behaviors (e.g., consider gambling, a non-substance "behavioral"



Concerns about diminishing the meaning of addiction have also been raised in the debate about "food addiction" [24]. Yet, like with tobacco, excessive consumption of calorie-dense, highly processed foods is an important factor relating to preventable death in the USA [10]. Overconsumption of these foods may not lead to overdoses, life-threatening withdrawal, or illegal behaviors, but may lead to premature deaths. Akin to tobacco, minimizing the significance of potentially addictive foods (or food additives) may miss the substantial public health consequences of a legal, cheap, advertised, available, and potentially addictive substance.

Can You Be Addicted to Something You Need to Survive?

Another possible critique of the concept of "food addiction" is that food is necessary for survival and therefore cannot be addictive. This seemingly accurate criticism is not without flaws. First, people can become addicted to something even if it contains components necessary for survival. For example, water is completely necessary for us to survive. Humans are at risk of dying from dehydration within 3 days of going without water [25]. There is no evidence of an addictive process developing to water even though thirsty people may find drinking water very rewarding [26]. In contrast, the addition of an addictive ingredient to water is sufficient to give it an addictive potential. For example, beer can be up to 97 % water, but it is addictive due to the presence of ethanol. Thus, a substance once necessary for survival (e.g., water) can become addictive with the addition of certain ingredients.

Similar to water, food consumption is necessary for our survival. Nutrient-rich foods, such as fruits, vegetables, and lean meats, are essential to our ability to exist. Yet, processed foods with high levels of added refined carbohydrates (e.g., sugar) and fats provide little health benefit and are linked to



diseases including diabetes, cancer, and heart disease [27]. Theories of "food addiction" have focused on highly palatable, highly processed foods (e.g., ice cream, chocolate, pizza, French fries) as those most likely types of foods to trigger an addictive response [28, 29]. Hedonic desire, not homeostatic need, is a major motivator for consumption of these types of foods [30]. For example, sugar-sweetened soft drinks are composed largely of water, but sugar, caffeine, and flavor enhancers are added to create a beverage that is not consumed for health benefits, but for pleasure. These highly processed foods are more likely to trigger cravings [31••] and bingeeating episodes [32] compared to more nutritious, non-processed foods. Thus, food is necessary for survival, but the types of foods most strongly implicated in "food addiction" are not.

Yet, at this point, there is limited empirical research that has focused on identifying which foods or food characteristics might trigger processes that are potentially related to those in addictions. An addiction perspective suggests an interactive effect between an individual's predisposition for developing an addiction and an addictive agent [33-35]. Further, addictive substances are capable of triggering neuroplastic changes in reward-related brain regions that may contribute to compulsive use [36–38]. While it is unknown whether certain foods or nutritional characteristics (e.g., sugar, food additives) are capable of triggering addiction-like responses in humans, it appears that highly processed foods may share features with drugs of abuse. The addictive potential of a drug of abuse is increased when the dose, or amount, of the addictive agent is highly concentrated and is rapidly absorbed into the central nervous system [39, 40]. For example, cigarettes allow for a high dose of nicotine to be quickly transmitted into an individual's central nervous system [41]. Similarly, some foods are processed to have unnaturally high levels of highly rewarding ingredients (e.g., fat, refined carbohydrates). Additionally, the refined carbohydrates can be rapidly absorbed into the system, which results in a greater blood sugar spike [42-44]. Thus, like drugs of abuse, highly processed foods contain an elevated dose of potentially addictive food characteristics, such as refined carbohydrates, which are rapidly absorbed in the body and may rapidly exert central effects.

Limited research has examined which food attributes may trigger an addiction-like process. Some studies have found that foods high in sugar activate the reward system in a manner similar to drugs of abuse, whereas foods high in fat activate the somatosensory system [45••]. This appears to corroborate evidence in animal models demonstrating markers of an addictive process in response to sugar, such as opiate-like withdrawal and continued use of sugar, but not fat, despite negative consequences like foot shock [46]. While it has been suggested that "food addiction" may reflect an "eating addiction" [47], researchers have observed that binge-prone rats do not demonstrate addictive-like eating behaviors in response to

their chow, even when the chow is administered intermittently or in times of stress [48, 49]. Thus, examining whether certain foods or food characteristics have an addictive potential is an essential next step for evaluating the "food addiction" concept.

