

# Can food be addictive? Public health and policy implications

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## ABSTRACT

**Aims** Data suggest that hyperpalatable foods may be capable of triggering an addictive process. Although the addictive potential of foods continues to be debated, important lessons learned in reducing the health and economic consequences of drug addiction may be especially useful in combating food-related problems. **Methods** In the current paper, we review the potential application of policy and public health approaches that have been effective in reducing the impact of addictive substances to food-related problems. **Results** Corporate responsibility, public health approaches, environmental change and global efforts all warrant strong consideration in reducing obesity and diet-related disease. **Conclusions** Although there exist important differences between foods and addictive drugs, ignoring analogous neural and behavioral effects of foods and drugs of abuse may result in increased food-related disease and associated social and economic burdens. Public health interventions that have been effective in reducing the impact of addictive drugs may have a role in targeting obesity and related diseases.

**Keywords** Addiction, food, obesity, public health.

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Submitted 26 August 2010; initial review completed 28 September 2010; final version accepted 9 November 2010

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## INTRODUCTION

The food environment has changed dramatically with the influx of hyperpalatable foods that are engineered in ways that appear to surpass the rewarding properties of traditional foods (e.g. vegetables, fruits, nuts) by increasing fat, sugar, salt, flavors and food additives to high levels (Table 1). Foods share multiple features with addictive drugs. Food cues and consumption can activate neurocircuitry (e.g. meso-cortico-limbic pathways) implicated in drug addiction [1,2]. Animals given intermittent access to sugar exhibit behavioral and neurobiological indicators of withdrawal and tolerance, cross-sensitization to psychostimulants and increased motivation to consume alcohol [3]. Rats consuming diets high in sugar and fat demonstrate reward dysfunction associated with drug addiction, downregulation of striatal dopamine receptors and compulsive eating, including continued consumption despite receipt of shocks [4].

In humans, diminished striatal dopamine receptor availability and striatal dysfunction have been associated with obesity [5] and prospective weight gain [6]. Foods

and abused drugs may induce similar behavioral sequelae, including craving, continued use despite negative consequences and diminished control over consumption [7]. If foods are capable of triggering addictive processes, applying lessons learned from drug addiction to obesity, associated metabolic problems and diet-related diseases would suggest policy directions and prevention and treatment interventions [2,8].

## SUBSTANCE-RELATED FOCUS

Genetic and environmental (e.g. psychosocial) factors contribute to drug addiction. These factors can interact with drugs that may directly alter brain function, reinforce drug-seeking behaviors and shift attention to substance-related cues; i.e. substances may promote repeated over-consumption [9]. Although an acknowledgement of personal responsibility for one's behaviors remains an important component of many addiction interventions, progress was made in addressing drug addiction when focus changed from blaming individuals with addictions to understanding that drugs may 'hijack'

**Table 1** Composition of traditional and hyperpalatable foods.<sup>a</sup>

Food	Portion size	Type of food	Sugar	Fat	Sodium	# of ingredients
Apple	1 medium	Traditional	19 g	0 g	2 mg	1
Chicken breast, roasted	3 ounces	Traditional	0 g	3 g	63 mg	1
Lettuce	1 cup shredded	Traditional	0 g	0 g	10 mg	1
Tomato	1 medium	Traditional	3 g	0 g	6 mg	1
Orange	1 cup, sections	Traditional	17 g	0 g	0 mg	1
Coca-cola	1 can	Hyperpalatable	39 g	0 g	45 mg	6
Dairy queen chocolate ice cream cone	1 medium cone	Hyperpalatable	34 g	10 g	160 mg	22
McDonald's French fries	1 medium	Hyperpalatable	0 g	19 g	270 mg	9
Cinnamon toast crunch cereal	3/4 cup (milk not included)	Hyperpalatable	10 g	3 g	217 mg	27
DiGiorno pepperoni pizza	1/6 of pizza	Hyperpalatable	7 g	13 g	910 mg	8

<sup>a</sup>Foods were chosen based on their inclusion in the United States Department of Agriculture (USDA) report on foods commonly eaten in the United States. All nutrition information is based on the USDA Nutrition Facts, nutrition information from the company website or nutrition information provided on the product's packaging.

brain circuitry. A similar conceptual shift may help in the food and obesity arena.

Consider tobacco. It can be argued that for years tobacco companies emphasized personal responsibility over corporate responsibility for developing addictive products. This perspective probably delayed drug-related interventions and policy changes by focusing attention on individual-based treatments [10]. Although individual-focused treatments for drug addictions are helpful and cost-effective [11], a more constructive view of tobacco-related behaviors ultimately also incorporated a focus on the addictive drug and implemented bold legal and policy alterations to the tobacco environment (e.g. taxation, limits on marketing and access and actions of the state attorneys general).

