Article:
Finlayson, GS orcid.org/0000-0002-5620-2256 (2017) Food addiction and obesity: unnecessary medicalization of hedonic overeating. Nature Reviews Endocrinology, 13. pp. 493-498. ISSN 1759-5029

https://doi.org/10.1038/nrendo.2017.61

Opinion

Food addiction and obesity: unnecessary medicalization of hedonic overeating

Graham Finlayson

Abstract

The concept of addiction is loaded with connotations and often used for its political as much as its medical utility. The scientific case for 'food addiction' as a clinical phenotype currently rests on its association with generic diagnostic criteria for substance-related disorders applied to everyday foods and eating-related problems. This has fused the concept of obesity with addiction regardless of whether it fits the definition. The hedonic/reward system can account for ingestion of foods and drugs, confirming that they share neural substrates that differentiate liking and wanting. These are normal processes recruited for natural homeostatic behaviours and can explain the phenomenon of hedonic overeating as a consequence of human motivation pushed to extremes by an obesogenic environment. Food addiction constitutes a medicalization of common eating behaviour, taking on the properties of a disease. Use of this medical language has implications for the way in which society views overeating and obesity.

Controversy over the concept of 'food addiction' and its viability as a distinct clinical disorder is being fuelled by misconceptions on both sides of the debate[1,2]. Much of the confusion has stemmed from academic commentary debating the status of food addiction in the context of obesity[3-5]. Importantly, food addiction is not a validated concept at the moment; it is not approved by either of the two leading classification systems for diagnosing mental diseases, which include all medically recognised sub-types of substance use disorders and eating disorders. No clear scientific proof in humans that certain biochemical properties in foods are addictive exists. Agreement is also lacking on what symptoms of food addiction a patient might present with in the clinic that would distinguish them from the defined clinical symptoms of recognised aberrant forms of eating (that is, binge eating disorder, bulimia nervosa and anorexia nervosa). Several major critical reviews have given detailed criticisms examining neurobiological[4,6], phenomenological or phenotypic[7,8,75] and psychometric or diagnostic[9] deficiencies that are damaging for the concept of food addiction. Some researchers have expressed concern that the concept invites the medicalization of natural motivational needs[10,11]. Indeed, no strong evidence exists to substantiate the existence of food addiction that cannot be adequately explained through normal (biopsychological) adaptations to unhealthy lifestyles shaped by powerful socio-cultural pressures from the modern (obesogenic)

environment. And yet the food addiction concept persists. If one conducts an internet search of the term 'food addiction', they will find over 12 million results on self-diagnosis, treatment and support for this unfounded condition and over one-third of these results make explicit reference to obesity. In the scientific literature, ardent advocates assert the existence of food addiction in reviews and commentaries, which are often uncritically accepted as evidence. Several conflicting accounts now exist to describe numerous potential aetiological pathways and a range of clinical manifestations attributed to food addiction. A clear definition and test of the validity of food addiction is badly needed. In the meantime, some of the misconceptions that persist around food addiction need to be dispelled, and these misconceptions should be replaced with reasoned biopsychological argument drawing on evidence for the role of appetite and the hedonic (or reward) system in natural homeostatic eating patterns, pushed to extremes.

The purpose of this Perspectives is to critically evaluate the current status of the food addiction hypothesis and to scrutinise its relevance for understanding patterns of eating behaviour associated with obesity or disordered eating. A particular challenge is to address some of the inconsistencies in academic discussion surrounding the meaning of food addiction. Here, it is necessary to briefly distinguish scientific and non-scientific uses of the concept and to consider its status in relation to contemporary definitions of addiction. Researchers who advocate food addiction usually adopt a narrow definition of addiction based on substance-related disorders as described in the Diagnostic and Statistical Manual of Mental Disorders (DSM; **Box 1**). Therefore, attention will be given to the value of this diagnostic-centred approach for understanding what food addiction might be, and whether a screening tool based on associations between eating-related problems and generic criteria for substance-use disorders is an appropriate platform for considering the existence of food addiction. Finally, the two key assumptions that underpin the food addiction theory (that certain foods carry specific biochemical or physiological properties that make them analogous to addictive drugs; and that certain individuals can develop harmful patterns of ingesting these foods with distressing clinical symptoms, which makes them analogous to patients with a substance use disorder) will be examined in relation to explanations drawing on existing knowledge of the biopsychology of hedonic eating and its functioning in an obesogenic food environment.

[H1] The biomedical approach

Contemporary changes in the food, physical activity and socio-economic environments have resulted in a doubling in the worldwide incidence of obesity since 1980[12] and predicted rates of >50% adult obesity in the UK and USA within the next 2-3 decades[13,14]. Considerable progress in understanding and managing obesity has been possible due to the predominance of the biomedical approach. This

approach is integral to the work of clinicians, has been adopted by researchers and policy makers and has widespread acceptance by the public. At the heart of the biomedical model is the gathering of empirical evidence by scientific observation. Within the past 20 years, advances in human neuroscience have revealed the importance of the hedonic, or reward, system in accounting for regulated and dysregulated patterns of eating behaviour in conjunction with neural, behavioural and metabolic factors associated with the obese state[15,16]. A further element inherent to the biomedical approach is the classification and diagnosis of disease to identify aetiologies and administer specific treatments. This aspect is more controversial than other aspects of the biomedical approach because it requires a consensus based on interpretations of existing evidence, demonstrable clinical utility and consistency with prevailing political and cultural attitudes.

