



Sugar addiction: pushing the drug-sugar analogy to the limit

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Purpose of review

To review research that tests the validity of the analogy between addictive drugs, like cocaine, and hyperpalatable foods, notably those high in added sugar (i.e., sucrose).

Recent findings

Available evidence in humans shows that sugar and sweetness can induce reward and craving that are comparable in magnitude to those induced by addictive drugs. Although this evidence is limited by the inherent difficulty of comparing different types of rewards and psychological experiences in humans, it is nevertheless supported by recent experimental research on sugar and sweet reward in laboratory rats. Overall, this research has revealed that sugar and sweet reward can not only substitute to addictive drugs, like cocaine, but can even be more rewarding and attractive. At the neurobiological level, the neural substrates of sugar and sweet reward appear to be more robust than those of cocaine (i.e., more resistant to functional failures), possibly reflecting past selective evolutionary pressures for seeking and taking foods high in sugar and calories.

Summary

The biological robustness in the neural substrates of sugar and sweet reward may be sufficient to explain why many people can have difficulty to control the consumption of foods high in sugar when continuously exposed to them.

Keywords

addiction, animal models, cocaine, craving, dopamine, sugar

INTRODUCTION

The current global increase in obesity prevalence and the difficulty of containing it despite the negative consequences have recently led several researchers, mostly neuroscientists, to compare obesity to drug addiction [1–9,10[■],11] and palatable foods, particularly those high in added sugar (i.e., sucrose), to addictive drugs like cocaine [4,12–14]. Volkow – the current head of the American National Institute on Drug Abuse – and O'Brien [9] were among the first to suggest that the concept of addiction may shed some new light on obesity – an idea that has proven quite influential, as shown by the exponential rise of the use of the expression ‘food addiction’ (and related terms) in the biomedical and scientific literature ever since [15[■]]. In parallel, others have strongly argued that hyperpalatable foods rich in added sugar and/or fat could be genuinely addictive, at least in a significant proportion of exposed people [12,13]. One currently estimates that about 10–20% of people would present addiction-like symptoms

toward hyperpalatable foods [16,17] – a proportion that is not different from the proportion of cocaine or heroin users who go on to develop addiction [18]. The widespread introduction of hyperpalatable foods during the 20th century could be likened to the introduction of distilled drinks (i.e., gins, whiskeys) in the 17th century or of injectable synthetic drugs at the end of the 19th century, each spurred its own addiction epidemics [19]. Finally, people are as ill prepared biologically to foods high in added sugar and/or fat, as they are to drugs in pure or highly concentrated form [4]. In this regard, the ubiquity, ready availability, and affordability of

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KEY POINTS

- Sugar and sweetness can induce reward and craving in humans that are at least comparable in magnitude to those induced by addictive drugs.
- Sugar and sweet reward cannot only substitute to cocaine but can even be more rewarding and attractive than cocaine in animals.
- The neural substrates of sugar and sweet reward are more robust than those of cocaine (i.e., more resistant to functional failures) in animals.
- Additional research using more valid psychophysical and behavioral methods is required in humans to more directly compare addictive drugs and sugar.

those foods make them a serious modern hazard to public health [1,10[■]].

However, although the concept of food and sugar addiction is gaining momentum, it is also currently a subject of intense debate and no solid evidence-based consensus has emerged yet [20–22,23[■],24[■]]. What is really at stake is, first, the drug-like status of some food ingredients, notably sugar, and, second, the relevance of the concept of addiction to understanding obesity. The latter issue has been recently discussed at length elsewhere, particularly the overlap in the neurobiological substrates between drug addiction and obesity [10[■],12,13,23[■]]. Here we will be mainly concerned with the strengths and limitations of the analogy between drugs of abuse and hyperpalatable foods, with a particular, although not exclusive, focus on foods or drinks containing high levels of added sugar (i.e., sucrose). The world history of sugar is not different from that of many psychoactive drugs, including cocaine. It initially began as a medicine for the rich and the powerful and ended up as a product of mass consumption [19]. Today, the ‘sweetening of the world’s diet’ is almost total [25] and there is growing evidence linking increased sugar availability and consumption to overweight and obesity [26,27].