Initial approaches to obesity and associated metabolic disorders focused primarily on individual risk factors (e.g. genetics, personal responsibility and individual behavior change) [12], mirroring early 'individualistic' approaches to tobacco use that had important but arguably limited public health impact. Little attention has been given to how the engineering and marketing of food may interact with possible risk factors to generate brain responses similar to those of traditional drugs of abuse. If hyperpalatable foods have a fraction of the effects of addictive drugs, the public health significance could be substantial because of widespread access and exposure to highly marketed, low-cost, nutrient-poor and calorie-dense products. If the biological effects approach those of addictive drugs, far-reaching policies may be indicated. Given the public health impact, attention should be given to the properties of foods and industry's responsibility in creating them.

## PUBLIC HEALTH PERSPECTIVE

Considering addictions within a public health model is important. A sizable proportion of the population

develops addictions, and an additional proportion experiences 'subclinical' problems with addictive substances, resulting in significant social cost. For example, although 12.5% of Americans develop alcohol dependence [13], alcohol misuse contributes to 4.0% of the global burden of disease [14]. With food, the public health significance may not occur solely from a relatively small group who might become clinically dependent on foods, but from the probably larger group of adults and children who overeat enough to compromise their health. Reports of emotional eating, strong food cravings, difficulty controlling high-calorie food consumption despite known consequences and subclinical binge eating are widespread, with health-care costs associated with being overweight or obese projected to exceed 850 billion dollars annually by 2030 in the United States alone [15]. To reduce these costs, it will be necessary to focus beyond personal responsibility or clinical disorders, a lesson learned from addressing nicotine and drug use. Policy focused on changing the availability, attributes and costs of tobacco products has resulted in significant public health gains. Similar environmental interventions may be needed to reduce problematic over-consumption of potentially addictive foods.

## DIVERGENT APPROACHES

Contrasts between historical tobacco-related versus current food-related interventions are both striking and illustrative. First, the cost of tobacco products in the Western world has increased, due primarily to taxation and discontinued government subsidies [16]. In contrast, ingredients for potentially addictive foods (e.g. corn, sugar) are inexpensive because they are heavily subsidized by many governments. Suggestions to tax hyperpalatable foods, such as soda, are currently being debated [17]. Evidence from tobacco suggests that increasing the price of hyperpalatable foods through taxation and

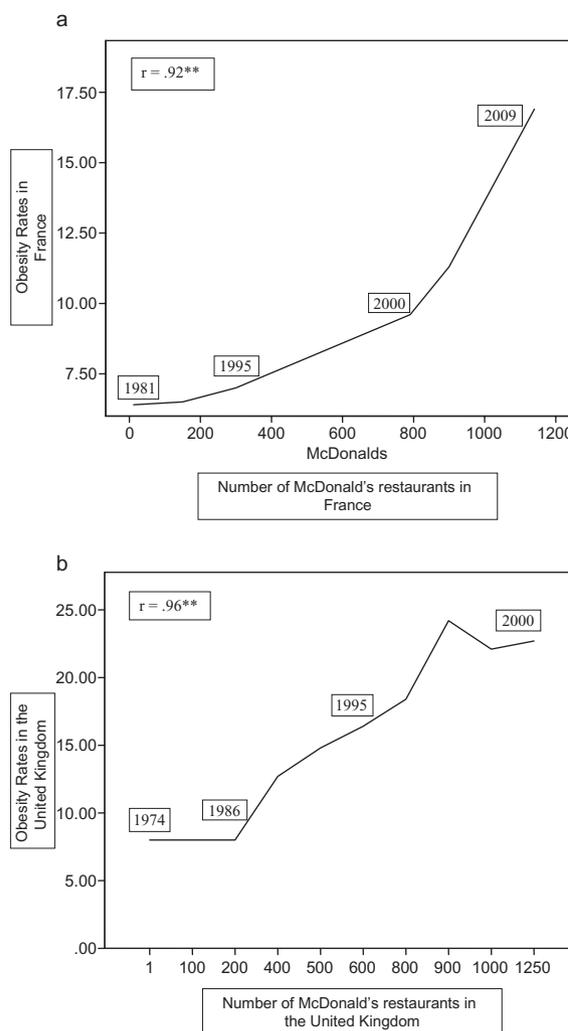
shifting subsidies could have beneficial effects on consumption. Secondly, restrictions placed on marketing tobacco directly to children have contributed to reduced tobacco use. In contrast, hyperpalatable foods are the most frequently marketed products specifically targeting children and adolescents [18]. Food advertising has become increasingly difficult for parents to monitor, given the increase in product placements, 'advergaming' (i.e. the use of videogames to promote products or ideas) and school-related marketing enterprises [19]. Following the tobacco precedent, restricting childhood exposure to advertising of potentially addictive foods may be an important public health strategy.

