OPINION

Food addiction and obesity: unnecessary medicalization of hedonic overeating

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Abstract | The concept of addiction is loaded with connotations and is 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 being 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, or reward, system can account for the ingestion of foods and drugs, confirming that they share neural substrates that differentiate liking and wanting. These are normal processes that are 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 behaviours, taking on the properties of a disease. The 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 currently a validated concept; it has not been approved by either of the two leading classification systems for diagnosing mental diseases, which include all medically recognized subtypes of substance-use disorders and eating disorders. No clear scientific proof exists in humans that certain biochemical properties in foods are addictive. Agreement is also lacking on the symptoms of food addiction that a patient might present with in the clinic that would distinguish these symptoms from the defined clinical symptoms of recognized 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[7–9], and psychometric or diagnostic[10], deficiencies that are damaging to the concept of food addiction. Some researchers have expressed concern that the concept invites the medicalization of natural motivational needs[11,12]. Indeed, no strong evidence exists to substantiate the existence of food addiction that cannot be adequately explained through normal (biopsychological) adaptations to unhealthy lifestyles that are shaped by powerful socio-cultural pressures from the modern (obesogenic) environment. However, the food addiction concept persists. If one conducts an Internet search of the term 'food addiction', more than 12 million results will be found 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 clinical 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 when pushed to extremes.

The purpose of this Opinion article is to critically evaluate the current status of the food addiction hypothesis and to scrutinize its relevance for understanding patterns of eating behaviour that are 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 between 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 is 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, making them analogous to patients with a substance-use disorder) are examined in relation to explanations drawing on existing knowledge of the biopsychology of hedonic eating and its functioning in an obesogenic food environment.

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[21] and the International Statistical Classification of Diseases and Related Health Problems, tenth revision (ICD-10) from the WHO[64]. Both systems recognize 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 owing to its ambiguous definition and potential to stigmatize those diagnosed with the condition. Food addiction, whether framed as a substance-related or as a non-substance addictive disorder, has not been approved as a diagnosable entity in the DSM or the 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 criticized for its lack of validity and for promoting a short-hand approach to diagnosis, bypassing the comprehensive clinical assessment that is necessary to find out more about the course and stability of illness, familial predisposition, biomarkers and response to treatment[65,66]. This criticism should serve as a caution that considerable doubt currently exists about the authenticity of food addiction as a clinical condition.

The biomedical approach

Contemporary changes in food, physical activity and socio-economic environments have resulted in a doubling in the worldwide incidence of obesity since 1980 (REF. 13) and have led to predicted rates of >50% adult obesity in the UK and the USA within the next 2–3 decades[14,15]. 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 that are associated with the obese state[16,17].

A further element inherent to the biomedical approach is the classification and diagnosis of disease to identify aetiologies and to 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 organizations support its recognition as a disease (including the American Medical Association, the WHO and The Obesity Society)[18–20]. However, it is acknowledged that this recommendation is primarily based on its utility for focusing resources into obesity prevention, treatment and research[18,19]. Binge eating disorder is now well established as a clinical entity that is distinct from obesity, and its inclusion in the DSM-V[21] 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 neurobehavioural profile, which therefore has specific treatments[22].

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 of 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: first, that some foods warrant classification as addictive substances; and second, that some individuals are prone to becoming 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[23], and that people who might have food addiction should be diagnosed, investigated and treated[24]. However, two crucial pieces of evidence for the substantiation of the food addiction hypothesis are missing: no addictive biochemical property of 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[25] and endorses the unscientific belief in food addiction that is held by the majority of the public[26]. 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[27]. 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[10]. 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[11].

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[2,6–8,10,12,17,28–31]. 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 to 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[32]. Moreover, by over-pathologizing 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.

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 is 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 a medical term, with a traditional meaning, derived from the Latin *addicere*, "the state or condition of being dedicated or devoted to a thing, [especially] an activity or occupation" (REF. 33). However, the term addiction is now in common use as a casual label for any excessive habitual behaviour[34]. The original definition of addiction is neutral in that the target behaviours do not necessarily harm the individual and some of these behaviours could even be beneficial (for example, devotion to a project, to a charity or to one's family). Therefore, this definition is also indiscriminately broad, as the list of objects to which the addiction label could be applied is unlimited (including, television, social media, shoes and dancing), and scientifically redundant. The use of addiction in its medical sense has only emerged in the past century (the term was not formally included in the main 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 closely corresponded to public and political opinion of drug use in general[35].

In societies in which drugs are deemed a social problem and are criminalized, drug 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[11]. Therefore, when used in a medical sense, the term addiction currently implies illness, disease and the need for treatment; at the same time, the term is inextricably connected to moral disapproval towards socially undesirable drug-related behaviour. Importantly, the expert working group responsible for shaping the current clinical guidelines in the USA on the diagnosis of addiction refer directly to the difficulties arising from the use of the term and justify its omission from diagnostic terminology owing to "its uncertain definition and its potentially negative connotations" (REF. 21). Therefore, it is unfortunate that the advocates of food addiction have firstly appropriated the term and also 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 on food dependence or food use disorder that omit the term 'addiction' have been conspicuously absent from the literature[36] 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 recognized substance-use disorders in which behaviour has a prominent role[34,35]. Currently, the scientific 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.

