Food addiction – What is the evidence?

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On 7 October 2013, the British Nutrition Foundation held a half-day symposium in London, ‘Food addiction – what is the evidence?’, to consider the topic of food addiction in detail and examine the evidence behind claims that have been made and reported in the popular press. The event was chaired and introduced by Professor John Blundell (University of Leeds).

What is food addiction?

The term food addiction has been in use for a number of decades. Following publication of a number of papers that focused on food addiction with particular regard to sugar, fat and processed foods, there has been extensive publicity in the press on the supposition that certain foods are ‘addictive’ and that food ‘addiction’ is contributing to the current obesity epidemic. Despite this media exposure, there is a certain degree of confusion around the term ‘food addiction’, its diagnosis, definition and whether it even exists.

Associate Professor Graham Finlayson (University of Leeds) explored the scientific validity and measurement of food addiction in humans and discussed its contribution to overeating or as a subtype of obesity. More recently, the term addiction is being used colloquially to describe anything done to excess (e.g. addictions to gambling, Internet, sex and food). A key task for researchers in this area is to discern which addictions are ‘real’. Classifications of clinically defined psychiatric conditions for which there are agreed diagnostic procedures are incorporated into the Diagnostic and Statistical Manual (DSM) of the American Psychiatric Association (APA). In previous versions, there were no

mentions of ‘addiction’; instead the term ‘Substance-Related Disorders’ was used. In the latest version, published in 2013, this definition has been extended to cover ‘Substance-Related and Addictive Disorders’. In early 2013, prior to publication, the APA’s Classification Committee rejected ‘food addiction’ as a diagnosable entity in the fifth edition of DSM (DSM-V), published in late 2013. This decision should signal caution and add to the considerable doubt about the authenticity of food addiction as a recognisable condition.

The question of whether food addiction is a valid scientific concept is not new but its reexamination has been stimulated by recent methodological and scientific developments. It has been claimed that findings from rodent studies show similarities between the behavioural patterns elicited by foods and drugs, as well as correspondence between the effects of food and (hard) drugs on brain neurotransmitters. But the concept of food as a ‘drug’ is controversial, as will be discussed later in this report.

Another reason for the returning interest in food addiction is the development of ‘The Yale Food Addiction Scale’ (YFAS), described as ‘a measure that has been developed to identify those who are most likely to be exhibiting markers of substance dependence with the consumption of high fat/high sugar foods’ (Gearhardt et al. 2009). Psychometric scales, such as the YFAS, can capture individual differences in susceptibility to overeating. Although this approach holds promise for the investigation of hedonic responses (i.e. pleasure/reward systems) to the sensory features of food and the specific eating behaviours that characterise susceptible individuals at risk of weight gain, these characteristics should not be regarded as being markers of addiction. Moreover, there are problems when using this type of assessment in research. It assumes that addiction is stable, quantifiable and measurable; provides no clear threshold to differentiate between addictive and normative eating behaviours; and overlaps with other scales (e.g. the scale used to assess the severity of emotional eating).

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Food as an addictive substance

In his introduction to the topic, Professor Blundell highlighted that in the past, food addiction has been used to indicate strong preferences and food habits. However, in recent years it has been given a new interpretation, namely, that food addiction has the same properties as addiction to hard drugs. This claim has been characterised by strong language, emotive claims and the use of extreme slogans, such as ‘fat may rewire the brain like hard drugs’, ‘sweetness surpasses cocaine reward’ and ‘sugar is the new tobacco’.

Blundell advised that there is a growing understanding of the chemical pathways in the brain that encode reward and pleasure, as well as those that can differenti ate between liking and wanting. These systems are thought to have developed during evolution from the requirement to maintain blood levels of essential commodities necessary for survival and to have served as a basis for animals (including humans) to learn behaviours that would lead to food acquisition. It is, therefore, unsurprising that food consumption generates activity in these systems. It has been said that drug substances ‘hijack’ the food reward systems and in this way bring about their potent effects. Foods and drugs generate neural activity in common systems but this does not imply that the effects of foods are identical to the effects of drugs. The two materials share a common substrate but it should be remembered that it is the drugs that exploit food-based reward pathways and not the converse.