In addition to cost and marketing issues, accessibility is another critical factor in limiting tobacco use. Cigarettes were once widely sold in vending machines in public locations. In addition to providing greater general access, tobacco vending machines provided a major point of access for minors to purchase cigarettes illegally [20]. As of 2003, most American states have restricted the use of tobacco vending machines [20] and similar regulations limit accessibility to alcohol, with greater restrictions for more potent alcoholic beverages. Beer is typically more widely available for purchase (e.g. in gas stations, grocery stores) and subject to less taxation than liquor. Alcohol potency is associated with abuse potential; hence, liquor sales are sometimes restricted to state-run stores and subject to higher taxes [21]. In contrast, foods with lower nutritional value and arguably greater abuse potential (i.e. high sugar, high fat) are typically more widely available and cost less than foods with higher nutritional value and arguably lower abuse potential (i.e. fruits, vegetables) [22]. Based on approaches to alcohol, food-related problems may be diminished by reducing the availability of less nutritious, hyperpalatable foods while increasing access to healthier ones.

## GLOBAL IMPACT

Another important issue is the global marketing and sale of addictive products. Facing declining sales in the western world, tobacco companies appeared to become more aggressive elsewhere. As tobacco use decreased by approximately 50% over the past three decades in the United States, it increased simultaneously by 3.4% per a year in developing countries [23]. As the diet of hyperpalatable, heavily marketed foods becomes a global phenomenon, protective policies across nations warrant consideration.

Obesity rates have been rising rapidly throughout the world, first in developed nations and more recently in poorer countries. Although many contributing factors may exist, the changing food environment warrants particular attention. For example, obesity rates in countries



**Figure 1** (a) Temporal plots of obesity rates and McDonald's fast food venues in France. All obesity data were acquired from the World Health Organization and the United Kingdom Health Behaviour Survey. All McDonald's data were acquired from press releases, McDonald's fact sheets, Fantasia 1995 [24] and DeBres 2005 [25].  $**$ Correlation is significant at 0.01 level (one-sided). (b) Temporal plots of obesity rates and McDonald's fast food venues in the United Kingdom  $**$ Correlation is significant at 0.01 level (one-sided)

such as France and the United Kingdom have been rising in parallel with increases in availability of highly processed foods and fast-food chains [24,25] (Fig. 1a,b). Similar trends have been found between sugar-sweetened beverage consumption and obesity rates [17], with increased sugar-sweetened beverage consumption prospectively predicting obesity in children [26]. Countries that have historically been successful in reducing diet-related disease, such as Finland, have seen rising obesity rates in the current food environment [27]. As food markets become more global, trade boundaries between countries become more porous, allowing for a greater influx of hyperpalatable foods. Traditionally, addiction

prevention across borders (e.g. supply-focused efforts to restrict drug trafficking) has been challenging and costly, and applying lessons learned from such international endeavors could be valuable. As food advertising focuses increasingly on global forms of media, such as the internet and product placements in film, it becomes increasingly difficult for any single government to regulate food marketing effectively. As with tobacco, global interventions may reduce the world-wide impact of potentially addictive foods most effectively.

## RELEVANT DIFFERENCES

Although foods share characteristics with addictive drugs, important differences exist. Unlike drugs, foods are necessary for survival. The essential nature of eating contrasts with the use of traditionally addictive substances and complicates food-related interventions. Multiple addictive drugs include few ingredients, and the addictive component has been identified (e.g. ethanol, heroin). In contrast, hyperpalatable foods typically include multiple ingredients, and research into which components may be addictive is at a relatively early stage. Policy and regulatory efforts will be aided by research into which food elements may trigger addictive processes. Such information may help to generate improved interventions early in development. As foods are consumed more frequently and earlier in life than are abused drugs, early and repeated exposure during childhood may have long-lasting effects and prevention strategies targeting youth could have important implications as people mature.

## SUMMARY

Foods, particularly hyperpalatable ones, demonstrate similarities with addictive drugs. Although the potential addictive nature of foods may not fully explain obesity or excessive food consumption, important lessons learned from drug addictions can inform methods to reduce escalating food-related problems and the associated personal, public health and economic costs. Corporate responsibility, public health approaches, environmental change and global efforts all seem essential in reducing food- and substance-related problems. Such approaches could be enacted in conjunction with individual-focused behavioral and pharmacological efforts that could also benefit from considering similarities between food-related conditions such as obesity and drug addiction [2,8]. Ignoring the analogous neural and behavioral effects of foods and drugs of abuse may result in a substantial loss of time, resources and lives, as we rediscover lessons learned in reducing the impact of addictive substances.

## Declaration of interests

All authors report no conflict of interest with respect to the content of this paper. Dr Potenza has received financial support or compensation for the following: Dr Potenza consults for and is an advisor to Boehringer Ingelheim; has financial interests in Somaxon; has received research support from the National Institutes of Health, Veteran's Administration, Mohegan Sun Casino, the National Center for Responsible Gaming and its affiliated Institute for Research on Gambling Disorders and Forest Laboratories pharmaceuticals; has participated in surveys, mailings or telephone consultations related to drug addiction, impulse control disorders or other health topics; has consulted for law offices on issues related to addictions or impulse control disorders; has provided clinical care in the Connecticut Department of Mental Health and Addiction Services Problem Gambling Services Program; has performed grant reviews for the National Institutes of Health and other agencies; has guest-edited journal sections; has given academic lectures in grand rounds, CME events and other clinical or scientific venues; and has generated books or book chapters for publishers of mental health texts.

