The neurobiology of overeating

Treating overweight individuals should make use of neuroscience research, but not at the expense of population approaches to diet and lifestyle

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Excess adiposity—being overweight or obese—is a rapidly growing global public health problem that contributes to 2.8 million deaths per year and has become the fifth leading cause of premature death [1]. Being seriously overweight is a major risk factor for chronic diseases and is responsible for 44% of the global disease burden attributed to diabetes, 23% of ischaemic heart disease and 2% of all cancers [1]. At the same time, obesity has become an epidemic in itself: in 2008, 1.5 billion adults over 20 years of age were classified as overweight globally, and another 500 million as obese [1], and the numbers have been growing. In the USA, more than two-thirds of all adults are overweight and half of these are obese. Similar patterns are apparent in many other developed countries, including Australia, the UK and Canada [1], and even in developing countries.

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The prime cause of being overweight is an energy imbalance resulting from chronic excessive caloric intake coupled with a deficit in caloric expenditure—in other words: too much food and too little exercise. Other indirect causes include obesogenic environments that promote the overconsumption of sugary or fatty foods, genetic susceptibility, biological dysfunctions and psychosocial factors such as depression,

low socioeconomic status or substance abuse [2]. The link with substance abuse, as well as similarities between the behaviour of those who overeat and those who abuse drugs or alcohol, has led to the suggestion that overeating ought to be treated as a form of ‘food addiction’. Advocates of the ‘food addiction’ hypothesis argue that its acceptance could lead to an important shift in our perception of and responses to obesity, both clinically and in public health policy, and it could have implications for the treatment and prevention of obesity and the chronic diseases that follow.

Obesity, which the World Health Organization defines as a “condition of abnormal or excessive fat accumulation in adipose tissue, to the extent that health may be impaired”, is commonly measured using the body mass index (BMI). A person with a BMI score of 25 to 29.9 is considered ‘overweight’ and a BMI of 30 or greater is termed ‘obese’. Obesity is not included as an eating disorder in the American Psychiatric Association’s Diagnostic and Statistical Manual of Mental Disorders 4th Edition (DSM-IV), which classes eating disorders as “severe disturbances in eating behaviour”. At present, eating disorders in the DSM-IV include anorexia nervosa and bulimia nervosa, but some have proposed the inclusion of overeating as an addictive disorder in the upcoming DSM-V [3].

Aside from the policy and treatment implications, it is worth considering the evidence used to argue for such an inclusion. At present, DSM-IV defines substance dependence as meeting three or more of the following seven criteria: tolerance; withdrawal; substance taken in larger

amounts or for longer than intended; persistent desire to reduce intake or unsuccessful efforts to limit or control use; large amount of time spent obtaining, using or recovering from the substance; neglect of social, occupational or recreational activities due to use; and continued use despite a “recurrent physical or psychological problem [...] caused or exacerbated by the substance”. Joan Iffland, Chair of the Refined Food Addiction Research Foundation, a charitable organization based in Texas, USA, and co-authors [4] have applied these criteria to the overeating of refined foods by people they classify as ‘food addicts’. As such, they argue that these individuals meet the DSM-IV criteria for substance dependence [4]. There is also supporting neurological evidence for their argument from the observed behavioural similarities between overeating and substance dependence, neurobiological results from animal models, human neuroimaging studies and genetic research on susceptibility to obesity.

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Indeed, the similarities between some forms of overeating and drug addiction might derive from the same imbalance in certain neural pathways that mediate the motivation to eat certain foods or to take drugs, and those that control these urges [5]. The dopamine reward pathway is central to