DRUG VERSUS FOOD PSYCHOACTIVE INTOXICATION

At first glance, the analogy between foods high in added sugar and drugs of abuse, such as cocaine, may seem overstretched, not to say absurd. Unlike sugar, drugs of abuse, except ethanol, are non-nutritive molecules that, once self-administered (e.g., through inhalation or via the intravenous route), quickly cross the blood-brain barrier to

physically interact and interfere with specific endogenous molecular substrates and processes, generally at the surface of brain cells [28]. For instance, cocaine binds to the dopamine transporter, among other molecular targets, thereby blocking the neuronal reuptake of dopamine and causing an abnormally high surge of dopamine in innervated brain regions [29]. Drug-induced changes in neuronal and synaptic activity in different brain regions and circuits lead in turn to alterations in behavioral dispositions (e.g., aggressiveness, risk-taking), mood (e.g., euphoria) and other mental functions (e.g., judgement, decision-making) [30]. At high doses, these psychoactive effects may considerably impair normal functions (e.g., distorted perception; altered judgement; diminished self-control) and can be hazardous to both the self and others. In contrast, other psychoactive effects can be advantageous or functional under some circumstances (e.g., disinhibition of sex) and, of course, even highly stimulating and rewarding (e.g., euphoria) [31].

Foods high in sugar can also change brain activity but via more natural routes than drugs of abuse. They can change brain activity, first, via the stimulation of specialized sweet taste cells in the mouth (and in the gut) [32,33[■]] and, second, via postabsorptive brain mechanisms involving glucose signaling [34] – the latter being the most drug-like. Nevertheless, people now often report seeking and consuming sweet foods for their drug-like psychoactive and mood-altering effects [35]. They eat sweet foods to experience highly rewarding sensations, to cope with stress (e.g., stress or comfort eating), pain or fatigue, to enhance cognition and/or to ameliorate bad mood (e.g., relief of negative affect). This anecdotal evidence is also confirmed by research in different populations (e.g., adolescents at risk to develop depression; obese women) showing that sweet foods can indeed elicit different desirable drug-like psychoactive effects, including affective comfort and alleviation of depressed mood [35,36–38]. In most cases, however, the magnitude of the experienced psychoactive effects of sweet foods is mild and does not seem to match those of drugs. In other words, sweet foods are clearly not as behaviorally and/or psychologically toxic as drugs of abuse can be, especially at high doses. For instance, unlike drugs, consumption of hyperpalatable foods, even extremely high in sugar, does not produce any abnormal mental state or change in behavioral disposition. This explains why no policeman will arrest a driver because he/she had eaten several doughnuts before driving his/her car or why no judge will consider drinking half a gallon of sugar-sweetened soda before committing a crime

as a mitigating circumstance. If any, foods rich in sugar seem to produce more advantageous effects on decision-making and self-control than disadvantageous ones. For instance, sugar has been shown to boost self-control under some circumstances [39]. Sugar and sweetness can also promote helping attitude and thereby encourage cooperation among people [40].

DRUG VERSUS FOOD REWARD

The analogy between foods high in sugar and drugs of abuse, like cocaine, may also seem exaggerated because drug reward is generally thought to be incommensurably more intense than food reward. This difference is generally attributed to drugs' ability to activate brain reward circuits, notably midbrain dopamine neurons, more potently than any other nondrug reward [10⁴¹]. Drug addicts often report that the first drug experiences can be even better than sex orgasm. In Western societies, sex orgasm is generally placed at the top of the human hedonic scale, well above foods [42]. This is memorably illustrated in the movie 'Meet Joe Black' where Joe Black – who has recently developed a taste and craving for peanut butter – confesses to Suzan Parrish that making love with her was better than peanut butter. In fact, however, there is little direct evidence showing that drug reward is indeed more intense than food or sex reward, even in drug addicts. In one unique comparative study that has begun to address this question in cocaine addicts, addicts reported liking food as much as they liked cocaine or sex [43]. Interestingly, in this study, healthy individuals also reported liking food almost equally as sex. More research is clearly needed here to address this important issue about the relative intensity of food versus drug reward (see also below).