## Acknowledgements

This research was supported by the National Institutes of Health grants P50 DA016556, UL1-DE19586, K24 DK070052, RL1 AA017537 and RL1 AA017539, the Office of Research on Women's Health, the NIH Roadmap for Medical Research/Common Fund, the VA VISN1 MIRECC and the Rudd Center. The contents are solely the responsibility of the authors and do not necessarily represent the official views of any of the other funding agencies. We would like to thank Carolyn Mazure, Rajita Sinha and other members of the Yale Interdisciplinary Research Consortium on Stress, Self-Control and Addiction for helpful discussions and suggestions leading to the generation of this manuscript.

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## Commentaries on Gearhardt *et al.* (2011)

### OBESITY – IS FOOD ADDICTION TO BLAME?

Gearhardt and colleagues [1] propose that certain foods are addictive and that this has important implications for efforts to combat obesity. Their argument is that shifting blame for overeating from the individual to the availability of ‘hyperpalatable’ addictive foods leads to the result that ‘corporate responsibility, public health approaches, environmental change and global efforts all warrant strong consideration in reducing obesity and diet-related disease’ (p. 1208). Undoubtedly, the rising tide of obesity world-wide is a major challenge of our time, and Gearhardt *et al.*’s [1] analysis adds to the many calls for action to be commensurate with the size of the problem. Their contribution is to draw parallels with interventions including taxation and curbs on marketing, which have been successful in reducing tobacco use, in particular.

Claiming that foods such as popular products of fast-food restaurants can be addictive is provocative, and might help to provoke policy changes, but can obesity really be understood and treated as food addiction? Of course, this depends partly on how addiction is defined. That seeing and eating liked foods activates brain circuitry also activated by addictive drugs is unsurprising if these drugs are ‘highjacking’ pathways that evolved to regulate dietary behaviour. This issue is, perhaps, one concerning the relative potency of foods and drugs consumed by people (and differences in food-related brain activity associated with or predictive of obesity may be consequences rather than causes of the obese state or eating behaviours that precede it). It would seem reasonable to label binge eating, as occurs in bulimia nervosa and binge eating disorder, as food addiction (e.g. there is compulsion, marked change in affect and long-term harm, and physiologically based satiety tolerance enables escalation of food ‘dose’); however, the route to obesity is generally a rather modest average daily excess of energy intake over energy expenditure.

What, then, are the characteristics of food that increase consumption (of calories)? Gearhardt *et al.* [1] identify palatability as a key factor. They label certain foods as ‘hyperpalatable’ and contrast these with some ‘traditional’ foods. All else being equal, we eat more of what we like, but it is far from clear that the foods listed by Gearhardt *et al.* [1] vary as much in palatability as they imply—for this author, an orange (‘traditional’) is as palatable as sweetened breakfast cereal (‘hyperpalatable’). In Italy, at least, pizza is very much a traditional food. It may

also be highly liked, although the prevalence of obesity in Italy is among the lowest in Europe. The difficulty is that there is a lack of objective evidence for increases in food palatability paralleling increases in obesity rates [2], and there also seems to be rather little variation between people and foods in the palatability of what is typically eaten [2]. Note that palatability (the hedonic or liking response to oro-sensory stimulation during eating) is modified by individual eating experiences and is not a fixed property of individual foods.

Rather than palatability, a key characteristic of the foods identified by Gearhardt *et al.* [1] that poses a risk of over-consumption is their relatively high energy density. Two inter-related features of energy-dense foods would appear to be important here. They are more attractive; that is, they have greater incentive salience, because for the same portion (per unit volume or weight) their capacity to satiate (satisfy hunger) is greater than foods lower in energy density. However, on a calorie-for-calorie basis they are less satiating, and therefore they are selected in larger (calorie) portions than energy-dilute foods, irrespective of palatability [3] (palatability was found to be unrelated to energy density [3]). Reducing consumption of energy-dense foods could involve the approaches advocated by Gearhardt *et al.* [1], as changes in cost, accessibility, serving size and even prevalence of eating while engaged in other activities may all contribute significantly to the risk of obesity. Additionally, it might be possible to formulate foods to increase their satiating capacity (calorie-for-calorie), and to enhance the attractiveness of lower-calorie, less energy-dense foods.

#### Declaration of interest

Peter Rogers has received funding for research from UK food and pharmaceutical companies, UK research councils and the European Commission.