Fig 1 | Model of brain circuits implicated in drug addiction and obesity. (A) Illustration depicting the structures of the brain involved in addiction and obesity. The nucleus accumbens is thought to be important in identifying stimulants by assessing reward and saliency (which is the feature of a thing that makes it stand out from all others). The orbitofrontal cortex is involved in decision-making and determining the expected rewards and punishments of an action. The amygdala and hippocampus are involved in forming memories of the stimulus/reward relationship, whilst inhibitory control and emotional regulation are provided by the prefrontal cortex and the anterior cingulate gyrus. Addictive drugs and food, particularly in obese individuals, are believed to cause neurons from the ventral tegmental area to release the neurotransmitter dopamine in the nucleus accumbens. These regions regulate activity in the frontal cortical regions. This pathway is referred to as the mesolimbic reward pathway (arrows marked in red). (B,C) Schematics showing the reward pathways in the non-addicted and addicted brain. In a person suffering from addiction, the reward pathway is disrupted such that the prefrontal cortex and cingulate gyrus are no longer controlling factors and compulsive behaviour is driven by the enhanced activation of the reward and saliency and memory and conditioning regions of the brain. As such, when an individual is exposed to the reinforcing stimulant—drug or food—the system goes into overdrive. Figure adapted from Baler RD, Volkow ND (2006) Drug addiction: the neurobiology of disrupted self-control. *Trends Mol Med* 12: 559–566, © 2006 with permission from Elsevier.

this goal-directed behaviour (Fig 1): pleasurable activities such as eating, sex or drug use trigger the release of dopamine into an area of the midbrain called the nucleus accumbens. This release of dopamine signals that these activities are worth repeating. However, the excessive consumption

of food or drugs can dysregulate this system to the point that pursuit of these rewarding activities dominates an individual's behaviour [5]. However, although most abused drugs act directly on the dopamine reward pathway, repeated carbohydrate and sugar ingestion act indirectly by affecting

palatability and increasing insulin and glucose secretion [5]. The opioid neurotransmitter system then links the palatability or pleasure effects of food with the reward system [6]. Endogenous opioids subsequently trigger higher levels of dopamine release in the striatum, thereby reinforcing the consumption of food. Opioids might also reinforce food cravings, as indicated by the analgesic effects of sweet foods and the finding that drugs blocking opioids also reduce food intake [6]. It is the overuse of these endogenous opioids that dampens the system so as to produce the high levels of endorphins observed in obese individuals [6]. In fact, the abnormal neurotransmitter levels found in obese animals and humans are similar to those seen in chronic drug abusers.

Other insights into the neurological basis of obesity come from animal studies of excessive sugar consumption. After periods of fasting, the act of bingeing on sugar triggers excessive levels of dopamine and opioid release in the brain that are associated with pathological consumption. Binge-eating rats will also endure stronger electric foot shock than control rats to access sweet cookies [7]. Similarly, the chronic administration of a high-sugar diet reduces dopamine activity in the limbic reward pathway; a change also associated with an increase in the thresholds necessary to deliver reward as measured by electrical brain stimulation [5]. This evidence suggests that rats chronically exposed to sugar develop a reward deficiency state similar to that seen in cases of drug addiction. The abrupt cessation of chronic sugar consumption also produces similar behavioural symptoms to heroin withdrawal in animals, such as teeth chattering and indicators of anxiety [7]. As with addictive drugs, animals withdrawn from a diet of highly palatable food rapidly reinstate pathological eating after being given food-related cues or being exposed to stress.

Many of the observations made in animal models have been further supported by human neuroimaging studies. Functional magnetic resonance imaging (fMRI) during the presentation of food cues to people with binge-eating disorders, for example, found activation patterns in regions of the brain similar to those seen in addicted persons responding to drug-related cues [5]. Obese individuals also share several personality features with substance abusers. One such study [8] compared food addiction scores on the Yale

Food Addiction Scale (YFAS) with patterns of neural activation that usually indicate substance dependence. The researchers found a significant positive association between YFAS scores and neural activation of the reward pathways in response to food stimuli, and a negative association between YFAS scores and neural activation in inhibitory regions after food intake. An earlier study that similarly used fMRI to examine the neuroanatomical basis of food craving [6], found that participants fed a monotonous diet showed greater craving-related activation in specific brain areas when imagining well-liked foods. Both studies support the hypothesis that natural and drug-related rewards act on common brain reward pathways.

Twin and adoption studies suggest that heritable factors account for 40–70% of an individual's risk of becoming obese, which is comparable with the contribution of genetic factors to addiction risk [9]. Identical twins raised apart are more similar in weight than non-identical twins raised together. Similar results have been described for bulimia nervosa for which 54–83% of the risk is attributed to heritability [5]. There are also higher rates of substance use disorders among relatives of individuals with anorexia nervosa and bulimia nervosa [10], which suggests shared neuropsychological mechanisms.