Individual Differences are Important in Addiction

Another possible misconception is that all people who use an addictive substance will become addicted to it. This is false. For example, the majority of people drink alcohol during their lifetime, but only 5-10 % become addicted to alcohol, depending on the threshold used for defining cases of addiction [50, 51]. Similarly, only 16.7 % of cocaine users appear to become addicted [52]. Therefore, individual differences are important in determining who does or does not become addicted to a substance. If certain highly processed foods have an addictive potential, one might not expect everyone who eats them to become addicted. However, addictive substances that are legal, cheap, and easily accessible are related to widespread subclinical problems that result in high public health costs [23]. Thus, if certain foods (or food additives) are addictive, a large segment of society may experience subclinical responses that result in significant public health consequences.

Multiple factors have been implicated in increased addiction risk, such as a family history of addiction [53], motivations to engage in the behavior to cope with negative emotions [54], impulsivity [55], and genetic alleles associated with reward dysfunction [56]. Interestingly, all of the individual differences have also been implicated in problematic eating behaviors [57–60]. Other components of addictive disorders, such as individual differences in sensitization risk and expectancies about outcomes of use, have arguably received less attention in the context of eating problems. If these factors are similarly implicated in compulsive eating behaviors, this could provide further support than an addiction-like process is involved.

Expectancies, or beliefs about the outcomes of use/engagement, can affect addictive behaviors. Positive expectancies about the outcomes (e.g., use will enhance positive or reduce negative emotions) have been related to increased drinking and drug use [61–63]. Additionally, individuals with an addiction to alcohol report more positive alcohol expectancies compared to non-problem drinkers [64]. Expectancies about the affective outcomes of eating may be similarly related to problematic eating behavior. Similar to the consumption of alcohol, the consumption of food may be performed to enhance positive or reduce negative emotions. Overeating, and especially binge-eating, is often preceded by negative affect [65–68], which may suggest that individuals expect eating to relieve these negative emotions. Studies have found negative reinforcement eating expectancies to be related to binge-

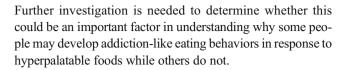


eating behaviors and to symptoms of bulimia nervosa [69–71]. Given the role of expectancies in other addictions, expectancies may represent an important factor in distinguishing individuals likely to develop addiction-like eating behaviors.

The incentive sensitization theory of addiction may further explain why some individuals develop addictions while others do not. In this theory, intermittent access to drug rewards leads the neural regions involved in reward processing (particularly those associated with dopamine) to become sensitized to drug-associated cues [72, 73]. This sensitization leads to compulsive use by increasing "wanting" in response to drug-related cues. The cues acquire greater incentive salience (i.e., the ability to enhance motivation to obtain the reward). Notably, sensitization is not necessarily associated with increased "liking," or the pleasurable subjective effects of the reward, and elevated "wanting" is associated with increased use even with no increase in "liking" [74].

While much existing research on incentive sensitization has been done in the context of drug rewards, it has been suggested that incentive sensitization may also occur in response to highly palatable foods [75, 76]. Like drug rewards, palatable foods appear to activate neural pathways implicated in assigning reinforcing value to rewards, such as the mesolimbic dopamine system [73, 76, 77]. Animal models have shown this system to be related to "wanting" without "liking" of sweet rewards [74, 76]. However, little research has examined whether incentive sensitization occurs in human eating behavior. A few studies have attempted to assess "liking" and "wanting" for food rewards, but there is disagreement on the best way to measure these or even whether they are separable in humans [78–80]. Without a methodological consensus, it is difficult to compare findings across studies.