The fact that most foods share features with addictive drugs, in the way in which they affect the brain, is a key concept in the food addiction debate. As described by Professor Peter Rogers (University of Bristol) in his talk, Epstein & Shaham (2010) conducted an animal study in which the reward areas in the brains of obese rats were monitored during exposure to and withdrawal from energy-dense foods. With intakes of both the drugs and foods, the reward value of electrical brain stimulation decreased, and this was interpreted by the authors as showing that energy-dense foods can, like addictive drugs, cause reward ‘dysfunction’. However, when the drugs were withdrawn, the reward value of electrical brain stimulation returned to normal, whereas when the food was removed the values remained high, in parallel with those animals’ increased weight. This suggests that reduced reward is a consequence of obesity rather than a cause of overeating and that rather than being dysfunctional it is adaptive in helping to curb overeating.

Professor Ian Macdonald (University of Nottingham) discussed sucrose and fructose and the evidence behind the strong opinions that have been expressed regarding the toxic effects of these sugars. The majority debating this topic claim that blaming these individual sugars as the root of the problem is premature, given that evidence for this is lacking and research in this area is ongoing. However, in theory, sucrose and fructose could be contributing to obesity and type 2 diabetes due to their different effects on metabolism compared to glucose. Although it is suggested that these sugars lead to increased fat deposition in the liver and muscle tissue and increased insulin resistance, this is not supported by empirical evidence under controlled isocaloric conditions, as discussed below (Johnston et al. 2013). In addition, it has been speculated that the sweetness of sucrose and fructose affects the nervous system and causes behavioural changes that could lead to cravings and even ‘addiction’. But many studies in this area lack the appropriate control conditions to differentiate between sweetness per se (without energy) and intake of sugars such as glucose, sucrose and fructose (sweetness with energy).

Cravings and hyperpalatability

Professor Macdonald also covered the topic of food cravings, which are likely to involve the activation of dopaminergic pathways in the brain, known to play a role in the processing and learning of food reward. Activation of dopaminergic neurones in the brain has been observed in animals fed with a high-sucrose diet (Bernal et al. 2009; Malkusz et al. 2012), as well as in human studies in response to consumption of sweet-tasting high glycaemic index foods, or when photographs of these foods are seen (Lennerz et al. 2013; Macdonald et al. 2013). However, as well as sweet-tasting foods, many other stimuli can cause activation in these dopaminergic areas (e.g. low blood glucose caused by excess insulin action), yet these would not be claimed to be addictive.

Professor Rogers also discussed cravings. Although food cravings are often reported alongside so-called addiction, they can also exist independently and may actually be an expression of normal appetite control. Rogers and Smit (2000) conducted a review of the evidence for food cravings and food addiction and concluded that the majority of cases of ‘food addiction’ should not in fact be viewed as addictive behaviour. Instead, it was proposed that desires to eat specific foods are often aroused by habitual cues. These desires are not always classified as cravings (e.g. the desire to have
sugar solution (Avena et al. 2008; Corwin et al. 2011). These studies are often used as evidence for the addictive properties of sugar; however, the negative reactions to withdrawal observed are most likely due to the specific regimens to which the rodents were subjected. Furthermore, the experiments bear little resemblance to the complex and integrated human social behaviour of eating, therefore any claims of relevance to human actions must be made with caution.

As discussed by Professor Macdonald, the brain requires a constant supply of glucose (approximately 6 g/hour) for normal function. Therefore, achieving an adequate intake of carbohydrate-rich foods is the physiological basis of our appetite system and a drive to consume sweet foods is a basic survival function that would be expected to cause activation of reward centres. Experimental studies have found that fructose or glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver glucose consumption only caused an increase in liver. This emphasises that overconsumption of energy in this study is more likely to be the cause of the weight gain, rather than the effect of the consumption of specific sugars (Johnston et al. 2013).

As research on cravings and hyperpalatability does not seem to provide an explanation for the possibility that food could be addictive, it is important that the features of diets that cause overeating are identified and understood. Energy density is one possible feature, with evidence from both human and rodent studies to suggest that the energy density of foods does influence consumption (Stubbs et al. 1995; Mela & Rogers 1998; Ledikwe et al. 2006). This is potentially explained by the lower satiating value calorie for calorie of energy-dense foods.

In addition to the research presented at this symposium, researchers involved in NeuroFAST (an EU project commissioned to investigate the common neurobiology involved in eating behaviour, addiction and stress, www.neurofast.eu) published a consensus opinion (in April 2013) stating that current evidence does not suggest that ‘a single food via a single specific neurobiological mechanism . . . can account for the fact that people overeat and develop obesity’. Furthermore, it was concluded that no food or drink, except for caffeine-containing foods and drinks and alcohol, can cause a substance-based type of addiction (NeuroFAST 2013).