**Keywords** Energy density, food, incentive salience, liking, obesity, palatability, satiety.

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## FOOD AND ADDICTION – SUGARS, FATS AND HEDONIC OVEREATING

'Food addiction' has been postulated as one possible cause of the obesity epidemic [1–4]. This has been a controversial topic, with many wondering whether it is appropriate or even reasonable to categorize food, which is something we all need to consume in order to survive, with drugs of abuse, which are generally considered along with other 'natural reinforcers' such as sex, to be very different from gambling, alcohol and illicit drugs. However, there has been mounting evidence that supports the idea of 'food addiction'. Preclinical studies, beginning in Bart Hoebel's laboratory at Princeton University, have shown that rats overeating a sugar solution develop many behaviors and changes in the brain that are similar to the effects of some drugs of abuse [5,6], including naloxone-precipitated withdrawal [7], and others have shown complementary findings that suggest reward dysfunction associated with addiction in rats that overeat highly palatable foods [8]. These studies are supported by clinical research showing similarities in the effects of increased body weight or obesity and abused drugs on brain dopamine systems, as well as the manifestation of behaviors indicative of addiction [9–12].

Gearhardt and colleagues [13] ask the next important question in furthering our understanding of 'food addiction': if food addiction is real, what should we do about it? Drawing on examples from what we have learned from efforts to reduce tobacco use, the authors discuss potential approaches to reduce excessive intake of 'hyperpalatable' foods that contribute to obesity. Advertising, availability, public health and cost-related measures are discussed, each of which has proved successful with tobacco and alcohol. If these policy measures could similarly reduce the incidence of obesity and its concomitant threats to health and wellbeing, it would be of great importance, as the damaging effects of obesity are even more widespread than those of tobacco.

Gearhardt *et al.* address the need for additional research to understand the effects that specific components of 'hyperpalatable' foods have on the development of addiction. Indeed, it is important for researchers to refine the terminology associated with the study of 'food addiction'. Clearly, not all foods would be candidates for addiction: Gearhardt *et al.* argue that 'hyperpalatable' foods rich in fats, sugars and/or salts, which are often comprised of synthetic combinations of many ingredi-

ents, may have greater addictive potential than traditional foods such as fruits, vegetables and lean protein. We know from studies of feeding behavior that different nutrients can affect specific brain neuropeptide and neurotransmitter systems [14,15]. Further, preclinical studies suggest that overeating sugar produces different addiction-like behaviors compared with overeating fat [5]. There is also a nutrient specificity in the effect that some pharmacological treatments have on reducing overeating [16,17]. Thus, additional knowledge of how different food elements activate brain systems that affect addiction-like behavior will be crucial to developing targeted interventions for people who exhibit signs of 'food addiction'. Further, pharmacotherapies may be aimed at reducing resultant reinforcing effects of 'hyperpalatable' foods, rather than hunger or eating behavior *per se*. This may give rise to a treatment paradigm in which the reduction of body mass may depend on reducing reinforcement and attachment for certain foods.

In conclusion, Gearhardt *et al.* have taken a controversial, yet important and emerging line of research and brought it to the forefront for us to consider on a more global level. While 'food addiction' certainly does not explain all obesity, it seems that the overwhelming interest that many individuals have in eating, for reasons other than energy intake, suggest that it is no longer only for survival. With the soaring number of individuals affected by obesity, many whom are children, we need to begin to consider alternatives to traditional efforts for combating this often deadly and costly condition. Perhaps 'food addiction' will soon join other non-drug addictions, such as sexual compulsivity and gambling.

### Declaration of interest

None.

**Keywords** Dopamine, food addiction, obesity, overeating, palatable food, preclinical models.

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## MATERNAL DIET AND OFFSPRING DEVELOPMENT

The paper by Gearhardt and colleagues [1] makes a valuable contribution to the debate about hyperpalatable foods, their abuse potential and how they impact the increasing prevalence of obesity. Importantly, they discuss how strategies that have successfully lessened the public health consequences of drug addiction may be applied to the toxic food environment. They also address relevant differences — for example, that drug exposure

generally occurs in our teen years, while consumption of foods unnaturally high in fat, sugar and salt begins earlier in life, and typically has a chronic course. Consequently, they argue for intervention policies aimed largely at children and adolescents. In this author's view, however, that may be too late.

There is now compelling evidence that *in utero* events can have long-lasting, and sometimes dire, consequences for the offspring [2–4]. We have learned recently how maternal diet can modify the fetal genome substantially and contribute to deleterious health outcomes for the developing child. For instance, high-fat consumption during pregnancy has been shown to induce long-term alterations in dopamine and opioid gene expression in animal offspring, and to enhance their preference for palatable foods [5]. In addition, there is evidence that children of obese mothers are at increased risk for insulin resistance and subsequent obesity and metabolic dysfunction [6].

