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Symposium—Food Addiction: Fact or Fiction?

Food Addiction in Humans[1–3]

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Abstract

Most of the evidence for or against food addiction in humans focuses on similarities between food craving and drug craving. There are numerous parallels in neuroanatomy, neurochemistry, and learning. Indeed, brain mechanisms for craving probably evolved to promote seeking of natural rewards and are taken over by drugs of abuse. Healthy, normal weight individuals, by definition, do not suffer from food addiction; however, overweight and obese individuals could meet clinical criteria. Palatable foods are not responsible for the obesity problem, because even nonpalatable foods can come to be desired and potentially overconsumed. It may be the way in which foods are consumed (e.g. alternating access and restriction) rather than their sensory properties that leads to an addictive eating pattern. J. Nutr. 139: 620–622, 2009.

Introduction

This is a brief review of the concept of food addiction in humans. Most of the evidence for or against addiction (now called dependency in the Diagnostic and Statistical Manual of Mental Disorders) focuses on similarities and differences between food craving and drug craving. If food is addictive then other questions arise such as: "Is food then bad for us? Is palatable food particularly bad for us? Should we then outlaw extremely palatable food? Will the obesity epidemic go away if we all stick to gruel?" Drug addicts crave, but do not enjoy, a drug (1). In such discussions, the terms liking, wanting, and reward are frequently used. Yet there tends to be a great deal of confusion over the definitions of and the relations among these terms. Liking is defined as the hedonic response to a stimulus. Liking is synonymous with pleasantness or the evaluative response to a stimulus. We take wanting to mean desire and will discuss evidence showing that it is possible to want a stimulus without liking it. Craving is simply a very strong desire. Reward, or reinforcement, has traditionally had a rather circular definition as that which increases the probability of the behavior that precedes it. It is often assumed that rewards are pleasurable, but see below for evidence to the contrary. Pleasure may be rewarding, but other reinforcers such as glucose in the gut may be rewarding without the conscious experience of pleasure. A reasonable working hypothesis is that, going in the other direction, reward enhances desire for and pleasure derived from a stimulus. Liking and wanting differ from hunger in that they have specific objects of reference. Indeed, during craving (i.e. strong desire) functional MRI (fMRI) recordings show strong activation of sensory-memory–related brain areas (2). Hunger has traditionally been the focus of studies on obesity. So, many question the practical value of understanding desire for a specific food, or craving. However, evidence has recently been building that craving and related phenomena do predict intake. Binge eating/bulimia have long been associated with food craving (3). Cravings are associated with more snacking and less compliance with dietary restrictions (4) and also predict higher BMI (5). In 1954, Olds and Milner (6) reported on brain sites that, when electrically stimulated, were extremely rewarding and led to very high rates of bar pressing in rats. So these were clearly reward "centers," but the assumption that wanting and liking were one and the same was so strong that they were frequently called "pleasure centers." In the 1960s a handful of patients with intractable brain diseases were implanted with such electrodes. The assumption was that the stimulation must be pleasurable, because the patients reported that they felt compelled to continue pressing the button for stimulation (7). Yet their self-reports were that the stimulation felt strange, not pleasant.

Wanting/liking distinction

The most commonly cited example of the wanting/liking distinction is that drug addicts report that they continue to crave their drug long after they have stopped enjoying it (1). Although craved foods are generally also liked, there are some examples of a wanting/liking distinction for food as well (8). The purpose of the study was to determine whether nutritional deprivation was necessary to produce food craving. The experimental manipulation was to place subjects on a nutritionally adequate but boring and restrictive diet, a vanilla-flavor dietary supplement beverage, for 5 d. By the end of the monotony phase of the study, subjects reported that they did not find the beverage to be particularly palatable and there was, indeed, a large increase in

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frequency of food cravings during this period. So nutritional deprivation is not necessary to produce food cravings. An unexpected outcome of the study was that a few of the subjects reported craving the beverage during the week in which they returned to baseline eating even though they did not like it.

Neurochemistry

A study of the neurochemistry of reward provides a great deal of evidence for similarity between food and drug cravings. Two of the major players in the reward circuit are dopamine and the endogenous opiates. Cocaine use causes release of striatal dopamine [see (9) for a recent review] and in a raclopride binding study, Small et al. (10) demonstrated that the same is true for food. Drug abuse is associated with decreased sensitivity of the dopamine-reward system (9). The same is true in obese individuals (11). Wang et al. (11) used raclopride binding to measure density of D2 receptors and found an inverse relationship between D2 receptor density and BMI. Of course the direction of causality for these differences in sensitivity to dopamine is not known. Is it a result of repeated overstimulation of the system? Or is it a preexisting risk factor for obesity or drug abuse? Although dopamine is rewarding, its depletion or blockade doesn't diminish pleasurable responses to palatable foods in animals or humans (12). So dopamine may play a more important role in wanting than in liking.

Endogenous opioids also play a role in the reward circuit. Release of these transmitters leads to higher levels of striatal dopamine, that is, to what we think is rewarding. And indeed, the rewarding properties of alcoholic beverages (13) and of sweet (or palatable) foods (14) are thought to be mediated by this pathway. However, opioids also play a role in pleasure. Naltrexone, an opioid blocker, reduces short-term food intake, but more importantly, this effect may be limited to palatable foods. Naltrexone treatment does lead to reduced pleasantness ratings for foods, but it does not appear to affect hunger [see (15) for a review].