Proponents claim that a more scientific explanation of obesity, on the basis of evidence from neuroscience, will lead to new treatments for overeating

The underlying genetic causes, however, remain to be determined. Several susceptibility genes, such as *MC4R* and *FTO*, have been shown to predispose individuals to obesity, either through increasing appetite or through encouraging fat mass accumulation [9], but the strength of their association with obesity is weak. Generally, the specific candidate genes for obesity identified so far only account for a small fraction of the total genetic risk. Proponents of genetic explanations of obesity argue that more research will eventually locate candidate genes and variants that would explain the heritability of obesity [9]; however, as many common variants have small effects, these alleles are

Table 1 | Prevalence of food addiction as diagnosed by the Yale Food Addiction Scale

Weight	BMI	Sample size (n)	Diagnoses (%)
Underweight	<18.5	60	10.0
Normal weight	18.5–24.9	572	6.3
Overweight	25–29.9	86	14.0
Obese	≥30	32	37.5

Adapted from reference [13].

still improbable explanations for a risk for obesity among a large population [11].

Given the apparent paucity of specific genetic factors, it is possible that epigenetics holds the key to understanding obesity risk. Environmental factors can modify DNA in ways that prevent certain genes from being expressed—thus causing epigenetic modifications—which can affect neural functioning and thereby cause extreme forms of obesity or increase susceptibility to obesity [9]. Intrauterine environmental influences—such as maternal diet and weight—produce these kinds of epigenetic variation that increase disease risk [9]. In fact, epigenetic changes might well be important in drug addiction [12]. Although research in this area is at an early stage, new therapeutic advances that affect epigenetic changes might have the potential to block or reverse addiction [12].

Taken together, the neurobiological evidence suggests that certain styles of eating, such as binge eating, compulsive eating and chronic overeating, resemble the behaviour of people addicted to drugs. In addition, certain foods, particularly high-sugar foods, might produce addictive patterns of eating in some individuals. Is this evidence strong enough to include overeating and obesity caused by overconsumption in the DSM-V? We urge caution, because there are still several limitations to the research that supports the concept of food addiction. First, although neurobiological studies show differences in brain structure and function between normal-weight individuals and overweight or obese individuals, the correlations do not prove that certain foods cause these neurobiological changes. It is not clear whether the differences in the dopamine reward pathways in compulsive overeaters lead to overeating or are a consequence of overeating. Second, although the results identify average differences between normal-weight individuals and overweight or obese

individuals, there is substantial intragroup variation and overlap between the two groups. A similar criticism has been made of neuroimaging studies of drug-addicted individuals and controls. Third, the most compelling evidence for the concept of food addiction comes from animal models, but the applicability of these findings to obese humans is uncertain. Finally, human studies are limited by the absence of reliable and valid diagnostics tools and criteria to define and identify cases of food addiction: not all people who are obese meet the criteria for food addiction and, vice versa, individuals who reportedly display characteristics of food addiction might be underweight or normal weight (Table 1).

Nevertheless, the proponents of food addiction argue that treatments based on recognition of the neurological factors in overeating might prove more effective in reducing obesity than the more common therapies. In terms of the latter, dietary interventions, exercise prescription and therapeutic support groups are the three most widely used lifestyle treatments. Dietary interventions generally involve a sustained reduction in energy intake to produce weight loss, often coupled with exercise, which increases overall health and reduces cardiometabolic risk factors by improving lipid profiles and reducing blood pressure [14].

Another potential concern is that addiction-based treatments of overeating might distract attention away from the significant role of exercise

Pharmacological treatments for obesity are often used in conjunction with lifestyle modifications and counselling. Until recently, the only prescription approved for long-term weight loss was orlistat [15]. The FDA has recently approved two new weight-loss drugs: Belviq (lorcaserin), an

appetite suppressant (<http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm309993.htm>); and Qsymia, a combination of an appetite suppressant (phentermine) and an anti-seizure medication (topiramate) (<http://www.fda.gov/NewsEvents/Newsroom/PressAnnouncements/ucm312468.htm>). Combination therapies, such as naltrexone—an opioid antagonist—with bupropion—a smoking cessation drug—are being trialled [15].