Using data collected from three affluent Scandinavian societies, a prospective study also showed, for the first time, that maternal obesity increased the risk of having a child with symptoms of attention deficit hyperactivity disorder (ADHD) compared to children of normal-weight mothers [7]. Moreover, these findings persisted after controlling for baby's birth weight, maternal age and mother's smoking status. To test whether this relationship occurred because a genetic predisposition accounted for both the mother's weight status and the subsequent ADHD symptoms in the child, a large replication study extended this research. Again, it was found that children of obese mothers had a twofold increase on teacher-rated inattention scores compared to those of normal-weight mothers [8]. These associations also remained statistically significant after controlling for ADHD symptoms in both parents.

Of relevance to these findings are the strongly established links between prenatal alcohol exposure and symptoms of ADHD [9] and the high co-occurrence of ADHD symptoms and obesity, both in children and in adults [10]. It is noteworthy in this context that alcohol and sugar are biochemically congruent substances, because ethanol is simply the fermented by-product of fructose [11]. In our current food environment, with its superfluity of highly palatable foods, mothers who are overweight and obese are likely to consume larger and more frequent quantities of sweet foods than their normal-weight counterparts. Currently, high fructose sweeteners are used liberally in most of the processed foods we eat, and in much greater amounts than are found naturally in fruits and vegetables [12]. When taken in large quantities, both alcohol and fructose have considerable abuse potential due to their potent activation of brain reward pathways [13,14]. They can also

foster neuroadaptations that lead to compulsive use and dependence similar to other drugs of abuse [15,16]. It is therefore plausible that highly processed foods taken in abundance during pregnancy could contribute to deleterious outcomes for the unborn child such as those seen in the studies described above. In other words, excessive maternal ingestion of sweet foods could produce what I will loosely call a 'fetal sugar spectrum disorder', with symptoms that are not dissimilar to those seen in the offspring of women who drank alcohol during their pregnancy. Because postnatal life appears to be linked inextricably with environmental influences during fetal development, and because approximately 20% of women of reproductive age are obese, it seems of utmost importance to target pregnant women for prevention measures in a manner similar to the aggressive warnings about gestational alcohol use. It will behave health-care providers to issue strong nutritional guidance to women during pregnancy to avoid events that may have irreversible consequences for their children later in life.

#### Declaration of interest

None.

**Keywords** *In utero*, nutrition, obesity, pregnancy.

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#### FOOD ADDICTION NOT HELPFUL: THE HEDONIC COMPONENT – IMPLICIT WANTING – IS IMPORTANT

The concept of food addiction threatens to add a further level of confusion to our apparent inability to account for conspicuous overweight and obesity at the societal level, and to explain why some people eat well beyond any biological requirement for nutrients. The question posed by Gearhardt *et al.* [1] has both political and scientific connotations, and we comment on each of these aspects in turn. At the outset it should be considered that over-consumption of food is one example of a more widespread acquisition of material objects well beyond any limits defined by personal need. People in industrial societies are encouraged to purchase more clothes, shoes, TVs, motor cars, refrigerators, furniture and palatable foods; only the last of these is strongly blamed for obesity. However, the acquisition of possessions beyond need extends well beyond the food repertoire. The prevailing socio-economic system encourages a philosophy of materialistic self-interest and unnecessary consumption (and purchasing) which is required in order to drive economic growth. Therefore, over-consumption takes place in a climate of abundance, aggressive advertising and easy accessibility in which food consumption is promoted strongly by the socio-economic market. In addition, it is undeniable that the food industry strives to

produce foods of ever increasing palatability in order to make them more attractive to the consumer (this is not an industrial secret). Over-consumption is legitimized, and not prohibited, by the prevailing cultural values. Given this situation (abundance, palatability and promotion), together with the operation of a powerful and well-functioning reward system in the brain, it is a surprise to us that the level of obesity is not even higher than its current level. The human biological system permits over-consumption but strongly opposes under-eating [2], and the gain of adipose tissue creates a positive feedback (in which satiety is impaired) rather than a negative feedback [3]—via insulin [4] and leptin resistance [5]—in which appetite would be suppressed [6]. Over-consumption, which can be witnessed by the behaviour of people in any airport, food mall and high street in much of the United States and Europe, fits very comfortably into the environment in which it takes place. People are not expending energy or making any effort to seek out palatable foods or to eat them furtively far from the public gaze (except for people with binge eating disorder), features which lessen the proposed resemblance to drug consumption. Therefore, at the level of phenomenological description, a term such as food addiction is not required to explain the massive intake of eatables in many of our societies.