Neuroanatomy

Anatomy is another point of comparison between food and drug cravings. Although there have been numerous brain imaging studies on subjects who were thinking about food (16), few of them have made an attempt to limit the images to craving. In an fMRI study by Pelchat et al. (2), great care was taken to isolate craving-related activation from activation related to hunger or liking. Two groups of subjects were studied. One was on a nutritionally adequate but monotonous diet for 1.5 d before imaging and the other group consumed an unrestricted diet with sampling of the monotonous diet so that they would become familiar with it. During the scanning session, all subjects were asked to imagine 2 foods that they "really liked a lot" (these foods were, of course, temporarily forbidden to the members of the monotonous diet group). These blocks alternated with blocks during which the subjects imagined the monotonous diet or rested. Imagination was used rather than food videos so that the participants could imagine ideal versions of their favorite foods. Behavioral results were quite conclusive. All participants in the monotonous diet group experienced cravings when imagining the liked foods and no one experienced cravings when imagining the monotonous diet. Success at turning craving on and off in 30-s bins was probably due to the fact that subjects were given a task that was incompatible with craving (imagining the monotonous diet) rather than simply being asked to stop craving.

The areas of craving-specific activation were identified through a double subtraction process. They included the caudate nucleus, hippocampus, and insula. First, blocks in which subjects imagined the monotonous diet were subtracted from those in which they imagined liked foods. This subtraction eliminated general processes related to imagining food, leaving areas of activation related to thinking about liked foods and/or thinking about craved foods. To procure a more craving-specific pattern, images for the normal diet group (who reported only a smattering of food cravings) were subtracted from the images for the monotonous diet group (all of whom experienced cravings). Craving-related changes in fMRI signal were identified in the hippocampus, insula, and caudate, 3 areas reported to be involved in drug craving (17,18) Thus, this work supports the common substrate hypothesis for food and drug cravings. The prominent representation of memory and sensory integration structures in this study is consistent with the central role of sensory memory in the experience of food cravings. It is as if, when craving, one has a sensory template of what has to be eaten to satisfy the craving.

Learning

Food and drug cravings may also be learned in similar ways. Conditioning effects have been used to explain the once puzzling compulsion to use drugs long after withdrawal (19). The classic example is that a highly motivated individual goes through withdrawal and finishes rehab with flying colors. He sets forth into the outside world with no intention of relapsing. Yet, when he returns to the old neighborhood and old friends, he finds himself craving and using drugs even though he is no longer experiencing withdrawal symptoms. The usual explanation is that cues in the environment that were associated with drug use had triggered the craving. Just as withdrawal is not necessary to produce drug cravings, nutritional deprivation is not necessary to produce food cravings (8). Environmental cues are effective triggers for food craving as well; the sight or smell of food or even food imagery may serve as triggers (2,20,21). Gibson and Desmond (22) reported that craving may be an acquired response based on repeatedly eating the craved food when hungry. Relatively little is known about learning mechanisms in food craving and this could be a fruitful area for future investigations.

Conclusion

There are many parallels between food and drug cravings in humans and in animals (see other articles in this session). Do these parallels demonstrate that food is addictive? Of course, that depends on the definition of "addictive." Two of the criteria mentioned in the Diagnostic and Statistical Manual of Mental Disorders are tolerance and withdrawal and there is evidence for food withdrawal and tolerance in animals (23,24). However, many of the clinical criteria for addiction/dependence focus on the consequences of continued use or on failure to discontinue use. If there are no negative consequences of eating food and there are no failed attempts to discontinue eating large amounts or certain types of food, there is no diagnosis of addiction. On this basis, most healthy, normal-weight people would not be diagnosed as food addicts and food would not be considered an addictive substance, because, for the most part, it produces positive rather than negative consequences. Drugs of abuse are different from food. The shared neural substrates for food and drug cravings probably evolved to encourage healthy behaviors such as eating and reproduction (25). Drugs of abuse are undesirable because they are able to take over these substrates and to divert efforts away from healthier goals.

However, overweight or obese individuals probably do meet the clinical criterion for food addiction (e.g. persistent desire or repeated unsuccessful attempts to quit; important social, occu-

pational, or recreational activities given up or reduced; continued use despite knowledge of adverse consequences). It may be worthwhile to consider some parallels between responses to food and to alcohol; just as some individuals can drink alcohol responsibly and others cannot, there are individual differences in reactions to food (due to genetic predisposition or to prior experience) and some people can consume food in moderation more easily than others.

It is sometimes asserted that obesity rates have increased because there is too much palatable food available. However, although palatability does increase intake in the short term, it is not clear that palatable food leads to overeating over the long term (26). Indeed, naltrexone treatment, a pharmacological manipulation that does lead to reduced palatability ratings, is not associated with increased weight loss (15,27). Furthermore, subjects in a monotonous diet study learned to crave a not-very-palatable dietary supplement beverage (8). Thus, food does not need to be palatable to be craved.

The Corwin and Hoebel (23,24) articles suggest that alternating restriction and availability of food, i.e. the way in which the food is used rather than its sensory or nutritional properties, may produce the addictive pattern of eating. Going on and off diets may be the human analogue to the restricted access paradigm.

There are many parallels between feeding behavior and drug addiction. Treatments for drug abuse focus on craving, impulsivity, and learning and are not generally focused on withdrawal or other physiological measures of addiction. In contrast, many, if not most studies of obesity focus on minimizing hunger. But not all diet failures are due to hunger; some of them are probably due to nonhomeostatic eating or impulsive eating. Given the many parallels between food and drug cravings, it would make sense to use lessons from drug addiction to aid in the fight against obesity.

Other articles in this symposium include references (23,24,28).

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