Food Addiction?

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Public and professional acceptance that use of drugs and alcohol could lead to physical and psychiatric disease states, that is, substance use disorders, required enormous clinical and research efforts. Now, some feel that this hard earned status is threatened by discussions suggesting that gambling, sex, and food may result in similar outcomes.[1,2] However, the manner by which research was used to establish the addicting characteristics of drugs and alcohol must be the gold standard against which further considerations are made. In terms of the question of gambling, much of this work has been accomplished with studies demonstrating striking similarities between gambling and exogenous addictions. Even more valuable lessons have been learned regarding the processing of odds, assessing risk, and decision-making deficits evidenced in both behavioral and neuroimaging studies of compulsive gamblers and patients with substance use disorders.[3]

Through all of the debate and as a result of systemic study, we have learned that addiction is an acquired, chronic relapsing disorder that begins with some experimentation and pleasurable responses, and for subgroups of individuals is followed by preoccupation, escalation, tolerance, denial, a series of medical, psychologic, and social consequences that relate directly to the continued use, and what has been referred to as a "fatal attraction" between the substance (or activity, eg, gambling) and the patient.

Obviously, the process resulting in addiction is not fulfilled in all or even most users. There are a number of host or individual factors, such as genetics, intrauterine exposure, early childhood, and adolescence experience, which influence susceptibility and/or resilience. Furthermore, pharmacological factors such as route of administration are also important. To describe coca leaf chewing as cocaine taking does little to capture the intensity of coca paste or cocaine freebase or crack smoking. Technologic inventions have also been applied to drugs of abuse making them more reinforcing, compelling, and addicting. Finally, the same drug can be used by the same person but the experience is different. For example, nicotine when administered through cigarettes or smokeless tobacco is highly addicting. Yet, nicotine patches and gum are so unappealing that many State programs give them away.

Drugs of abuse or gambling are not, however, a part of everyday life and survival. Food and sex are and therefore pose particularly difficult questions for clinicians and researchers including what separates periodic excessive intake (ie, during periods of vacation or special holidays) from abnormal or pathologic intake, and to what extent must life activities be compromised to meet diagnostic criteria. Fundamentally, we must ask if we can apply standards used in defining neurobehavioral changes associated with recognized addictions to the issues of food and sex, as well. If we can, do we have a choice to recognize the potentially addicting capacities of food and sex?

For these "survival-related" addictions, the degree of access and availability may be essential in creating the opportunity for addiction. For sex, "high-speed," rather than slower "dial-up," universally accessible pornography has provided world-wide access and

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created the need for treatment across the globe.[4] Similarly, fast food companies have created foods that are widely available, highly palatable, low volume, low cost, high fat, "high carb," easy to eat and enjoy—food consumed so quickly that satiety has no time to intervene. Fast food companies thrive because they are able to provide foods that stimulate additional intake and do so with minimal inconvenience using quick "drive-thrus," 24-hour service, and with relatively little impact on family spending. Typically, their food experience is compelling and they have addict-like customers who eat there every day or many days a week.

Salt has been proposed as a significant mediating factor toward the development of obesity, with mu opioid activity having a possible role in food-associated pleasure (Cocores and Gold, submitted for publication). It follows, although more data are needed, that daily use of these types of foods can produce dependence. If so, strong urges for salted food may reflect some level of opiate receptor withdrawal. When a patient with sexual addiction thinks about sex or the patient with food addiction thinks about their favorite food or restaurant or restaurant logo, priming doses of dopamine are released in the nucleus accumbens.[5] Furthermore, highly recognizable food trademarks can trigger brain changes similar to those triggered by drug or drug paraphernalia when shown to a patient with substance use disorder.[6] The neurobehavioral outcome may be noted by changes similar to those of drugs and alcohol.