DRUG VERSUS FOOD CRAVING

The analogy between hyperpalatable foods high in sugar and drugs of abuse, like cocaine, may also seem absurd because drug craving is by definition a pathological desire for a specific substance that is unwanted but difficult to resist [44]. Can food craving be as intense as drug craving? Once again there is little empirical comparative research on this question. Most people do report experiencing food cravings, sometimes very intense ones [45]. Chocolate and sweet foods are the most common craved foods [35,46,47]. In a recent large-scale experience sampling study on everyday desires and urges, food desires were by far the most frequent, with a large proportion of these desires being felt as conflictual and eliciting resistance

[48⁴⁹]. The key issue here, however, is to determine whether food cravings can be comparable in magnitude to drug cravings. In one rare study addressing this problem, cocaine addicts reported wanting food as intensely as they wanted cocaine or sex [43]. In another recent study, craving for a cigarette or for a palatable food was induced in chronic smokers by exposure to cigarette or food cues, respectively. Overall, the intensity and resistibility (i.e., ability to resist it) of food craving were comparable to those of cigarette craving [49]. At the neurobiological level, a recent meta-analysis of neuroimaging research revealed a large overlap in the brain networks underlying cue-induced food versus cigarette craving [50⁵¹]. A similar overlap in neural substrates is also observed in animal models of food and drug craving [51].

WHEN SUGAR AND SWEET REWARD SURPASSES COCAINE REWARD

Overall, available research shows that, although hyperpalatable foods high in sugar are clearly not as behaviorally and psychologically toxic as cocaine and other drugs of abuse, sweet reward and craving are apparently comparable in intensity or magnitude to drug reward and craving, thereby providing some support to the analogy between hyperpalatable foods and drugs. However, solid evidence for the food-drug analogy is still scant and most of it is based on poorly validated intersubjective comparisons and evaluations by people with drug addiction who are clearly not representative of the general population currently exposed to foods high in sugar. There are valid psychophysical methods that could allow one to compare drug reward (or craving) with food reward (or craving) but these methods have yet to be tested [52]. Similarly, there are objective behavioral methods for measuring reinforcing strengths between different kinds of rewards but they have not been systematically used to directly compare hyperpalatable foods with drugs of abuse [53]. Finally, at the neurobiological level, drug and food cues clearly recruit the same overall brain networks [50⁵¹] but more direct quantitative comparisons between brain activation patterns during drug versus food reward (or craving) are still lacking.

Another possible approach to explore the drug-food analogy could be to compare drug versus food reward in nonhuman animals, such as laboratory rats or mice. Rats have been shown to self-administer most addictive drugs, including cocaine [54], and to develop most of the behavioral signs of addiction after prolonged drug self-administration [55]. Like humans, rats have also an inborn sweet

tooth that has been systematically exploited over the past 60 years for research on the neural basis of reward and motivation. In fact, many important discoveries about the neurobiology of reward and motivation have been and are still made using sugar and sweetness as a reward or incentive [2]. Sugar and sweetness are also often used in drug addiction research to train animals before drug self-administration training. More relevant to the present review, under certain circumstances, sugar and sweet reward can substitute to cocaine, thereby decreasing cocaine self-administration [11]. Furthermore, when directly compared together, sugar and sweet reward can even be more rewarding and attractive than cocaine [14,56–59]. For instance, in a recent series of choice experiments, we found that when rats are offered an exclusive choice between sucrose (or saccharin) and cocaine self-administration, they develop a strong and persistent preference for sucrose (or saccharin) [14,56–58]. Similar findings have also been obtained in rats offered an exclusive choice between nicotine and sucrose [60]. Sugar withdrawal can also induce behavioral and neurochemical signs similar to those of heroin withdrawal [12]. Table 1 summarizes behavioral evidence from different studies that have compared sucrose (or sugar-sweetened foods) with cocaine under similar testing conditions. Clearly, rats can be at least as rewarded or motivated for sucrose as for cocaine and sometimes even more.

