

Food Addiction: Clinical Reality or Mythology



In the last 5 years, there has been an astonishing interest in the notion of food “addiction.” Medline has seen a sevenfold increase in the number of papers indexed by the term “food addiction” since 2008.¹ The ideology of food addiction posits that foods that are eaten frequently become substances of abuse and could cause health problems for the consumer if the affected individual suddenly discontinued the food in question. This “withdrawal” is often described as the resulting “hangover,” and craving is equated with a reaction that could only be mitigated by eating a further portion of the “addictive food.”

The persistence and escalating intensity of the public debate about the potentially addictive quality of sugar may be one of the most remarkable social phenomena of the new millennium. Authoritative headlines proclaim the “Perils of Sugar” from the covers of health and fitness magazines (for example, *Muscle & Performance*²; or “Junk Food Masks Reveal The Nightmarish Depths Of Sugar Addiction,”³ from sites such as *The Huffington Post*.

Consumers are warned that the food industry offers them “a seemingly endless supply of sugar grenades that provide mega shots of sweeteners.”² It is also asserted, in the same article, that “just as morphine or cocaine addicts need extra hits in order to feel an effect,” a typical consumer can begin to “crave” more sugar to feel satisfied. The equation of sugar “addiction” with drug or medication dependence and addiction now runs throughout both popular and general medical literature alike,⁴ especially because neural reward pathways “light up” with the various research imaging modalities that demonstrate activation with sex, drugs, rock-and-roll, and a good workout.⁵

According to the American Society of Addiction Medicine,⁶ addiction is a highly complex primary, chronic disease of brain reward, motivation, memory, and related circuitry. Dysfunction in associated neurochemistry leads to characteristic biological, genetic, psychological, social, and spiritual manifestations. It is pointed out that differences in addiction and intoxication, and the interaction between

neuropeptides and other hormonal axes with dopamine and opioid activity, are very challenging biopsychosocial processes.

The fact of physiologically violent withdrawal syndromes in response to the abrupt discontinuation of certain drugs of abuse and dependence both phenomenologically and neurobiologically differentiate behaviors such as a craving for sweets or training for bicycle racing from, for example, opioid addiction.

Consider opiate dependence. Redmond and Krystal⁷ reviewed the medical conception of the opiate withdrawal syndrome as it relates to the sudden abstinence from opiates. They reviewed the clinical data that describe withdrawal varying with specific agent, dose, and duration of exposure. They described a syndrome that includes hot and cold flashes, piloerection, diaphoresis, febrile illness, myalgia, arthralgia, severe colic, nausea, sometimes-severe emesis, diarrhea, lacrimation, rhinorrhea, anxiety and restlessness, dysphoria, insomnia, tachypnea, tachycardia, and hypertension. Kosten and O’Connor⁸ point out that initial symptoms of [opiate] withdrawal are similar for those who have become addicted to alcohol, sedatives, and stimulants.

Because of the broad phenomenon of receptor desensitization or “tolerance” at the receptor level, most addicts fairly rapidly reach a point where they become slaves to acquiring and consuming the substance to which they are addicted, NOT to “get high,” but to avoid withdrawal, which is beyond aversive for most, and which renders the addict incapable of functioning. The Public Policy Statement of the American Society of Addiction Medicine⁶ states:

Persons with addiction compulsively use even though it may not make them feel good, in some cases long after the pursuit of “rewards” is not actually pleasurable. Although people from any culture may choose to “get high” from one or another activity, it is important to appreciate that addiction is not solely a function of choice. Simply put, addiction is not a desired condition.⁶

Longstanding clinical wisdom describing the emergence of life-threatening complications during withdrawal defines and declares the distinctly serious nature of withdrawal. Consider delirium tremens in alcohol withdrawal; cardiac instability, and suicidal ideation or psychosis in patients withdrawing from stimulants; exquisitely painful myalgias, colicky pain, and watery explosive diarrhea in opioid

Funding: None.

Conflict of Interest: None.

Authorship: All authors had access to the data and played a role in writing this manuscript.

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withdrawal. Any individual who is debilitated, volume depleted, or suffering with comorbid conditions such as cardiac disease or diabetes may experience life-threatening complications in the first 72 hours of withdrawal, and the risk may persist for up to 14 days.