It is plausible that the concept of food addiction may have come to the forefront because we are in an environment where foods that are highly liked and wanted are readily accessible. Reliable identification and measurement of these hedonic processes in human behaviour are important steps for research. However, it is a matter of judgement as to whether the severity of these behaviours justifies an attribution of food addiction, or whether ‘addiction-like eating behaviour’ should be considered as an alternative expression.

In his talk, Professor Rogers also discussed the potential association between binge eating disorder and food addiction. The definition of binge eating refers to features associated with addiction (e.g. lack of control) (APA 2013). Indeed, ratings on the Binge Eating Scale are highly correlated with ratings on the YFAS. Although it seems reasonable to recognise that binge eating and binge eating disorder contain components of addictive behaviours, binge eating is not a major cause of obesity. In the consensus opinion published by NeuroFAST, it was concluded that an addiction-like eating behaviour may, in rare circumstances, be a result of gene mutations that increase hunger (NeuroFAST 2013).

Leptin

The topic explored by Professor Sadaf Farooqi (University of Cambridge) was ‘Reviewing the neuroscience – what do brain techniques tell us?’ She explained that identification of the hormone leptin was a remarkable breakthrough in our understanding of the molecular components of the central systems involved in energy homeostasis. Leptin is a circulating protein, secreted by adipocytes, that could regulate bodyweight through effects on neural circuits. Identification of humans with mutations in the gene encoding leptin and...
characterisation of the associated clinical phenotype have provided insights into the role of leptin-responsive pathways in the regulation of eating behaviour and food reward. Novel genome-wide approaches are increasingly important to expand understanding of the genetic heterogeneity associated with common obesity. The discovery of how genetic variation at individual and population levels contributes to weight gain by acting on neural circuits involved in eating behaviour will drive further understanding of the central pathways involved in energy homeostasis.

Is the term ‘food addiction’ helpful?

Professor Blundell emphasised that the language being used to describe the eating behaviour of obese people makes a clinical condition out of a common eating behaviour. This medical language has implications for the way in which people view their own behaviours and lives. It has been argued that this form of explanation adds a further layer of confusion to the description of obesity for which rational and meaningful explanations already exist. In addition, the use of the term ‘addiction’ implies that eating certain foods has become a biological imperative, minimising the capacity for personal action and disqualifying the role of personal responsibility. This particular argument is also rendered meaningless by the recognition that all food consumption represents a biological imperative.

At an individual level, ‘food addiction’ may not be a helpful term. By attributing overeating to addiction, it removes personal responsibility and undermines self-efficacy, which may be counterproductive in terms of changes in eating behaviour towards healthier eating habits. Ogden and Wardle (1990) reported that dieters who attribute lapses in their diets to poor self-control (e.g. ‘I ate cake because I can’t say no’) are more likely to break their diets than those who attribute lapses to social cues (e.g. ‘I ate cake because I was celebrating’). Preliminary results from a study investigating whether suggesting if the term food addiction helps or hinders someone’s dietary choices indicate that the notion of food addiction leads to an increase in food intake (Lund et al. 2011).

Conclusion

Currently, the notion of food addiction is a hypothesis that lacks conclusive evidence and whose status is controversial. Nevertheless, it is important to understand why people are drawn to consume certain foods and overeat, in order to assist in the development of the best forms of treatment for appetite-related problems. However, the assumption that some foods are intrinsically addictive substances that induce features of drug addiction is not helpful and may need to be reevaluated. Overall, it is essential that the notion of food addiction is differentiated from the complex homeostatic mechanisms involved in controlling food intake and obesity, which have at their core the importance of eating for energy and nutrition. Moving forward, research into the full spectrum of individual differences in neurobiological processes influencing food intake and food choice is required.

No doubt it has been alarming for consumers, as well as scientists, to read the recent pronouncements made confidently by some scientists about the existence of food addiction as a subtype of drug addiction. In fact, the evidence base is partial, controversial and far from conclusive, yet dominated by extravagant claims. Further scientific debate on food addiction is warranted and experimental evidence associated with the concept needs to be evaluated carefully and in context to ensure that messages communicated to the public and reported in the media are accurate and qualified.

For more information, live broadcasts and presentations from the symposium, as well as 10 Key Facts from the event, are available to access online at www.nutrition.org.uk/bnfevents/pastevents/foodaddiction.

Conflict of interest

The author has no conflict of interest to disclose.

References