Diagnostic approaches to food addiction

Over 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 mostly been directed by the development of a questionnaire-based self-report screening tool named the Yale Food Addiction Scale (REFS 37,38). The well-publicized name of the questionnaire is regrettably value-laden considering the emotive and hypothetical status of food addiction[30] 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" (REF. 38). 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 using 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 the frequency of endorsement from "never" to "every day" (REF. 37,38). Two additional questions are intended to represent significant clinical impairment or distress resulting from food and eating (for example, question 16: "My eating behaviour caused me a lot of distress" (REF. 37,38)). 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 (two or three criteria plus clinical significance), moderate food addiction (four or five criteria plus clinical significance) or severe food addiction (six or more criteria plus clinical significance). Whether the diagnostic approach is the most appropriate platform for demonstrating the existence of food addiction is not widely agreed on; however, its intuitive appeal has 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.

Limitations of the diagnostic approach

One early problem that has arisen from the diagnostic approach is its inability to distinguish whether the diagnostic criteria

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 to 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[67], fat-bingeing[68] and sweet–high-fat diets[69]) 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[29]. In humans, the glycaemic load or the glycaemic index of foods have been proposed as the addictive element[70,71], 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 new and unscientific classes of food such as 'hyper-palatable' and 'ultra-processed'.

What all candidate addictive agents proposed so far have in common is 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[72] or in energy deficit[73]. Passive overconsumption[74] that arises from exposure to a high-energy food supply is more than sufficient to account for the prevalence of individuals with overweight or obesity in modern society[75,76]. The essence of the issue is that making a natural reward, such as 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 that is observed throughout the animal kingdom to maximize survival and reproductive success[77]. In the modern environment, the phenomenon extends to all commodities, not only so-called hyper-palatable or ultra-processed foods[78].

relate to the addictive effects of certain foods or to the act of eating itself[7,39]. 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 towards whether food addiction is a behavioural addiction[3,40] or is simply a rebranding of existing clinically recognized eating disorders[8,10]. In this respect, controversy over using the diagnostic approach to food addiction follows a familiar trope that has been criticized 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 and romantic love)[41]. One group of researchers succinctly delineated the process into three steps: first, a new class of addiction is hypothesized based on untested assumptions and strong beliefs; second, an ad hoc screening tool is developed based on the loose application of DSM diagnostic criteria for addiction; and third, the tool is deployed to generate information on its psychometric reliability, prevalence estimates in different populations and correlations with known risk factors for recognized substance-related disorders[42]. These findings are then miscommunicated as validation for the existence and importance of the hypothesized addiction, and the hypothesized addiction is given the appearance of widespread acceptance through repeated assertion and cross-citation among researchers[41].

Therefore, the diagnostic approach to food addiction is 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[23] or a specific biochemical attribute[43]. 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[44]. 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. It is unclear 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). In addition, responses to the Yale Food Addiction Scale cannot be used to determine whether the problems referred to are even attributable to an addiction-like process rather than to 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 and clinical significance of their reported distress is impossible. Adopting the diagnostic approach gives a form of impartiality and administrative efficiency[35], 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 characterization of food addiction based on clinical observations that would enable specific diagnostic criteria to be defined in operational terms. Consequently, it is not possible to distinguish between numerous different possible causes of obesity or to clarify the psychological processes that might be sustaining different clusters of behavioural symptoms that might or might not be appropriately labelled as food addiction. 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.

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 foods 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[45]. 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 the pleasure obtained from consuming specific foods) is a natural consequence of living in an environment that legitimizes 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 medicalization 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 range of hedonic eating behaviour within a framework of regular appetitive motivation[46].

This approach accounts for 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[47]), without recourse to a disease notion of food behaviour.

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 that are involved in hedonic overeating has progressed immeasurably thanks to the concepts and methodologies derived from neurobiological theories of drug addiction[16,48]. 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[49,50]. The incentive sensitization theory[51] describes how intense stimulation from drugs (to an intensity that far exceeds that from any food) can cause dysfunction of the natural reward system, including the sensitization 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 food and to measure them separately using functional MRI[52,53] and behavioural laboratory studies[54–56]. A procedure has been developed and refined over a number of years to simultaneously measure the liking and the wanting components of reward using direct and indirect measures of behavioural responses to objective dimensions (sensory and nutrient components) of food[57]. Liking and wanting pathways interact with pathways for hunger[57], influence the strength of satiety[58] and can be used to predict the amount of food that will be consumed over the course of a day[59], 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, people with high scores on the binge eating scale[60] are characterized 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[61]. Nevertheless, it is the sensitization rather than the 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[47]. Foods and drugs generate neural activity in common systems, which might help to explain the excessive use of either commodity, but no evidence currently demonstrates neural sensitization 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 medicalized concept of food addiction can be explained through the operation of normal (not pathological) hedonic processes in an energy-dense, culturally permissive food environment.

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 energy expenditure, respectively. In humans, biological predispositions have evolved that favour a strong attraction to energy-dense foods and a tendency to minimize intense physical activity[62], mediated by mechanisms (liking and wanting) that are related to the hedonic system. These tendencies only become relevant for obesity under 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 consumption of commodities[14]. Therefore, from a biopsychological perspective, the complex processes underlying overconsumption and obesity can be understood as normal biological adaptations to lifestyles that are shaped by powerful pressures from the modern obesogenic environment[63]. 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 laws and regulations and/or diagnosis and treatment). However, they avoid the dilemma posed by food addiction in which its promoters are caught between an over-pathologization of common processes that are involved in hedonic eating behaviour and a broadening of the medical concept of addiction to cover the entire spectrum of appetitive human motivation, rendering it meaningless.

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