Surgical treatments for obesity are higher-risk interventions reserved for patients who have failed to respond to lifestyle and pharmacological treatments. The most common interventions are laparoscopic gastric bypass, adjustable gastric band, laparoscopic sleeve gastrectomy and biliopancreatic diversion. Although the overall amount of weight loss varies by procedure, the average long-term excess weight loss is 47–70% [16]. As with all surgical procedures, bariatric surgery can result in postoperative complications that include infection, metabolic disorders and cardiovascular events.

Proponents claim that a more scientific explanation of obesity, on the basis of evidence from neuroscience, will lead to new treatments for overeating. For instance, psychotherapies based on addiction treatment could improve emotion regulation and tolerance, strengthen impulse control and replace food and eating as sources of reward and pleasure [3]. Contingency management—an intervention based on operant conditioning principles that provides rewards or incentives, such as payment or vouchers, for achieving certain outcomes—has already proven effective in reducing drug use in addicted individuals. It also has the potential to reduce overeating and encourage weight loss in overweight people [17].

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An addiction-based approach might also lead to new pharmacological treatments that target the dopamine system and thereby make overeating less rewarding [15]. Appetite suppressants, such as lorcaserin, that act

on satiety signalling are already being trialled in combination with behavioural therapies [15]. However, the limited effectiveness of the pharmacological treatments for addiction derived from neuroscience research should temper enthusiasm for their use in treating overeating.

There is also reasonable concern that neurobiological explanations of addiction might be seen as justifying more invasive or high-risk interventions, as has occurred in the treatment of drug addiction [18]. Deep brain stimulation (DBS)—an invasive neurosurgical procedure to treat pharmacologically resistant Parkinson disease—has already been proposed for the treatment of drug addiction and obesity [19]. Proponents justify its use by appealing to the social burden of addiction and the high rate of relapse after conventional treatments [19]. Whilst initially positive effects of DBS have been reported in the treatment of isolated cases of obese individuals, DBS is an expensive procedure that will probably benefit few patients. Most importantly, there are serious ethical concerns raised by the use of DBS in desperate patients [18].

Another potential concern is that addiction-based treatments of overeating might distract attention away from the significant role of exercise. In addition to the associated health benefits that occur regardless of changes in BMI [14], exercise might yield additional psychological and behavioural benefits.

In most Western countries, obesity is stigmatized. Obese individuals are often seen as lacking self-control, motivation and intelligence [20]. Conditions for which individuals are held to be minimally responsible, such as Alzheimer disease, engender more pity and empathy than conditions for which individuals are held personally responsible, such as obesity and drug addiction. Some argue that neurobiological explanations of obesity might help to reduce such attitudes [20]. However, the limited empirical evidence on the impact of neuroscientific explanations on the stigmatization of obesity, alcohol dependence and other mental illnesses suggests that this optimism might be misplaced. A survey of public attitudes in the USA found that increased public endorsement of neurobiological explanations of alcohol dependence was not accompanied by any reduction in stigma [21].

There is also concern that the possible adverse effects of a diagnosis of ‘disease of food addiction’ might outweigh the benefits for obese individuals; namely, that such a diagnosis will undermine their motivation to reduce their caloric intake or adopt healthy eating and exercise patterns. It is not clear what impact neurobiological explanations might have on most of the population who struggle to avoid highly palatable foods but are not ‘addicted’. These potential repercussions need to be empirically investigated before we assume that neuroscientific explanations of obesity will produce only desirable changes in individual and public perception.

Another danger is that focusing on ‘food addiction’ might distract attention from obesogenic environments; environments that act to promote overconsumption by making high-caloric foods readily available at a low price. This would undermine public health policies that aim to limit the availability of foods with a higher potential for overconsumption and weight gain. Ultimately, the impact that the concept of ‘food addiction’ as a disease might have on obesity policy will depend crucially on how it is portrayed. It could, for example, be seen as a rare disorder that affects only a small proportion of the population, as is the case for alcohol and drug addiction. This perspective would support a high-risk strategy to treat obesity; policies that attempt to identify those with the disorder, or who have a high risk of developing it, with the aim of targeting them for earlier preventive measures. This approach could come at the expense of population-wide approaches to reduce the availability of obesogenic foods. It is clear that most citizens in the populations of developed countries suffer to some extent from being overweight or obese, so a high-risk approach, even one solely focused on obesity caused by overconsumption, is unlikely to have much impact.