However, this categorical argument bypasses the scientific issues raised by the question posed. One of these concerns the strength and function of hedonic processes that influence the motivation for specific foods. Although we feel that the term 'food addiction' is inappropriate if applied universally to account for the eating behaviours of many millions of citizens, the hedonic processes implicated may be relevant to a much smaller number of individuals who display behaviours related to substance dependence [7,8]. One relevant issue is the distinction between liking and wanting [9]. Separate neural pathways exist to mediate these processes [10,11], and experimental human methods have been developed to distinguish between the operation of these processes and to measure them separately [12–14]. It is frequently said that maintained drug consumption involves a wanting but not a liking for the target substance. A key component of our experimental platform (Leeds Food Preference Questionnaire) is the capacity to measure, through separate experimental procedures, explicit liking and implicit (covert) wanting [12]. In susceptible individuals at risk of weight gain [15], and defined by scores on the Binge Eating Scale [16], those participants with a high tendency to binge eating showed an increased liking for all food categories of foods but an increase in implicit wanting only for high-fat sweet foods [17]. These foods could be defined as highly palatable (at least for these individuals), and the implicit wanting scores correlated

highly with the amounts of these foods freely selected and consumed [Finlayson, Arlotti, Dalton, King & Blundell; unpublished date]. People who did not show any tendency for binge eating did not display an implicit wanting for any type of food. Therefore, our view is that a hedonic process operating at an unconscious, compulsive level (measurable by implicit wanting), which appears to be regarded as one feature of drug taking, is one component that is apparent in the motivational repertoire of a susceptible phenotype for over-consumption. However, a point of departure is that enhanced implicit wanting for some foods occurs in the presence of a maintained level of overall liking for foods. We feel that the measure of 'implicit wanting' shows great promise as an objective and quantifiable index of uncontrollable eating (not dependent on self-report) which could be used to investigate further the role of hedonic processes in susceptibility to over-consumption [17]. It is a matter of judgement whether or not the presence of implicit wanting for a food justifies an attribution of food addiction.

#### Declarations of interest

None.

#### Acknowledgement

The authors acknowledge support from BBSRC grant: BB/G005524/1 – Drivers of eating behaviour during chronic overconsumption. International Standards Randomized Control Trial Number – ISRCTN47291569.

**Keywords** Cultural values, food addiction, hedonics, implicit wanting, over-consumption, socio-economic perspective.

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### **ALL FOODS ARE HABIT-FORMING – WHAT I WANT TO KNOW IS WHICH WILL KILL ME!**

Gearhardt *et al.* [1] are right to point out that ‘food addiction’ is much more than a metaphor, owing to the fact that the neuroendocrine mechanisms underlying the formation of dietary habits are often precisely those acted on by drugs of addiction. Just as exposure of tobacco companies’ explicit efforts to increase the addictive potential of their products helped to generate public support for stricter

regulation in the United States, evidence of similar actions by food producers may well serve the same purpose.

It is telling, however, that the taxes and restrictions on marketing eventually placed on tobacco were applied to *all* tobacco products, regardless of how addictive or carcinogenic they happened to be [2]. An analogous outcome for processed foods, I would argue, is not feasible. The cost efficiencies inherent in industrial-scale food production are enormous, making it unrealistic to expect most consumers to return anytime soon to the days of fresh fruits, vegetables and meats purchased directly from the local grower. Every one of the remedies the authors mention—taxation, subsidies, restrictions on marketing, availability or trade, and even corporate responsibility—will require the development of very specific rules that differentiate ‘bad’ products (which will be subject to regulation) from ‘good’ (which presumably will not). The food industry has a long history of reformulating products in response to conditions in the marketplace [3–5], and public health improvements will hinge critically on the extent to which producers are induced to deliver healthier industrial foods to the consumer.

With regard to choosing the particular product characteristics that most damage public health, ‘hyperpalatability’ seems a poor candidate. There is very little evidence that hyperpalatability, considered in isolation, causes illness. It can be argued that today’s highly processed mass-marketed foods have been (perhaps unintentionally) designed to induce a biological addiction event. However, the reason industry methods succeed so persistently is that they take advantage of evolved predilections that served important adaptive purposes in the pre-industrial world, and probably still do [6,7]. There is every reason to expect that people also become ‘addicted’—in both the physiological and behavioral senses of the word—to *any* delicious food (healthy or not), experienced in the right context.

Consider again the case of tobacco. Instead of targeting the leaf itself, public health advocates could have sought to place restrictions on particular characteristics, such as nicotine or ‘tar’; but scientific uncertainty regarding the contribution of particular product characteristics to addiction or health outcomes would have provided easy fodder for defensive legal action by the industry. Moreover, singling out tobacco’s most potent addictive properties might well have exacerbated the public health problem, by inadvertently stimulating sales of ‘bad’ versions of the product, as sometimes occurs with alcohol content regulation [8,9]. It is not hard to imagine restrictions on the ingredients that make processed foods ‘hyperpalatable’ generating a similar response.

Unfortunately, there does not appear to be a consensus view among public health advocates as to what ‘healthier’ processed foods might look like. It has been

variously argued, for instance, that obesity prevention efforts should focus on calories [10], fats and sugars [11], refined carbohydrates [12,13] or on industrial processing in and of itself [5,14–16]. If the food industry is to be pushed, by regulation or public pressure, into developing healthier products, the development of clear parameters by which their products will be judged should become a priority of the research community.

