

Symposium Overview—Food Addiction: Fact or Fiction?^{1–3}

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Abstract

Food addiction is a pervasive, yet controversial, topic that has gained recent attention in both lay media and the scientific literature. The goal of this series of articles is to use a combination of preclinical and clinical data to determine whether foods, like drugs of abuse, can be addictive, the conditions under which the addiction develops, and the underlying neurophysiological substrates. Operational definitions of addiction that have been used in the treatment of human disorders and to guide research in both humans and animals are presented, and an overview of the symposium articles is provided. We propose that specific foods, especially those that are rich in fat and/or sugar, are capable of promoting “addiction”-like behavior and neuronal change under certain conditions. That is, these foods, although highly palatable, are not addictive per se but become so following a restriction/binge pattern of consumption. Such consummatory patterns have been associated with increased risk for comorbid conditions such as obesity, early weight gain, depression, anxiety, and substance abuse as well as with relapse and treatment challenges. The topic of food addiction bears study, therefore, to develop fresh approaches to clinical intervention and to advance our understanding of basic mechanisms involved in loss of control. J. Nutr. 139: 617–619, 2009.

Food addiction: A pervasive, yet controversial, concept

The concept of “food addiction” has gained recent attention in both lay media (~334,000 hits on Google) and in the scientific literature. Food addiction has been implicated in craving, bingeing, and obesity. It has been used to support the avoidance of foods as diverse as carrots (1), cheese, and chocolate as well as foods containing specific compounds such as sugar and casomorphins [see Wadman (2) for an overview]. Treatment of food addiction has even been used as a strategy in the treatment of eating disorders (3). The concept of food addiction, then, is pervasive.

Although pervasive, the concept of food addiction is also controversial. Thus, although anecdotal reports abound, few studies have attempted to determine the “addictive” properties of foods using rigorous scientific criteria. One of the problems, of course, is that everyone eats. How can a label of “addictive” be applied to that which supports life itself? Rather than assume that all foods are addictive, we propose that some foods are

more addictive than others, especially foods rich in fat and/or sugar. These typically are the foods that become forbidden, are those on which people binge, and are often energy dense. Even so, we also propose that these foods, although highly palatable, are not addictive, per se, but become so following a restriction/binge pattern of consumption.

How to define addiction in humans

To define addiction, we turn to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV-TR), in which criteria for substance dependence are defined. Sweet and fatty foods are not considered “substances” in the classical sense. Substances are typically interpreted as drugs of abuse such as cocaine, ethanol, heroin, etc. For a person to be considered dependent on (or addicted to) any given substance, at least 3 of the following 7 criteria must be met at any time within a given year: 1) tolerance (more drug is needed for the same effect), 2) withdrawal, 3) taking a larger amount of the substance or taking the substance for a longer period than was intended, 4) experiencing a persistent desire for the substance or an inability to reduce or control its use, 5) spending much time seeking or consuming the substance or recovering from its effects, 6) use of the substance interfering with important activities, and 7) use of the substance continuing despite known adverse consequences. If either criterion 1 or 2 is met, then *physiological* dependence is diagnosed. However, a diagnosis of substance dependence does not require that criterion 1 or 2 be met. Thus, human substance dependence (or addiction) can be diagnosed using entirely behavioral criteria (4).

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Is there evidence for food addiction in humans?