If policy responses to ‘food addiction’ were to follow the approach used in alcohol policy, the availability of obesogenic foods would be less probable to change. ‘Food addiction’ would be presented by the food industry as a rare disorder that did not warrant policy changes to influence the general public’s eating. One would expect to see education programmes targeted at ‘food addicts’, despite evidence that education is not effective in

changing behaviour without supportive changes in the environment [22]. Commercial industries often have an interest in promoting strategies focused on high-risk groups, which can distract from or undermine broad-reaching approaches that reduce total consumption in the population and, subsequently, their profits [23]. The promotion of alcoholism as a rare form of addiction has, for example, been used by the alcohol beverage industry to counter demands for increased taxes and restrictions on the marketing and promotion of alcohol. In addition, the food industry might support research into identifying the genetic and neurobiological bases of individual vulnerability to food addiction. Although this might seem to be promoting scientific research, it might actually serve the food industry's interests by shifting attention away from obesogenic environments towards obese individuals, mimicking tactics used in the past by the alcohol, tobacco and gambling industries [23].

If specific foods, such as sugar, came to be seen as potentially addictive, population-wide approaches to reducing obesity might be better supported

The case for the substantial regulation of foods is arguably stronger in the case of children, given their susceptibility to advertising and lack of responsibility. This might allow policy-makers to argue for a broader regulatory approach to reduce overconsumption by changing obesogenic environments. If specific foods, such as sugar, came to be seen as potentially addictive, population-wide approaches to reducing obesity might be better supported. This perspective would attempt to increase the intake of healthy foods by increasing access, labelling and subsidies, whilst decreasing availability and using taxation to raise the price of energy-dense, nutrient-sparse foods.

Such policy changes are already taking place around the world. In October 2011, for example, Denmark enacted a tax on saturated fats, following the example of Hungary's tax on foods high in sugar, fat, salt and caffeine. Advocates seeking to highlight the addictive qualities of sugar have similarly proposed a tax on soft drinks and on all food containing

excessive amounts of sugar [2]. New York City's mayor, Michael Bloomberg, has proposed a ban on large sodas and other sugary drinks in restaurants, movie theatres and street carts [24]. In combination with taxation, advertising bans could also reduce the overconsumption of foods high in sugar, fat and salt. As children are particularly vulnerable to advertising, this could be an efficient way to reduce the alarming rates of childhood obesity. Incorporating public policy changes to protect children might well be the first step in regulating access to 'addictive' foods.

Neurobiological research into overeating could also distract attention from more ecological approaches that aim to provide a comprehensive analysis of the factors that lead to obesity and that guide interventions at the population level [25]. Such approaches could include increasing incentives for physical activity through tax breaks or insurance coverage, implementing community-based support groups, or designing more active urban environments with bicycle paths, parks and walkable neighbourhoods. Findings on the impact of sedentary behaviour and increased risk for morbidity [26] might also prompt public policies to increase physical activity in the workplace. As a reduction in sedentary behaviour generally improves health, even non-obese individuals would benefit. Population-level approaches are generally more cost-effective than clinical interventions aimed only at obese individuals [2]; the use of multi-level interventions is generally the most effective way to alter behaviour and support individual behavioural changes [25].

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It might sound appealing to classify certain foods and eating styles as addictive—on the basis of evidence from animal and human research—and to develop new treatment options and modify existing treatments for obese individuals. However, the concept of 'food addiction' is still underdeveloped and requires rigorous

research and analysis to better define and test its validity in humans. In considering policies that treat obesity as a form of 'food addiction'—and the implications that stem from such policies—researchers should heed the lessons from successful public health campaigns to reduce tobacco smoking. They should be wary of the potential misuse of the concept of 'food addiction' as a disease by the food industry and should consider carefully both the new opportunities for improving public health and the avenues of treatment and therapy they preclude. It is important that the results of neurobiological research, however compelling, are not used in isolation to treat severely obese people. Such biological insights should be incorporated into population-based approaches that make use of multiple levels of influence to provide treatment and intervention regimes.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest.

