

Food addiction: a critical reflexion

Ignacio Basurte,^{1,3} Nestor Szerman^{1,2,3}

Editorial

When the DSM-5 came out, substance use disorders (SUDs) were reclassified as substance-related and addictive disorders. Meanwhile, because gambling behaviours activate reward systems in a similar way to drugs of abuse, and given that they cause symptoms that overlap with those produced by SUDs, gambling disorder was included in the DSM-5 as the only non-substance-related addictive disorder.¹ Scientific evidence for including other addictive behaviour disorders such as internet addiction, sex addiction, or compulsive buying in this category was still too weak.

Nonetheless, with the changes to the DSM-5 chapter on feeding and eating disorders, it was acknowledged that some individuals report symptoms resembling those typically endorsed by people with a substance use disorder, such as craving and compulsive use. The resemblance may reflect the involvement of the neural systems associated with self-control and reward. However, the distinctive and shared factors between food addiction and substance use disorders are yet to be fully understood.¹

For similar reasons, obesity was not included in the DSM-5, despite the resemblance of the behaviours and the involvement of the same central pathways that are implicated in SUDs.² From a dimensional perspective, there is a spectrum of overeating, with overindulgence at one extreme, and at the other a "pathological" urge to consume highly rewarding foods.³ But we are seeing a paradigm shift, which will undoubtedly have an influence on future classifications, now that the term "addictive disorder" has entered the categorical system for non-substance-related addictive disorders and has left the door open for other addictions without substances to be included.

Addictions are currently within the scope of mental health thanks to the concept of dual disorders.^{4,5} This idea is relatively recent though, since addictions were once considered to be an indication of a lack of willpower – an idea that often led to stigmatisation of those who suffered from them.⁶ This old concept was traditionally applied to obesity,

failing to take into account factors such as individual vulnerability, the dysfunction of endogenous brain circuits that implicate neuropeptides with rewarding properties associated with the very act of eating, or the availability of certain foods that could be highly rewarding.

That is why the concept of dual disorders may help to make sense of this new behavioural addiction paradigm, and in the case of food addiction could introduce individual vulnerability as a criterion, since vulnerability might increase the likelihood of developing an eating disorder.

INDIVIDUAL VULNERABILITY, RISK FACTORS AND DUAL DISORDERS

According to the current DSM-5 psychiatric classification scheme, there is a phenomenological overlap between substance-related and addictive disorders, and feeding and eating disorders,¹ in that self-control plays a prominent role in the criteria for disorders within these two categories.

In relation to pathological eating behaviours, it has been suggested that these may be the result of a loss of ability to regulate the consumption of highly processed foods containing refined carbohydrates, fats, salt, and caffeine. However, while the lack of such ability is an objective factor in SUDs, this is not so clear in eating disorders, since obesity is not necessarily associated with a loss of self-control, nor does an eating disorder or bulimia nervosa necessarily entail obesity. For example, recent studies show that only 41.5% of obese people with an eating disorder meet the food addiction threshold.⁷

From an evolutionary perspective, animals naturally pursue rewards such as water, sex, and food, and pathology emerges⁸ when there is dysfunction in the reward circuits. Current research also suggests neurobiological similarities between obesity, binge eating disorder, and SUDs. This may suggest that advances in the field of SUDs could lead to improvements in the treatment of eating disorders.⁹

¹ University General Hospital Gregorio Marañón. Madrid. Spain.

² Retiro Mental Health Centre. Madrid. Spain.

³ Spanish Society of Dual Pathology (SEPD, in Spanish).

Correspondence: Nestor Szerman. Centro de Salud Mental Retiro. Hospital General Universitario Gregorio Marañón. c/ Lope de Rueda 43, 28009 Madrid, España. Tel: +34 91400 – 8690. Fax: +34 91573 – 9932. E-mail: nszerman@salud.madrid.org

Several studies have demonstrated that patients with SUDs can show high rates of co-occurring disorders. Likewise, obese patients have shown high prevalence of attention deficit/hyperactivity disorder, depressive disorder, and bipolar disorder as co-occurring.¹⁰ From a genetic point of view, it could be that there is a common genetic susceptibility that might increase the risk of developing SUDs and any other mental illness, including an eating disorder. Nevertheless, no heritability studies have been carried out on food addiction. Studies that have been undertaken suggest that in compulsive-addictive eating patterns, the presence of associated psychopathology could be a key vulnerability factor just like in SUDs.^{7,11}

DOES FOOD ADDICTION REALLY EXIST?