It is important to note that not all individuals appear to be equally prone to sensitization to rewards and their cues. Studies using animal models have shown individual differences in cue responsivity, identifying rats as being "sign-trackers" versus "goal-trackers" [81, 82]. Sign-trackers appear to be particularly responsive to a reward's associated cues, while goaltrackers appear to be more attuned to the reward itself. Signtracking animals may approach or engage with the cue itself, for example by trying to lick or eat a light that has been associated with a food reward. Goal-tracking animals, on the other hand, will, upon seeing the associated cue, go to the location where the reward or "goal" is expected to appear. Signtracking appears to be associated with impulsivity and may increase vulnerability to compulsive or addictive behaviors [82, 83]. Additionally, in a state of induced hunger, signtracking rats show more robust reinstatement of foodseeking behavior compared to goal-tracking rats [84]. While there is some evidence that increased cue responsivity is related to elevated consumption in human eating behavior [85], sign- and goal-tracking remains understudied in humans.



Is Abstinence the Only Treatment for Addiction?

Another possible misconception about "addictions" is that all addiction treatments require abstinence. While this is the case for many common treatment approaches (e.g., 12-step programs), empirically supported interventions exist in which abstinence is not required. For example, harm reduction, which aims to reduce the harm associated with addictive behaviors and increase the individual's quality of life, has been found to be effective without a treatment goal of abstinence [86–88]. A major concern about the application of an addiction approach to eating behavior is that it would require patients to abstain from all "addictive" foods, which may lead to more disordered eating [89]. Indeed, there currently exist multiple "addiction-based" eating programs that focus on the goal of not consuming certain food groups (e.g., Food Addicts Anonymous, Overeaters Anonymous), and the effectiveness of such programs has received almost no research study. In sharp contrast, effective cognitive-behavioral therapy (CBT) and behavioral interventions for overeating and binge-eating [90] focus on moderation rather than avoidance or abstinence from foods. The documented effectiveness of such CBT approaches is not at odds with many of the concepts reviewed here regarding potentially addiction-like processes or properties associated with foods. Rather, like various evidence-based methods for controlling, for example, alcohol misuse, such CBT methods use structure and coping methods to normalize eating and reduce vulnerability to disrupted eating in response to external/internal cues. Additionally, modified CBT-based interventions may help individuals with co-existing eating and substance use problems and individuals who struggle with food cravings and high responsivity to food cues [91–93].

Education to Combat Addiction

A possible policy misconception about addiction is that education is a particularly effective approach to reducing problematic use. For example, while education about alcohol abuse through school-based programs and public information campaigns are popular by public opinion (and remain widely used), these initiatives have been found in multiple instances to be ineffective at inducing long-term behavioral changes [94–96]. In contrast, environmental changes, such as increasing price and decreasing availability, have been effective at reducing negative outcomes associated with substance abuse (e.g., overdose, crime, driving under the influence), but these



strategies are not uniformly well received by the public [97–99]. When nicotine was identified as an addictive substance, there was an increase in policies that restricted or limited use, such as emphasizing clean-air acts, implementing a tax on nicotine products, and using media to educate about smoking consequences [100]. Thus, if certain foods (or food products or food additives) are associated with addiction-like behaviors, a similar increase in policy may warrant examination. Based on the history of other addictive substances, if certain foods are identified as "addictive," investments may be more effective if they are directed to environment-focused policies, such as taxation or restricted marketing practices, which may be more successful at inducing long-term change than education initiatives.

Conclusions

In sum, the question of whether "food addiction" is a valid concept is an empirical question. Potential misconceptions about applying an addiction perspective to problematic eating may be an impediment to scientific progress. Critiques of "food addiction" that emphasize descriptive differences between addictive-like eating and the consumption of intoxicating or illegal drugs may be repeating mistakes that delayed the identification of tobacco as an addictive substance. Important next steps include the evaluation of whether specific foods (or food additives) may be addictive and the examination of individual factors that may increase the risk of "food addiction." If an addictive process contributes to problematic eating, applying addiction treatments that focus on moderation (rather than abstinence) to food consumption may lead to novel and more effective intervention approaches. Finally, policies that focus on environmental (instead of educational) targets may have a large public health impact in reducing excessive consumption of potentially addictive foods.

Compliance with Ethics Guidelines

Conflict of Interest Erica M. Schulte and Michelle A. Joyner declare that they have no conflict of interest.

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