In the case of obesity, many researchers, clinicians and organisations support its recognition as a disease (American Medical Association, WHO and The Obesity Society)[17,18,19]. However, it is acknowledged that this recommendation is primarily based on its utility for focusing resources into obesity prevention, treatment and research[17,18]. Binge eating disorder is now well-established as a clinical entity that is distinct from obesity, and its inclusion in the Diagnostic and Statistical Manual of Mental Disorders, fifth edition[34] (DSM-V) as a 'feeding and eating disorder' has been important for raising awareness that this phenotype of disordered eating in obesity should be understood as a clinical condition with a distinct neuro-behavioural profile, which therefore has specific treatments[20].

[H1] The 'food addiction' hypothesis

Within the past 10 years, a new biomedical disease model for overeating has been suggested based on neurobiological theories of addiction and the application of generic clinical diagnostic criteria for substance dependence (DSM-IV-TR) and substance related and addictive disorders (DSM-V) to everyday foods and eating-related problems. The founding hypothesis for this model can be detailed as follows: the biochemical properties within certain common foods have the potential to cause an addictive process, leading to a typical range of addiction-related problematic behaviours that in some individuals are sufficient to cause clinically significant impairment or distress. Thus, the theory rests on two key assumptions: firstly, that some foods warrant classification as addictive substances; and secondly, that some individuals are prone to become addicted to such foods. Advocates of food addiction have used these assumptions to strongly argue that the availability of designated 'addictive' foods should be regulated or restricted by responsible policy and legislation in the food environment[21], and that people who might have food addiction should be diagnosed, investigated and treated[22]. However, two crucial pieces of evidence for the substantiation of the food addiction

hypothesis are missing: no addictive biochemical property within foods has been identified and no clinical syndrome for food addiction has been defined.

Nevertheless, the concept of food addiction has attracted the attention of clinicians and researchers, partly enabled by political and cultural pressures in response to the continued prevalence of obesity in modern society. Health professionals are using the language of addiction in a metaphorical sense in the management of obesity (for example, 'relapse', 'triggers' and 'craving-control'), which increases its use by patients with obesity[23] and endorses the unscientific belief in food addiction held by the majority of the public[24]. In the absence of a clinically defined syndrome or diagnostic thresholds, findings from studies on genetics, personality traits and human neuroimaging, as well as animal models, are being misused to imply the existence of food addiction, based on similarities between substance-use disorders and binge eating disorder[25]. Most importantly, a growing number of researchers are using an ad hoc tool that is claimed to quantify food addiction to generate 'prevalence' estimates in clinical and non-clinical samples and to correlate food addiction 'severity' with known risk factors for obesity and/or substance-use disorders[9]. Consequently, the discussion of food addiction in the media, frequently in relation to obesity and addiction to hard drugs such as heroin and cocaine, is shaping public attitudes towards certain foods (as being on a par with addictive drugs), and to food addiction being incorrectly adopted as a 'scientifically endorsed' explanation for obesity[10].

The assertions of some commentators that food addiction is a new category of psychiatric disorder (or brain disease) have provoked a steadily rising accumulation of concerns and counterpoints[2,6-9,11,16,26-29]. The central issue is that by prematurely propagating the idea of food addiction before it has undergone appropriate scientific scrutiny, scientists and proponents of food addiction are unfortunately conveying a spurious sense of understanding to the public and each other. This form of unilateral explanation for overeating is adding confusion to the description of obesity, a condition for which rational and meaningful explanations already exist[30]. Moreover, by over-pathologising common experiences of problematic eating and weight control, the popularity of the food addiction hypothesis could diminish the experience of individuals with specific food-related issues.

[H1] The meaning of addiction to food

A logical place to begin in a critical evaluation of the food addiction hypothesis is with the problems brought about by the use of the term 'addiction' itself. The debate on food addiction generally overlooks that the term addiction comes pre-loaded with unhelpful and emotive connotations that lead to misconceptions and confusion among scientists, clinicians and the public. Addiction was not originally a scientific or medical term, with a traditional meaning from the Latin verb *addicere* 'to

devote or attach (oneself) to an activity or cause; bound as, or as if, a servant'[31]. However, addiction is now in common use as a casual label for any excessive habitual behaviour[32]. The original definition of addiction is neutral in that the target behaviours do not necessarily harm the individual and some could even be beneficial (devotion to a project, a charity or ones family, for example). This definition is also therefore indiscriminately broad, as the list of objects to which the addiction label could be applied is unlimited (including, television, social media, shoes or dancing), and scientifically redundant. The use of addiction in its medical sense only emerged in the past century (the term was not formally included in the main text of the Oxford English Dictionary until 1989) and unlike the original definition, its meaning is unambiguously categorical (narrowing down the concept to designated classes of drugs) and explicitly negative (capable of causing significant harm or impairment to the individual or society). The rise of the medical concept of addiction has corresponded closely with public and political opinion on drug use in general[33].