The term 'food addiction' is now part of everyday language. The fact that it has become a widespread term in the scientific world is to some extent due to the use of the inappropriately-named *Yale Food Addiction Scale* (YFAS),¹² which actually focuses on the assessment of the addiction model in eating behaviour according to DSM-IV criteria as opposed to food addiction.

Labelling a food or nutrient as addictive implies that it contains or possesses an inherent property that can make susceptible individuals develop an addiction to such said nutrient or food, as is the case with drugs of abuse. Certain foods have rewarding and reinforcing properties; for example high sugar-high fat combinations that are rewarding for humans and rodents alike. It has been suggested that the recent increase in the prevalence of obesity reflects the emergence of food addiction in a significant fraction of the population.^{10,13,14} However, with the exception of caffeine,¹ there is insufficient scientific evidence at present to label any common food, micronutrient, standard food additive, singular ingredient, or combination thereof as addictive *per se*.¹⁵

The diet of subjects who overeat typically contains a broad range of different, subjectively palatable foods and does not indicate an addiction to a specific food or nutrient. We still have no evidence to show that a specific food can account for humans developing an eating disorder, although research does point to certain foods producing addictive behaviours in susceptible subjects, similar to the behaviours found in gambling disorder.¹⁵

Appropriate research still needs to be carried out to phenotypically characterise subjects with food addiction in

an effort to describe this disorder in clinical terms and to eventually produce specific diagnostic criteria. We perceive the simultaneous need for, and difficulty in clearly separating the causes of overeating, which could be qualified as behavioural addiction without knowledge of the underlying process. In the same way, and in light of the polygenic basis of body mass index variation in the general population,¹⁶ we need studies to address whether overeating and obesity can occur independently of a genetic predisposition to being overweight.

REFERENCES

1. American Psychiatric Association. American Psychiatric Association. DSM-5 Task Force. Diagnostic and statistical manual of mental disorders : DSM-5. 5th ed. Arlington, Va.: 2013; XLIV; pp947.
2. Volkow ND, O'Brien CP. Issues for DSM-V: should obesity be included as a brain disorder? *Am J Psychiatry* 2007;164(5):708-710.
3. Carlier N, Marshe VS, Cmorejova J, Davis C et al. Genetic Similarities between compulsive overeating and addiction phenotypes: A case for "food addiction"? *Curr Psychiatry Rep* 2015;17(12):96.
4. Marín-Navarrete R, Szerman N. Repensando el concepto de adicciones: pasos hacia la patología dual. *Salud Ment* 2015;38(6):395-396.
5. Szerman N, Martínez-Raga J, Peris L, Roncero C et al. Rethinking dual disorders/pathology. *Addictive Disorders Their Treatment* 2013; 12(1):1-10.
6. Roncero C, Diaz-Moran S, Comin M, Miquel L et al. Quality of life concept evolution: the subjective dimension as a relevant factor on drug addiction. *Vertex* 2011;22(99):343-349.
7. Vanbuskirk KA, Potenza MN. The treatment of obesity and its co-occurrence with substance use disorders. *J Addict Med* 2010;4(1):1-10.
8. Gearhardt AN, White MA, Masheb RM, Morgan PT et al. An examination of the food addiction construct in obese patients with binge eating disorder. *Int J Eat Disord* 2012;45(5):657-663.
9. Volkow ND, Wang GJ, Fowler JS, Tomasi D et al. Food and drug reward: overlapping circuits in human obesity and addiction. *Curr Top Behav Neurosci* 2012;11:1-24.
10. Gearhardt AN, Corbin WR, Brownell KD. Preliminary validation of the Yale Food Addiction Scale. *Appetite* 2009;52(2):430-436.
11. Gearhardt AN, Grilo CM, DiLeone RJ, Brownell KD et al. Can food be addictive? Public health and policy implications. *Addiction* 2011;106(7):1208-1212.
12. Davis C, Curtis C, Levitan RD, Carter JC et al. Evidence that 'food addiction' is a valid phenotype of obesity. *Appetite* 2011;57(3):711-717.
13. Rossetti C, Halfon O, Boutrel B. Controversies about a common etiology for eating and mood disorders. *Front Psychol* 2014;5:1205.
14. Blum K, Thanos PK, Gold MS. Dopamine and glucose, obesity, and reward deficiency syndrome. *Front Psychol* 2014;5:919.
15. Hebebrand J, Albayrak O, Adan R, Antel J et al. "Eating addiction", rather than "food addiction", better captures addictive-like eating behavior. *Neurosci Biobehav Rev* 2014;47:295-306.
16. Hinney A, Hebebrand J. Polygenic obesity in humans. *Obes Facts* 2008;1(1):35-42.