As we anticipate the next edition of the *Diagnostic and Statistical Manual of Mental Disorders*, the need to reconcile current thinking and data becomes increasingly important and we ask, can overeating become a pathologic attachment to food and what the media has coined "food addiction"? The easy answer is why not? The more difficult answer is what is the evidence supporting this view? Clearly, obesity has become a world-wide problem in a short time, a time sufficiently short that genetic changes cannot fully explain the outcomes.[7] Importantly, the brain reinforces saving and storing of food to be prepared for famine. Perhaps, the interaction between our saving, self-preservation, and famine proclivity genes and the bias toward consumption in an environment of abundance may account for some of the increase.

Satiety or the lack thereof may also contribute. For example, satiety for drugs is difficult to demonstrate, in fact, satiety for cocaine may be an oxymoron.[8] Stopping use is more likely related to financial or physical consequences than satiety, per se. Responding to satiety or fullness after eating is also inconsistently experienced. The brain says "eat" and leaves it to the stomach to say, "that seems like enough." This relationship was beneficial in earlier human development when food sources were scarce and foraging required enormous individual energy, but parameters no longer apply and entire days' worth of calories can be consumed between the fast food drive-thru and the office. The consumption process is initiated and concluded before stomach distention or physiologic feedback can be activated. Previous energy demands associated with searching, killing, cleaning, and preparing food is now reduced to walking the aisles of megastores where endless options await.

Eating meets not only physical but also psychologic needs. It is typically pleasurable, relaxing, and restorative. It is a social lubricant, facilitates personal and professional interactions and is often the centerpiece of significant celebrations. It is not surprising then that the logos, cartoons, and trademarks associated with food become potent stimuli, automatically eliciting mental and physical approach responses. Interestingly, drug abusers as well as patients who describe themselves as "food addicts" refer to their object seeking as hunger or cravings. The object of desire is different but the event is experienced similarly.

We have learned a great deal about drug reinforcement and brain changes and have looked at lessons from addiction medicine and neuropharmacology and applied these to food, overeating, and food addiction. Food and addiction for exogenous substances have been linked through personal stories, direct observation, and empirical studies.[9,10] Weight loss with drug use, including nicotine, and weight gain during abstinence are common observations and are often (at least anecdotally) linked to relapse. Although the mechanism(s) is poorly understood, it is widely accepted, particularly in self-help programs, that hunger often associated with relapse, and within reason, should be avoided.[11] Such observations were the extent of the data and support for food as a substance of abuse and pathologic attachment as one cause of the obesity epidemic at the first ASAM Symposium on this subject 15 years ago. This year in Toronto, the database had grown considerably. We continue those discussions in this issue.

For example, Gearhardt et al have shown that elements of substance dependence can be applied to relationships with food. Their work suggests that food addiction can be operationally defined and rating scales can be applied to identify relatively homogenous groups of hedonic overeaters.[12] People eat (and overeat) for a variety of reasons—some because of anxiety, [13] grief,[14] or the sight or smell of food may trigger persons to eat, even if they aren't hungry. Further, because of genetics and environment, children tend to have eating behaviors or attitudes that are similar to their parents or others in their immediate household. Merlo et al observed this phenomenon in a study of overweight and obese adolescents and their parents/guardians. Their cohort frequently self-reported symptoms of "food addiction," eg, emotional eating, uncontrolled eating, and eating attitudes and behaviors similar to their parents.[15]

Prader Willi Syndrome (PWS), a genetic disorder and one of the best naturally occurring human models of obesity, is reviewed by von Deneen et al.[16] PWS children have extreme hunger and overeating. They hoard food and may eat nonfood objects.[17] Food-related cues (ie, food images, scents, or viewing actual items) may play a role in PWS as fMRI data suggest that eating is over-reinforcing in these children. They continue seeking food despite satiety. It is likely that this phenomenon extends to other subgroups of children as well. However, genes are only one part of the story. What a mother eats during pregnancy and what an infant or child eats may also be important in gene expression and induction of obesity liability. Food preferences begin at infancy, thus future studies in food addiction should be focused on this population.