In societies where drugs are deemed a social problem and are criminalised, addicts tend to be associated with criminality and addiction is often seen as social degeneracy. In this sense, addiction is a political as much as a medical concept, classed according to the prevailing moral judgement as well as clinical harm[10]. Therefore, when used in a medical sense, the term addiction implies illness, disease and the need for treatment; at the same time, the term is inextricably connected with moral disapproval towards socially undesirable drug-related behaviour. Importantly, the expert working group responsible for shaping the current clinical guidelines on the diagnosis of addiction refer directly to the difficulties arising from use of the term and justify its omission from diagnostic terminology due to "its uncertain definition and its potentially negative connotations"[34]. Therefore, it is unfortunate that the advocates of food addiction have appropriated the term in the first place and continue to endorse it despite its meaning being so frequently misunderstood. Whether intentional or not, the use of the term 'addiction' in the food addiction literature is emotive, simultaneously invoking its medical and moral connotations. The term is also ambiguous and potentially self-contradictory; enabling the broadest construal of the word (any unwanted excessive eating), while also implying a specific, narrow explanation (a distinct clinical entity). Hypotheses about 'food dependence' or 'food use disorder' that omit the term 'addiction' have been conspicuously absent from the literature[35] and in the media; however, the implementation of these alternative diagnostic terms could be similarly problematic (see subsequent sections). Indeed similar controversies over what to term dependency or addiction exist for recognised substance-use disorders in which behaviour has a prominent role[32,33]. Currently, the literature contains no clarity on the meaning of addiction when applied to food. Until a clear definition of food addiction is established, there can be no scientific basis for its validation.

[H1] Diagnostic approaches to food addiction

In the past 7 years, much of the scientific literature proclaiming support for food addiction has circumvented the issue of having no defined syndrome or symptoms by adopting a proxy definition that is derived from the generic behavioural criteria for substance-use disorders as specified in the DSM. This diagnostic approach has been largely directed by the development of a questionnaire-based self-report screening tool named the 'Yale Food Addiction Scale'[36,37]. The well-publicised name of the questionnaire is regrettably value-laden considering the emotive and hypothetical status of food addiction[28] and might bias the interpretation of patterns of otherwise normal eating behaviour in studies using the tool. In the latest revision of the scale, 35 items that fall under the 11 generic diagnostic criteria for substance-related disorders in the DSM-V have been adapted so that the class of substance relates broadly to "certain foods" with which people sometimes "have difficulty controlling how much they eat" or "any other foods you have had difficulty with in the past year"[37]. In addition to this conflation of certain or any foods under one substance category, the scale provides 23 examples of potential 'certain foods' listed under five categories (sweets, starches, salty foods, fatty foods and sugary drinks). Therefore, the identification of the specific foods, food categories or biochemical properties that are the reason for a given patient's responses is impossible with this scale. However, researchers can refer to the foods listed in the scale to speculate that any one food, food category or property might account for scores and diagnoses using the Yale Food Addiction Scale.

Each question on the Yale Food Addiction Scale represents one diagnostic symptom with an assigned threshold according to frequency of endorsement from "never" to "every day". Two additional questions are intended to represent 'significant clinical impairment or distress' resulting from food and eating (for example, question 16: "My eating behavior caused me a lot of distress"). A 'diagnosis' of food addiction is subsequently applied when any two of the 11 criteria are endorsed along with one question relating to impairment or distress. Severity of the diagnostic score is further specified as 'mild food addiction' (2-3 criteria plus clinical significance), 'moderate food addiction' (4-5 criteria plus clinical significance) or 'severe food addiction' (six or more criteria plus clinical significance). Whether or not the diagnostic approach is the most appropriate platform for demonstrating the existence of food addiction is not widely agreed upon; however, its apparent validity and intuitive appeal have made it popular among researchers. The importance of the scale for the food addiction hypothesis should not be understated because this scale underpins much of the human evidence used to assert the legitimacy of food addiction as a clinical entity.

[H1] Limitations of the diagnostic approach

One early problem that has arisen from the diagnostic approach is its inability to distinguish whether the diagnostic criteria relate to the addictive effects of certain foods or the act of eating itself[7,38]. This limitation calls into question the first key assumption of the food addiction hypothesis (that foods contain addictive substances), and has somewhat derailed the debate toward whether food addiction is a behavioural addiction[3,39] or simply a rebranding of existing clinically recognised eating disorders[8,9]. In this respect, controversy over using the diagnostic approach to food addiction follows a familiar trope that has been criticised in relation to an array of behavioural 'addictions' that have been prematurely introduced to the literature (such as addiction to mobile phone use, fortune-telling or romantic love)[40]. One group of researchers succinctly delineated the process into three steps: first, a new class of 'addiction' is hypothesised based on untested assumptions and strong beliefs; secondly, an ad hoc screening tool is developed based on the loose application of DSM diagnostic criteria for addiction; and thirdly, the tool is deployed to generate information on its psychometric reliability, 'prevalence' estimates in different populations and correlations with known risk factors for recognised substance-related disorders[41]. These findings are then miscommunicated as validation for the existence and importance of the hypothesised addiction, and given the appearance of widespread acceptance through repeated assertion and cross-citation among researchers[40].