Clearly, even the most abrupt abstinence from habitual or excessive intake of sweet foods or repetitive behavior that may represent the popular conception of addiction does not meet the withdrawal criteria for true substance-driven addiction as articulated in the present model. Herein lies the major weakness of pencil-and-paper psychometric instruments such as the Yale Food Addiction Scale (YFAS) questionnaire.⁹

The most significant threat to validity with survey instruments like the YFAS is that they rely on subject self-report and self-assessment of items that require endorsement or rejection of phenomena such as “withdrawal,” which is likely to be subjectively misunderstood. Ziauddeen and Fletcher¹⁰ characterize a limitation of the YFAS in that it dichotomizes complex and subjective symptoms such as anxiety, agitation, and withdrawal.

Even in the face of excess consumption and over prolonged periods, dietary nutrient cessation simply does not appear to produce the same kind of overt or cellular pathology as prolonged administration of agents that are truly addictive. The syndrome of withdrawal that is recognized upon discontinuation of a substance that induces dependence is not seen in any valid or reliable investigation of acute abstinence from a macronutrient, or for that matter from the discontinuation of any habit or serially occurring behavior that results in pleasure, reward, or arousal.

The American Society of Addiction Medicine Public Policy Statement⁶ repeatedly emphasizes that the diagnosis of addiction requires a comprehensive biological, psychological, social, and spiritual assessment by trained and certified professionals. The American Psychiatric Association, in the *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*,¹¹ utilizes, in our view, a problematically broad definition of addiction (termed “substance dependence”) as a maladaptive pattern of substance use leading to significant impairment or distress as manifested by only 3 or more of any of 11 criteria occurring in a 12-month period.

In further effort to simplify the challenging distinctions among craving, abuse, dependence, and addictive behaviors and substances, the DSM-V¹² created a continuum from mild to severe, which seemed to obfuscate rather than simplify distinctions among pleasurable compulsions, craving, abuse, dependence, and addictive behaviors and substances. In what appears a final effort to respond to the fusion and confusion of substance-based and non-substance-related addictive disorders as described in DSM-V,¹² Hebebrand et al¹³ proposed the term “eating addiction” in an effort to distinguish certain behavioral patterns rather than imply specific neurophysiologic correlates of drug addiction.

The problems and weaknesses of discussion to date may be captured best by Ziauddeen & Fletcher,¹⁰ who conclude that the perspective of any food addiction is “not sustainable” in neurobiologic terms. Their 2012 article with Farooqi¹⁴ presents matrices that demonstrate that proposed phenomenology of food addiction does not meet severity and impairment thresholds for substance dependence. Moreover, they present detailed summary data of the functional neuroimaging literature that fails to support any single analytically illuminating view of overeating, much less food addiction.

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References

1. Pai N, Vella S, Richardson K. Is food addiction a valid phenomenon through the lens of the DSM-5? *Aust N Z J Psychiatry*. 2014;48(3):216-218.
2. Kadey M. Sugar shock. *Muscle Perform*. 2014;(September):55-61.
3. Frank P. Junk food masks reveal the nightmarish depths of sugar addiction. *Huffington Post*. August 25, 2015. Available at: http://www.huffingtonpost.com/2014/08/25/james-ostreer-_n_5689709.html. Accessed May 7, 2015.
4. Avena NM, Gold MS. Food and addiction—sugars, fats and hedonic overeating. *Addiction*. 2011;106(7):1214-1215.
5. Blum K, Werner T, Carnes S, et al. Sex, drugs, and rock ‘n’ roll: hypothesizing common mesolimbic activation as a function of reward gene polymorphisms. *J Psychoactive Drugs*. 2012;44(1):38-55.
6. American Society of Addiction Medicine. Definition of addiction. Available at: <http://www.asam.org/for-the-public/definition-of-addiction>. Accessed March 27, 2015.
7. Redmond DE Jr, Krystal JH. Multiple mechanisms of withdrawal from opioid drugs. *Annu Rev Neurosci*. 1984;7:443-478.
8. Kosten TR, O’Connor PG. Management of drug and alcohol withdrawal. *N Engl J Med*. 2003;348:1786-1795.
9. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale food addiction scale. *Appetite*. 2009;52(2):430-436.
10. Ziauddeen H, Fletcher PC. Is food addiction a valid and useful concept? *Obesity Rev*. 2013;14(1):19-28.
11. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-IV)*. 4th ed. Arlington, VA: American Psychiatric Association; 1994.
12. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders (DSM-V)*. 5th ed. Arlington, VA: American Psychiatric Association; 2013.
13. Hebebrand J, Albayrak Ö, Adan R, et al. “Eating addiction”, rather than “food addiction”, better captures addictive-like eating behavior. *Neurosci Biobehav Rev*. 2014;47:295-306.
14. Ziauddeen H, Farooqi IS, Fletcher PC. Obesity and the brain: how convincing is the addiction model? *Nat Rev Neurosci*. 2012;13:279-286.