There is one sense in which reforming the food industry will be easier than the fight over tobacco. Although years of mandatory health warnings proved to be largely ineffective for tobacco, the same is not likely to be true for food products—as long as ‘healthy’ does not become synonymous with ‘unpalatable’. Consumers have a long history of gravitating towards healthy foods when quality has been easily discernable [5], and taxes or subsidies (which would probably be ineffective anyway [17,18]) are unlikely to be necessary.

That the food industry uses modern technology to enhance sales is unsurprising. It is tempting to hope that restricting some of their more egregious practices will improve public health, and perhaps it would; but real change will come only when the public health community develops a clear vision of what efficiently produced healthy foods might look like.

#### Declaration of interest

None.

**Keywords** Economics, food addiction, food industry, tobacco.

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#### IMPORTANT NEXT STEPS IN EVALUATING FOOD'S ADDICTIVE POTENTIAL

These commentaries highlight important issues about the potentially addictive nature of foods. Although further evidence for the validity of food's addictive potential were discussed, including neurobiological and behavioral indicators of addiction in animal models [1], behavioral markers of addiction in eating disorders [2], shared neuroendocrine responses in the consumption of foods and drugs [3] and similar *in utero* influences of addictive drugs and processed foods [4], concerns were also raised. Blundell & Finlayson suggest [5] that although factors associated with food addiction (such as implicit wanting) may be useful, the concept of food addiction may increase confusion surrounding obesity. We suggest that implicit wanting is an example of a common behavioral mechanism and represents part of a repertoire of behavioral elements underlying food addiction. If an addictive descriptor for foods is supported empirically, novel legal, educational and policy

approaches to creating a healthier food environment may warrant stronger consideration. Such approaches could help to better educate, inform and protect consumers.

Concerns were raised that food addiction may only be relevant to eating disorders because the general public does not appear to expend excessive energy in their consumption of palatable foods [5], and obesity is associated typically with only a modest increase in food consumption, a pattern seemingly different from drug use in addictions [2]. However, some addictions (e.g. tobacco-related) involve low-to-moderate intake of easily accessible substances that over time have deleterious consequences. Importantly, the addictive properties of some foods probably apply to a great many people—that is, not only to those with ‘clinical’ levels of obesity and/or disordered eating. As such, foods may contribute to poor diets and poor health associated with nutritional deficiencies. Indeed, obesity and malnutrition frequently co-occur. Thus, from a public health viewpoint, we believe that the combination of an addictive substance with easy accessibility represents a dangerous public health combination, as seen with tobacco and alcohol. Further, these factors may make the likelihood of excess consumption and subclinical problems especially widespread. For example, a relatively small percentage of the public consumes alcohol in an addictive manner [6], but the rewarding nature, widespread availability and social acceptance of alcohol may increase the subclinical impact of alcohol-related consequences [7].

The commentaries also highlighted important future directions. Authors emphasized the importance of identifying which ingredients in foods may be addictive [1–4]. Whether specific macronutrients (e.g. fat, sugar), food additives (e.g. salt, high-fructose corn syrup) or food characteristics (e.g. palatability, calorie content), or combinations thereof (e.g. caffeine and sugar), influence the potentially addictive nature of certain foods is an important empirical question. Such questions may have important developmental implications for foods as with drugs [e.g. flavored cigarettes (bidis) and adolescent smoking] [8]. The food addiction concept will have limited impact on public health on policy or industrial food reformulation unless there is a scientifically informed answer to this question. In summary, the growing evidence that certain foods may be capable of triggering an addictive process suggests that applying lessons learned in reducing the impact of addictive substances may advance public health strategies addressing obesity.

#### Declarations of interest

All authors report no conflict of interest with respect to the content of this paper. Dr Potenza has received financial support or compensation for the following: Dr Potenza consults for and is an advisor to Boehringer

Ingelheim; has financial interests in Somaxon; has received research support from the National Institutes of Health, Veteran’s Administration, Mohegan Sun Casino, the National Center for Responsible Gaming and its affiliated Institute for Research on Gambling Disorders, and Forest Laboratories pharmaceuticals; has participated in surveys, mailings or telephone consultations related to drug addiction, impulse control disorders or other health topics; has consulted for law offices on issues related to addictions or impulse control disorders; has provided clinical care in the Connecticut Department of Mental Health and Addiction Services Problem Gambling Services Program; has performed grant reviews for the National Institutes of Health and other agencies; has guest-edited journal sections; has given academic lectures in grand rounds, CME events and other clinical or scientific venues; and has generated books or book chapters for publishers of mental health texts.

#### Acknowledgements

This research was supported by the National Institutes of Health grants P50 DA016556, UL1-DE19586, K24 DK070052, RL1 AA017537, and RL1 AA017539, the Office of Research on Women’s Health, the NIH Roadmap for Medical Research/Common Fund, the VA VISN1 MIRECC and the Rudd Center. The contents are solely the responsibility of the authors and do not necessarily represent the official views of any of the other funding agencies.

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