The diagnostic approach to food addiction is therefore flawed because it provides no explicit (non-proxy) definition for the concept of food addiction. Moreover, a clear definition is unlikely to be achieved as a consensus is not forthcoming on the alleged addictive agent: be it all foods[21] or a specific biochemical attribute[42]. Furthermore, a consensus is lacking on whether any excessive eating[7] or a specific pattern of binge or binge–purge behaviour should define food addiction[43]. Using the diagnostic approach, respondents who experience distress from eating can be given the same diagnostic label and severity (mild food addiction) from endorsing any one of 55 possible combinations of the 11 criteria. What one individual who frequently eats more than planned and puts themselves in danger by eating and driving (criteria one and eight) would have in common with another individual who spends a lot of time eating and in whom their eating is causing problems in their social relationships (criteria three and six) is unclear. In addition, responses to the Yale Food Addiction Score cannot be used to determine if the problems referred to are even attributable to an addiction-like process rather than another feasible explanation (such as poor driving habits caused by time pressure from a long commute, or family health concerns due to weight gain). In addition, verifying the nature or clinical significance of their reported distress is impossible. Adopting the diagnostic approach gives a form of impartiality and administrative efficiency[33], but a clear sense of what food addiction really is cannot be extracted from using this approach. Therefore, the approach

bypasses a much-needed phenotypic characterisation of food addiction based on clinical observations that would enable specific diagnostic criteria to be defined in operational terms. Consequently, distinguishing between numerous different possible causes of obesity, or clarifying the psychological processes that might be sustaining different clusters of behavioural symptoms that might or might not be appropriately labelled as food addiction is not possible. By using the DSM criteria as its proxy definition and as the basis for its assessment tool without addressing the core clinical syndrome, the food addiction concept takes on its addiction-like attributes by mere association and its existence remains questionable.

[H1] A biopsychological approach

A major caution in adopting the concept of food addiction is that it fails to define a profile of consumption or behaviour that delineates addictive from non-addictive foods (**Box 2**), or normal from abnormal patterns of food intake. Used metaphorically, the concept captures common human experiences around food, including loss of control over eating, the pleasure-giving properties of food and ambivalence towards foods that are attractive but resisted[44]. Therefore, distinguishing behaviours that might qualify as symptoms of food addiction from those that are pursued because they are pleasurable, popular and bound by context or culture is difficult, and arguably unnecessary. Hedonic overeating (defined here as eating beyond metabolic requirements from the expectation and/or experience (that is, wanting and liking (see next section)) of pleasure from consuming specific foods) is a natural consequence of living in an environment that legitimises excessive and indulgent food habits and is unlikely to be any better understood by terms such as 'addictive agents in foods' or 'addictive-like overeating'. To do so would imply a total medicalisation of eating behaviour. Instead, a greater depth of understanding is made possible by adopting a broader biopsychological approach that encompasses the mechanisms that underpin the full spectrum of hedonic eating behaviour within a framework of 'regular' appetitive motivation[45]. This approach accounts for the natural appetite for pleasure (which is essential in a well-functioning homeostatic system for the supply of energy) as well as forms of aberrant eating (natural behaviour taken to excessive levels[46]), without recourse to a disease notion of food behaviour.

[H1] Hedonic overeating

Whereas the concept of food addiction is problematic when used to explain obesity or extreme patterns of eating behaviour, understanding of the processes involved in hedonic overeating has progressed immeasurably thanks to the concepts and methodologies derived from neurobiological theories of drug addiction[15,47]. Of particular relevance are the constructs of liking (an experience of pleasure) and wanting (anticipatory motivation), which are distinct hedonic processes with

dissociable neural pathways that are thought to serve as a basis for animals (including humans) to learn behaviours that lead to the acquisition of energy and essential nutrients[48,49]. The incentive sensitisation theory[50] describes how intense stimulation from drugs (to an intensity that far exceeds any food) can cause dysfunction of the natural reward system, including the sensitisation of mesolimbic dopamine neurons, which are involved in the wanting process.

In the study of human appetite and obesity, experimental methods have been developed to distinguish between the hedonic components of liking and wanting for food and to measure them separately using functional MRI[51,52] and behavioural laboratory studies[53-55]. A procedure has been developed and refined over a number of years to simultaneously measure liking and wanting components of reward using direct and indirect measures of behavioural responses to objective dimensions (sensory and nutrient components) of food[56]. Liking and wanting pathways interact with pathways for hunger[56], influence the strength of satiety[57] and can be used to predict the amount of food that will be consumed over the course of a day[58], which suggests that these processes have an important role in normal eating behaviour. However, liking and wanting are also features that can explain patterns of hedonic overeating in certain susceptible phenotypes. For example, high scores on the binge eating scale[59] are characterised by differences in liking and wanting (decreased liking but increased wanting for high fat and sweet tasting food when satiated) compared with those who have a low score. Liking and wanting can also be used to distinguish between participants with and without obesity — those who 'want' and go on to overconsume when high-fat and sweet foods are available[60]. Nevertheless, it is the sensitisation rather than mere activation of the wanting pathways that is thought to account for why drug addiction becomes so motivationally compulsive and persistent to a devastating degree[46]. Foods and drugs generate neural activity in common systems, which might help to explain excessive use of either commodity, but no evidence currently demonstrates neural sensitisation to food[6]. The clinical concept of addiction is not reducible to neurochemical events in the brain, and the normal operation of the hedonic, or reward, system does not denote pathology. Neither does the capacity of a food to merely activate the reward system provide a basis for its classification as an addictive substance. Therefore, many of the claims regarded as the property of a medicalised concept of food addiction can be explained through the operation of normal (not pathological) hedonic processes operating in an energy dense, culturally permissive food environment.