Hoebel et al[18] reported that sugar has addictive properties similar to psychostimulants and opioids. They recently confirmed that there is evidence of sugar addiction,[19] and that sugared high-fat foods contribute most to weight gain. Wang et al,[20] using PET and other techniques, showed that the brain responds to food similarly to the way in which it responds to drugs. An increase in extracellular dopamine due to food cues in an environment is accompanied by a relative lack of dopamine 2 receptors. This down-regulation, similar to cocaine or alcohol addictions, could lead to increased food-seeking and an inability to control consumption. Such a relationship is also evident in those who gain weight when taking certain antipsychotics.[21,22]

Some signs we might associate with food addiction include continuing to overeat even though it may harm your health, family or social life; eating in secret; feeling compelled to finish all the food in your line of sight; and even eating to the point of discomfort. The presence of a psychiatric disorder and the use of medication may exacerbate the condition. Furthermore, the repercussions of obesity such as stigma, hypertension, and the rising risk of diabetes among other preventable illnesses increase the negative impact of obesity. Pischon et al[23] linked resultant adiposity – especially in the abdominal region – with increasing risk of death and thus the time to address our tainted relationship with food is now.

Leptin, ghrelin, orexin, galanin, neuropeptide Y, and cocaine-and amphetamine-regulated transcript, among others are messengers that may play a role in drug and food dependence and impact the direction of pharmacologic interventions.[24,25] However, the most effective current evidence-based treatment for obesity is not pharmacological or psychosocial but surgical.[26] Bariatric surgery for morbid obesity and even simple obesity with medical comorbidities is the fastest growing surgery in the United States and expanding with minimally invasive lap banding and outpatient procedures.

Although other therapies are in the pipeline, the importance of exercise and nutrition should not be underestimated. However, fad diets are not the answer, and even certain foods we deem as "healthy" can have negative repercussions. For example, restriction to a "low carb" diet leads to carbohydrate craving and overeating and is unhealthy. It is best to eat good carbohydrates from old-fashioned oatmeal, wild rice, legumes, and low glycemic index and fructose index produce (relatively high glycemic index whole wheat flour is not included). Low fat foods including low omega 3, 6, and 9 from unheated extra virgin olive oil can actually wreak havoc on the body and brain. Too much highly processed protein (including soy and whey) may be cardiotoxic and if minimally processed can lead to osteoporosis, not to mention an increased risk of cancer due to excess free radical bombardment. Overall, a reduction in food intake or intake of only minimally processed foods may be most beneficial.

It is challenging to give a DSM-diagnosis to any process addiction. Drugs of abuse have been extremely well studied in laboratory models/animals for many years. The addicted rat is a well-known model and studies have helped us understand the complex neurobiology and develop new treatments.[27] Human substance use disorders have also been well studied and their characteristics defined. Treatments and consensus have been developed that addiction is a disease. No such consensus exists for process addictions. However, progress has been made in building a human experimental model for gambling, and compulsive gambling seems to be gaining professional acceptance.[28] Carnes and Schneider[29] proposed that sexual compulsivity is an addictive disorder. However, animal models do not exist and what is compulsive and what is sex has led to controversy. The advent of high-speed internet and the emergence of compulsive pornography watching with masturbation seem to be a new addictive disease of the 21st century. Compulsive sexual behavior, pathologic gambling, and hedonic overeating are important problems, but are they addictions? In the absence of a blood, urine, or imaging test or an animal model, controversy will reign. However, clearly some patients have relationships with great tasting/looking food that mimic a DSM diagnosis for substance dependence. We have seen many patients who have lost control of their eating, eat more than they intended, eat to pain or discomfort, eat despite life-threatening and shortening medical or negative consequences; patients who are unable to abstain from eating certain foods; patients who have made countless attempts to reduce intake to no avail; patients who spend a great deal of time seeking, using, or recovering from food consumption. Further, in the face of a continued obesity epidemic, we cannot ignore the possibility that food (or at least certain food constitutes or types) can promote loss of control and continued compulsive use despite consequences.

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