REFERENCES

1. Finucane MM *et al* (2011) National, regional, and global trends in body-mass index since 1980: systematic analysis of health examination surveys and epidemiological studies with 960 country-years and 9.1 million participants. *Lancet* **377**: 557–567
2. Gortmaker SL *et al* (2011) Obesity 4: changing the future of obesity: science, policy, and action. *Lancet* **378**: 838–847
3. Davis C, Carter JC (2009) Compulsive overeating as an addiction disorder. A review of theory and evidence. *Appetite* **53**: 1–8
4. Ifland JR *et al* (2009) Refined food addiction: a classic substance use disorder. *Med Hypotheses* **72**: 518–526
5. Volkow ND, Wang G-J, Baler RD (2011) Reward, dopamine and the control of food intake: implications for obesity. *Trends Cogn Sci* **15**: 37–46
6. Pelchat ML, Johnson A, Chan R, Valdez J, Ragland JD (2004) Images of desire: food-craving activation during fMRI. *Neuroimage* **23**: 1486–1493
7. Avena NM, Rada P, Hoebel BG (2008) Evidence for sugar addiction: behavioral and neurochemical effects of intermittent, excessive sugar intake. *Neurosci Biobehav Rev* **32**: 20–39
8. Gearhardt AN *et al* (2011) Neural correlates of food addiction. *Arch Gen Psychiatry* **68**: 808–816
9. Herrera BM, Keildson S, Lindgren CM (2011) Genetics and epigenetics of obesity. *Maturitas* **69**: 41–49
10. Krug I *et al* (2009) Lifetime substance abuse, family history of alcohol abuse/dependence and novelty seeking in eating disorders:

- comparison study of eating disorder subgroups. *Psychiatry Clin Neurosci* **63**: 82–87
11. Manolio TA *et al* (2009) Finding the missing heritability of complex diseases. *Nature* **461**: 747–753
 12. Robinson AJ, Nestler EJ (2011) Transcriptional and epigenetic mechanisms of addiction. *Nat Rev Neurosci* **12**: 623–637
 13. Meule A (2011) How prevalent is ‘food addiction’? *Front Psychiatry* **2**: 61
 14. Green DJ, Maiorana AJ (2012) Why exercise is an important component of risk reduction in obesity management. *Med J Aust* **3**: 165–166
 15. Vetter ML, Faulconbridge LF, Webb VL, Wadden TA (2010) Behavioral and pharmacologic therapies for obesity. *Nat Rev Endocrinol* **6**: 578–588
 16. Eldar S, Heneghan HM, Brethauer SA, Schauer PR (2011) Bariatric surgery for treatment of obesity. *Int J Obes* **35**: S16–S21
 17. Marteau TM, Ashcroft RE, Oliver A (2009) Using financial incentives to achieve healthy behaviour. *Br Med J* **338**: 983–985
 18. Carter A, Bell E, Racine E, Hall W (2011) Ethical issues raised by proposals to treat addiction using deep brain stimulation. *Neuroethics* **4**: 129–142
 19. Halpern C *et al* (2011) Expanding applications of deep brain stimulation: a potential therapeutic role in obesity and addiction management. *Acta Neurochir (Wien)* **153**: 2293–2306
 20. Sikorski C *et al* (2011) The stigma of obesity in the general public and its implications for public health—a systematic review. *BMC Public Health* **11**: 661
 21. Pescosolido BA *et al* (2010) “A disease like any other”? A decade of change in public reactions to schizophrenia, depression, and alcohol dependence. *Am J Psychiatry* **167**: 1321–1330
 22. Pomeranz JL (2008) A historical analysis of public health, the law, and stigmatized social groups: the need for both obesity and weight bias legislation. *Obesity* **16**: S93–S103
 23. Miller P, Carter A, De Groot F (2012) Investment and vested interests in neuroscience research of addiction: why research ethics requires more than informed consent. In *Addiction Neuroethics: The Bioethics of Addiction Neuroscience Research and Treatment* (eds Carter A, Hall W, Illes J), pp 277–300. New York, USA: Elsevier
 24. Grynbaum MM (2012) Mayor planning a ban on big sizes of sugary drinks. *The New York Times* 31 May 2012
 25. Sallis JF, Owen N, Fisher EB (2008) Ecological models of health behavior. In *Health Behavior and Health Education: Theory, Research, and Practice*, 4th Edn (eds Glanz K, Rimer BK, Viswanath K) pp 465–485. San Francisco, California, USA: Jossey-Bass
 26. Owen N *et al* (2011) Adults’ sedentary behavior: determinants and interventions. *Am J Prev Med* **41**: 189–196

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