[H1] Conclusion

Obesity — the accumulation of excess levels of body fat — depends on an imbalance between energy intake and energy expenditure over time. Food preference and physical activity habits

(including sedentary activities) are important contributors to daily variation in energy intake and expenditure, respectively. In humans, biological predispositions have evolved that favour a strong attraction to energy-dense foods and a tendency to minimise intense physical activity[60], mediated by mechanisms (liking and wanting) related to the hedonic system. These tendencies only become relevant for obesity in the appropriate environmental conditions. The optimal conditions for obesity to develop include an abundant, energy-dense food supply, limited need or opportunity for physical activity, and socio-cultural values that encourage mass (over)consumption of commodities[13]. Therefore, from a biopsychological perspective, the complex processes underlying overconsumption and obesity can be understood as normal biological adaptations to lifestyles shaped by powerful pressures from the modern obesogenic environment[61]. These biopsychological explanations for overeating and weight gain do not draw the sensational headlines of food addiction and might be less appealing in the eyes of the public and those looking for clear-cut implementable solutions to the obesity crisis (that is, changes to law and regulation and/or diagnosis and treatment). However, they avoid the dilemma posed by food addiction in which its promoters are caught between an over-pathologisation of common processes involved in hedonic eating behaviour or a broadening of the medical concept of addiction to cover the entire spectrum of appetitive human motivation, rendering it meaningless.

G. Finlayson is at School of Psychology, Faculty of Medicine & Health, Lifton Place, University of Leeds, LS2 9JT, West Yorkshire, UK.

g.s.finlayson@leeds.ac.uk

1. Schulte, E.M., Joyner, M.A., Potenza, M.N., Grilo, C.M. & Gearhardt, A.N. Current considerations regarding food addiction. *Current psychiatry reports* **17**, 1-8 (2015).
2. Carter, A. et al. The Neurobiology of" Food Addiction" and Its Implications for Obesity Treatment and Policy. *Annual Review of Nutrition* **36**, 105-128 (2016).
3. Davis, C. A commentary on the associations among 'food addiction', binge eating disorder, and obesity: Overlapping conditions with idiosyncratic clinical features. *Appetite* http://dx.doi.org/10.1016/j.appet.2016.11.001 (2016).
4. Ziauddeen, H., Farooqi, I.S. & Fletcher, P.C. Obesity and the brain: how convincing is the addiction model? *Nature Reviews Neuroscience* **13**, 279-286 (2012).
5. Avena, N.M., Gearhardt, A.N., Gold, M.S., Wang, G.-J. & Potenza, M.N. Tossing the baby out with the bathwater after a brief rinse? The potential downside of dismissing food addiction based on limited data. *Nature Reviews Neuroscience* **13**, 514-514 (2012).

6. Westwater, M.L., Fletcher, P.C. & Ziauddeen, H. Sugar addiction: the state of the science. *European Journal of Nutrition* **55**, 55-69 (2016).
7. Hebebrand, J. et al. "Eating addiction", rather than "food addiction", better captures addictive-like eating behavior. *Neuroscience & Biobehavioral Reviews* **47**, 295-306 (2014).
8. Albayrak, Ö., Wölfle, S.M. & Hebebrand, J. Does food addiction exist? A phenomenological discussion based on the psychiatric classification of substance-related disorders and addiction. *Obesity facts* **5**, 165-179 (2012).
9. Long, C.G., Blundell, J.E. & Finlayson, G. A Systematic Review of the Application And Correlates of YFAS-Diagnosed 'Food Addiction'in Humans: Are Eating-Related 'Addictions'a Cause for Concern or Empty Concepts? *Obesity facts* **8**, 386-401 (2015).
10. Fraser, S. Junk: Overeating and obesity and the neuroscience of addiction. *Addiction Research & Theory* **21**, 496-506 (2013).
11. Corwin, R.L. & Hayes, J.E. in Fructose, High Fructose Corn Syrup, Sucrose and Health 199-215 (Springer, 2014).
12. WHO. Obesity and Overweight. Fact Sheet No 311. http://www.who.int/mediacentre/factsheets/fs311/en/ (2016).
13. Butland, B. et al. Foresight. Tackling obesities: future choices. Project report. *Gov.uk* https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/287937/07-1184x-tackling-obesities-future-choices-report.pdf (2007). **[AuOK?]**
14. Levi, J., Segal, L.M., Laurent, R.S., Lang, A. & Rayburn, J. F as in fat: how obesity threatens America's future 2012. *Trust for America's Health* http://healthyamericans.org/assets/files/TFAH2012FasInFat18.pdf (2012).
15. Stice, E., Figlewicz, D.P., Gosnell, B.A., Levine, A.S. & Pratt, W.E. The contribution of brain reward circuits to the obesity epidemic. *Neuroscience & Biobehavioral Reviews* **37**, 2047-2058 (2013).
16. Ziauddeen, H., Alonso-Alonso, M., Hill, J.O., Kelley, M. & Khan, N.A. Obesity and the neurocognitive basis of food reward and the control of intake. *Advances in Nutrition: An International Review Journal* **6**, 474-486 (2015).
17. AMA. Recognition of Obesity as a Disease. Resolution 420 (A-13). http://www.npr.org/documents/2013/jun/ama-resolution-obesity.pdf (2013).
18. Allison, D.B. et al. Obesity as a disease: a white paper on evidence and arguments commissioned by the Council of the Obesity Society. *Obesity* **16**, 1161-1177 (2008).
19. Bray, G.A. Medical consequences of obesity. *The Journal of Clinical Endocrinology & Metabolism* **89**, 2583-2589 (2004).