A key feature of any addiction is loss of control. The elemental behavior in a food addiction is the consumption of food; loss of control is manifest by either more frequent and/or larger meals. A meal that is larger than would normally be consumed in a given period of time under similar circumstances is a binge. Although not labeled as such, several of the diagnostic/research criteria for the bingeing-related eating disorders approximate criteria for substance dependence (4). These include the following: 1) binge-type consumption (taking the substance in larger amounts than was intended); 2) loss of control over eating (inability to control its use); 3) bingeing occurs at least twice a week for 3 mo in bulimia nervosa or for 6 mo in binge eating disorder (taking the substance for a longer period of time than was intended); 4) for bulimia nervosa, bingeing is followed by inappropriate compensatory behavior such as vomiting, laxative use, enemas, etc.; for binge eating disorder, bingeing is accompanied by disgust, embarrassment, guilt, and marked distress (use continues despite adverse consequences). A diagnosis of bulimia nervosa or binge eating disorder, then, could be considered to fulfill at least 3 of the criteria for substance dependence. In addition, although craving is not considered one of the diagnostic criteria for the bingeing-related eating disorders, craving has been associated with binge eating (5), and food craving activates areas of the brain reported to be involved in drug craving (6). Thus, both behavioral and neurobiological evidence support the conclusion that food, under conditions discussed more fully by Pelchat (6), can induce an addiction-like state in human subjects. Finally, it is important to note that comorbidities associated with binge behavior, such as obesity, early weight gain, depression, anxiety, and substance abuse, as described by Avena et al. (7), make the potential negative impact of this behavior on individuals and on society substantial.

Food addiction: Animal studies and underlying substrates

Although human studies are critical to our understanding of the addiction process, animal studies are crucial to our understanding of basic mechanisms that underlie that process. Work such as that done by Ann Kelley, to whom this symposium was dedicated, and her colleagues has clearly shown how homeostatic mechanisms involved in feeding interface with those involved in reward and addiction (8). Two of the articles from this symposium describe work in which rat models were used to examine basic mechanisms common to the consumption of food and drugs. Lutter and Nestler (9) describe how withdrawal from a high-fat diet leads to neurochemical responses comparable to those induced by withdrawal from drug (9). They also describe how affective states such as depression, a key feature of drug withdrawal, can modify endogenous signals involved in food intake regulation. Avena et al. (7) provide convincing evidence that bingeing on sugar can induce behavioral and neuronal changes in rats similar to those induced by drugs of abuse. In addition, Pelchat (6) discusses how external stimuli, such as cues, can modulate food seeking and food intake in important ways via classical conditioning. All of these articles illustrate the fact that food, at least in part, acts on the same neuronal systems as do drugs of abuse.

How to define addiction in animals

As with the study of human behavior, one challenge in the study of animal behavior is to determine when the transition from use to addiction has occurred. Because addiction in humans is primarily defined behaviorally, any claim of "addiction" in animals would be well served by the demonstration of clear behavioral

differences between addicted and nonaddicted subjects. Over the past several years, operational criteria have been developed for rats self-administering cocaine that parallel the DSM-IV-TR criteria (10–13). In addition to tolerance and withdrawal, the following behavioral criteria have been proposed: 1) escalation of intake across time (taking a large amount; loss of control); 2) willingness to work harder to get the substance (high motivation to obtain and consume the substance); 3) greater responding during periods when the drug is not available (increased time spent seeking the drug); and 4) increased responding despite aversive consequences (use continues despite adverse consequences).

Is there evidence for food addiction in animals?

Although several reports have used the proposed rat behavioral criteria when studying addiction to drugs, few have systematically applied similar criteria when studying addiction to food. Work from our group has made use of some of these criteria to demonstrate that addiction-like behavior (escalation of intake, increased willingness to work for fat or cocaine, increased cocaine seeking) can be induced in rats with a history of bingeing on fat (14–16). Moreover, we found that use of a more restricted access schedule to fat led to more addiction-like behavior in rats. The article by Avena et al. (7) describes strong evidence for addiction-like behavioral and neurological alterations in rats bingeing on sugar and how some of these effects differ from those that occur when rats binge on fat.

Summary and conclusion

Taken together, the articles from this symposium provide evidence that neurological similarities exist in the response of humans (6) and rats (7,9) to foods and to drugs. Two of the reports (6,7), as well as our own work (14–16), suggest that even highly palatable food is not addictive in and of itself. Rather, it is the manner in which the food is presented (i.e., intermittently) and consumed (i.e., repeated, intermittent "gorging") that appears to entrain the addiction-like process. Such consummatory patterns are associated with increased risk for comorbid complications as well as relapse and make treatment particularly challenging. The topic of food addiction bears study, therefore, to develop fresh approaches to clinical intervention and to advance our understanding of basic mechanisms involved in loss of control.

Other articles in this supplement include references (6,7,9).

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