20. Attia, E. et al. Feeding and eating disorders in DSM-5. *American Journal of Psychiatry* **170**, 1237-1239 (2013).
21. Gearhardt, A.N., Grilo, C.M., DiLeone, R.J., Brownell, K.D. & Potenza, M.N. Can food be addictive? Public health and policy implications. *Addiction* **106**, 1208-1212 (2011).
22. Meule, A. How prevalent is "food addiction"? *Frontiers in psychiatry* **2**, 61 (2011).
23. DePierre, J.A., Puhl, R.M. & Luedicke, J. A new stigmatized identity? Comparisons of a "food addict" label with other stigmatized health conditions. *Basic and Applied Social Psychology* **35**, 10-21 (2013).
24. Lee, N.M. et al. Public views on food addiction and obesity: implications for policy and treatment. *PloS one* **8**, e74836 (2013).
25. Smith, D.G. & Robbins, T.W. The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. *Biological psychiatry* **73**, 804-810 (2013).
26. Ziauddeen, H. & Fletcher, P.C. Is food addiction a valid and useful concept? *obesity reviews* **14**, 19-28 (2013).
27. Hone-Blanchet, A. & Fecteau, S. Overlap of food addiction and substance use disorders definitions: analysis of animal and human studies. *Neuropharmacology* **85**, 81-90 (2014).
28. Basurte, I. & Szerman, N. Food addiction: a critical reflexion. *Salud Mental* **39**, 107-108 (2016).
29. Benton, D. & Young, H. A meta-analysis of the relationship between brain dopamine receptors and obesity: a matter of changes in behavior rather than food addiction&quest. *International journal of obesity* **40**, S12-S21 (2016).
30. Blundell, J.E. & Finlayson, G. Food addiction not helpful: the hedonic component–implicit wanting–is important. *Addiction* **106**, 1216-1218 (2011).
31. Dictionary, O.E. "addicted, adj. and n." (Oxford University Press).
32. Stepney, R. The concept of addiction: its use and abuse in the media and science. *Human Psychopharmacology: Clinical and Experimental* **11**, S15-S20 (1996).
33. Alexander, B. The globalization of addiction: A study in poverty of the spirit (Oxford University Press, 2010).
34. Association, A.P. Diagnostic and statistical manual of mental disorders (DSM-5®) (American Psychiatric Pub, 2013).
35. Raymond, K.-L., Kannis-Dymand, L. & Lovell, G.P. A graduated food addiction classification approach significantly differentiates obesity among people with type 2 diabetes. *Journal of Health Psychology* **1**, 9 (2016).

36. Gearhardt, A.N., Corbin, W.R. & Brownell, K.D. Preliminary validation of the Yale food addiction scale. *Appetite* **52**, 430-436 (2009).
37. Gearhardt, A.N., Corbin, W.R. & Brownell, K.D. Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors* **30**, 113 (2016).
38. Schulte, E.M., Grilo, C.M. & Gearhardt, A.N. Shared and unique mechanisms underlying binge eating disorder and addictive disorders. *Clinical psychology review* **44**, 125-139 (2016).
39. Marks, I. Behavioural (non-chemical) addictions. *British Journal of Addiction* **85**, 1389-1394 (1990).
40. Blaszczynski, A. Commentary on: Are we overpathologizing everyday life? A tenable blueprint for behavioral addiction research. *Journal of behavioral addictions* **4**, 142-144 (2015).
41. Billieux, J., Schimmenti, A., Khazaal, Y., Maurage, P. & Heeren, A. Are we overpathologizing everyday life? A tenable blueprint for behavioral addiction research. *Journal of behavioral addictions* **4**, 119-123 (2015).
42. Avena, N.M., Rada, P. & Hoebel, B.G. Evidence for sugar addiction: Behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neuroscience and Biobehavioral Reviews* **32**, 20-39 (2008).
43. Davis, C. Compulsive overeating as an addictive behavior: overlap between food addiction and binge eating disorder. *Current Obesity Reports* **2**, 171-178 (2013).
44. Rogers, P.J. & Smit, H.J. Food craving and food “addiction”: a critical review of the evidence from a biopsychosocial perspective. *Pharmacology Biochemistry and Behavior* **66**, 3-14 (2000).
45. Dalton, M. & Finlayson, G. in *Satiation, satiety and the control of food intake: theory and practice* (ed. Blundell, J. E. & Bellisle, F.) 221-237 (Woodhead Publishing Ltd, Cambridge, 2013).
46. Berridge, K.C. 'Liking' and 'wanting' food rewards: brain substrates and roles in eating disorders. *Physiology & behavior* **97**, 537-50 (2009).
47. Finlayson, G., King, N. & Blundell, J.E. Liking vs. wanting food: Importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews* **31**, 987-1002 (2007).
48. Berridge, K.C. & Kringelbach, M.L. Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology* **199**, 457-480 (2008).
49. Berridge, K.C. & Robinson, T.E. Parsing reward. *Trends Neurosci* **26**, 507-513 (2003).

50. Robinson, T.E. & Berridge, K.C. The neural basis of drug craving: An incentive-sensitization theory of addiction. *Brain Research Reviews* **18**, 247-291 (1993).
51. Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. & Small, D. Relation of Reward from Food Intake and Anticipated Food Intake to Obesity: A Functional Magnetic Resonance Imaging Study. *Journal of Abnormal Psychology* **117**, 924-935 (2008).
52. Cambridge, V.C. et al. Neural and behavioral effects of a novel mu opioid receptor antagonist in binge-eating obese people. *Biological psychiatry* **73**, 887-894 (2013).
53. Finlayson, G., King, N. & Blundell, J.E. Is it possible to dissociate 'liking'and 'wanting'for foods in humans? A novel experimental procedure. *Physiology & Behavior* **90**, 36-42 (2007).
54. Rogers, P.J. & Hardman, C.A. Food reward. What it is and how to measure it. *Appetite* **90**, 1-15 (2015).
55. Epstein, L.H., Leddy, J.J., Temple, J.L. & Faith, M.S. Food reinforcement and eating: a multilevel analysis. *Psychol Bull* **133**, 884-906 (2007).
56. Finlayson, G., King, N. & Blundell, J. The role of implicit wanting in relation to explicit liking and wanting for food: implications for appetite control. *Appetite* **50**, 120-127 (2008).
57. Griffioen-Roose, S., Finlayson, G., Mars, M., Blundell, J.E. & de Graaf, C. Measuring food reward and the transfer effect of sensory specific satiety. *Appetite* **55**, 648-655 (2010).
58. French, S.A., Mitchell, N.R., Finlayson, G., Blundell, J.E. & Jeffery, R.W. Questionnaire and laboratory measures of eating behavior. Associations with energy intake and BMI in a community sample of working adults. *Appetite* **72**, 50-58 (2014).
59. Gormally, J., Black, S., Daston, S. & Rardin, D. The assessment of binge eating severity among obese persons. *Addict Behav* **7**, 47-55 (1982).
60. Dalton, M. & Finlayson, G. Psychobiological examination of liking and wanting for fat and sweet taste in trait binge eating females. *Physiology & Behavior* **136**, 128-134 (2014).
61. Eaton, S.B. & Eaton, S.B. An evolutionary perspective on human physical activity: implications for health. *Comparative Biochemistry and Physiology Part A: Molecular & Integrative Physiology* **136**, 153-159 (2003).
62. Tremblay, A. & Doucet, E. Obesity: a disease or a biological adaptation? *Obesity Reviews* **1**, 27-35 (2000).
63. Rada, P., Avena, N.M. & Hoebel, B.G. Daily bingeing on sugar repeatedly releases dopamine in the accumbens shell. *Neuroscience* **134**, 737-744 (2005).
64. Rada, P., Bocarsly, M.E., Barson, J.R., Hoebel, B.G. & Leibowitz, S.F. Reduced accumbens dopamine in Sprague–Dawley rats prone to overeating a fat-rich diet. *Physiology & behavior* **101**, 394-400 (2010).

65. Johnson, P.M. & Kenny, P.J. Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. *Nature neuroscience* **13**, 635-641 (2010).

66. Thornley, S., McRobbie, H., Eyles, H., Walker, N. & Simmons, G. The obesity epidemic: Is glycemic index the key to unlocking a hidden addiction? *Medical Hypotheses* **71**, 709-714 (2008).

67. Schulte, E.M., Avena, N.M. & Gearhardt, A.N. Which foods may be addictive? The roles of processing, fat content, and glycemic load. *PLoS One* **10**, e0117959 (2015).

68. Goldstone, A.P. et al. Fasting biases brain reward systems towards high-calorie foods. *European Journal of Neuroscience* **30**, 1625-1635 (2009).

69. Rosenbaum, M., Sy, M., Pavlovich, K., Leibel, R.L. & Hirsch, J. Leptin reverses weight loss–induced changes in regional neural activity responses to visual food stimuli. *The Journal of clinical investigation* **118**, 2583-2591 (2008).

70. Blundell, J. et al. The fat paradox: fat-induced satiety signals versus high fat overconsumption. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity* **19**, 832 (1995).

71. Swinburn, B.A. et al. The global obesity pandemic: shaped by global drivers and local environments. *The Lancet* **378**, 804-814 (2011).

72. Swinburn, B., Sacks, G. & Ravussin, E. Increased food energy supply is more than sufficient to explain the US epidemic of obesity. *The American journal of clinical nutrition* **90**, 1453-1456 (2009).

73. Barrett, D. Supernormal stimuli: How primal urges overran their evolutionary purpose (WW Norton & Company, London, 2010).

74. Goodwin, B., Browne, M. & Rockloff, M. Measuring Preference for Supernormal Over Natural Rewards A Two-Dimensional Anticipatory Pleasure Scale. *Evolutionary Psychology* https://doi.org/10.1177/1474704915613914 (2015).

75. Rogers, P.J. Food and drug addictions: Similarities and differences. *Pharmacology, Biochemistry and Behavior* **153**, 182-190 (2017).

76. McHugh, P.R. & Slavney, P.R. Mental Illness — Comprehensive Evaluation or Checklist? *New England Journal of Medicine* **366**, 1853-1855 (2012).

77. Cuthbert, B.N. & Kozak, M.J. Constructing constructs for psychopathology: The NIMH research domain criteria. *Journal of Abnormal Psychology* **122**, 928-937 (2013).

Acknowledgements

The author wishes to thank Prof John Blundell (University of Leeds) for his comments and discussion about this article and acknowledges the contribution of Cecilia Long and Dr Michelle Dalton (University of Leeds) in its preparation.

Competing interests statement

The authors declare no competing interests.

Publisher's note

Box 1 | Can the DSM be used to understand food addiction?

The two leading classification systems for determining medically accepted forms of addiction are the Diagnostic and Statistical Manual of Mental Disorders, fifth edition (DSM-V) published by the American Psychiatric Association[34] and the International Statistical Classification of Diseases and Related Health Problems, tenth revision (ICD-10) from the WHO[76]. Both systems recognise ten separate substance-use disorders, and the DSM-V also includes 'gambling disorder' as the only non-substance use addictive disorder. These conditions are generally conferred the status of discrete disease entities and are intended for widespread clinical use as a result of their convincing empirical evidence base or clinical utility. The DSM-V further proposes caffeine use and internet gaming as 'conditions for further study'. These potential conditions require more research before their inclusion or exclusion as additional distinct disorders can be judged. In the DSM, diagnostic criteria are provided for pica (eating items with no nutritional value), rumination disorder, avoidant or restrictive food intake disorder, anorexia nervosa, bulimia nervosa, and binge-eating disorder. The term addiction is omitted from the DSM-V diagnostic terminology due to its ambiguous definition and potential to stigmatise those diagnosed with the condition. 'Food addiction', whether framed as a substance-related or non-substance addictive disorder has not been approved as a diagnosable entity in the DSM or ICD. The classification of disorders by their common symptoms does not contribute to understanding of their underlying aetiology or mechanism and the DSM has been criticised for its lack of validity and promoting a short-hand approach to diagnosis, bypassing the comprehensive clinical assessment necessary to know more about the course and stability of illness, familial predisposition, biomarkers and response to treatment[77,78]. This criticism should serve as a caution that considerable doubt currently exists about the authenticity of food addiction as a clinical condition.

Box 2 | Is there an addictive substance in food?

For the food addiction hypothesis to be tested, it is necessary to identify the specific biochemical properties that might be capable of producing an addictive process in the brain. Frequently, the capacity of a food to release dopamine or produce activation in the nucleus accumbens is surmised as justification for its addictive potential, which is clearly inadequate. In animal studies, three separate models of food addiction have been examined (sugar-bingeing[63], fat-bingeing[64] and sweet–high-fat diets[65]) and suggest that under certain circumstances, and with certain feeding regimens, eating behaviours can take on a pattern that neurobiologically resembles addiction. However, these studies have weak relevance to human eating patterns[27]. In humans, the glycaemic load or glycaemic index of foods have been proposed as the addictive element[66,67], but the pharmacodynamic mechanism that explains the link between blood levels of glucose and addiction is missing[6]. These ambiguous and inconsistent findings are increasingly being extrapolated to create unscientific classes of food such as 'hyper-palatable' or 'ultra-processed'.

What all candidate addictive agents proposed so far have in common are that they are substances that make foods more appealing because they typically predict available energy. Energy density is a naturally preferred feature in foods and it is highly adaptive to be attracted to such foods, particularly when hungry[68] or in energy deficit[69]. 'Passive-overconsumption'[70] that arises from exposure to a high-energy food supply is more than sufficient to account for the prevalence of overweight and obesity in modern society[71,72]. The essence of the issue is that making a natural reward, like food, more appealing through any degree of processing is not the same as making it addictive. The preference for exaggerated versions of natural rewarding stimuli over less intense variants is an adaptive strategy observed throughout the animal kingdom to maximise survival and reproductive success[73]. In the modern environment, the phenomenon extends to all commodities, not only ('hyper') palatable or ('ultra') processed foods[74].

Author biography

Graham Finlayson is Associate Professor in the School of Psychology, Faculty of Medicine & Health, University of Leeds, UK. His research interests focus on the psychobiology of food choice and hedonics in human appetite. He also investigates the role of exercise on eating behaviour. Dr Finlayson received his PhD from the University of Leeds and post-doctoral training at Glasgow Caledonian University, UK. He currently co-directs the Appetite Control & Energy Balance laboratories at University of Leeds (http://aceb-research.leeds.ac.uk).

Competing interests statement

The author declares no competing interests.

Subject ontology terms

Health sciences / Endocrinology / Endocrine system and metabolic diseases / Obesity
[URI /692/163/2743/393]

Health sciences / Diseases / Psychiatric disorders / Addiction

[URI /692/699/476/5]

Biological sciences / Neuroscience / Reward

[URI /631/378/1788]

Table of Contents