At first glance, this conclusion seems to conflict with evidence showing that sugar and sweet reward are much less potent than cocaine to boost brain dopamine signaling [14]. However, this apparent discrepancy may also suggest that dopamine is probably not enough to drive preference and that sugar, unlike cocaine reward, involves more than brain dopamine [14,24^{***}]. This interpretation is supported by recent research using optogenetic methods in mice. Mice were allowed to choose between two sippers: licking one sipper delivered water and optogenetic stimulation of dopaminergic neurons whereas licking the other sipper delivered water sweetened with sucrose. When concentrations were sufficiently high, mice preferred sucrose over optogenetic stimulation of dopamine neurons [61] which can be rewarding alone [62]. In addition, there is ample, although disparate and overlooked, evidence that the neurobiological substrate of sucrose reward is more robust than that of cocaine reward. Table 2 summarizes the outcomes of all published studies ($n=75$) that have compared the effects of different genetic (e.g., gene knockout or knockdown), pharmacological (e.g., selective receptor antagonism) or neurobiological (e.g., selective brain lesion) interventions on cocaine versus

Table 1. Sucrose reward and motivation versus cocaine reward and motivation

Behavioral criterion	Sucrose/Sweet	Cocaine
Preference	++	+
Demand inelasticity	++	+
Punishment resistance	+	+
Breaking point	++	++

Preference refers to sucrose (or sweet) choice over cocaine in choice experiments [14,56–58]. Demand inelasticity refers to the degree of resistance of sucrose or cocaine consumption to increasing behavioral costs (e.g., increasing number of required responses) [63–65]. Similarly, resistance to punishment refers to the degree of resistance of sucrose or cocaine consumption to aversive electrical footshock or associated conditioned stimuli [66–70]. Finally, breaking point refers to the maximum acceptable work before giving up working for sucrose (or sweet reward) or cocaine [57].

sucrose under similar behavioral conditions (e.g., fixed-ratio or progressive-ratio schedule of reinforcement). Overall, most interventions affected cocaine-rewarded or cocaine-motivated behaviors (i.e., 88 out of a total of 91 interventions) but only a few of them (i.e., 17) impacted on sucrose-rewarded or

Table 2. Neurobiological interventions on sucrose versus cocaine reward or motivation

Interventions	N	Sucrose	Cocaine
Intracellular processes	8	25	100
Selective brain lesions	8	12.5	87.5
Neurotransmitters			
Dopamine	23	21.7	100
Glutamate	21	14.3	90
Serotonin	8	25	100
Acetylcholine	6	33.3	100
Neuropeptides	6	16.6	100
Adenosine	3	0	100
Norepinephrine	2	0	100
Cannabinoids	2	0	100
Steroids	4	25	100
Total	91	18.7	96.7

We conducted a search for articles containing the words 'sucrose' and 'cocaine' in the PubMed database (last accession date: March 11, 2013). A total of 256 articles were retrieved. Among these 256 articles, 75 were selected and retained as relevant to the present study based on information contained in the abstract. The full-text of the selected articles was then read and analyzed in-depth for interventions that were tested on both sucrose and cocaine reward (or motivation) and under similar experimental conditions (e.g., intervention X tested on both operant responding for sucrose and cocaine under a comparable schedule of reinforcement). The column N shows the number of interventions per specific biological target. The number in the other columns indicates the proportion of interventions in % that affected sucrose- or cocaine-related behaviors. Note that some articles can contain more than one neurobiological intervention. The list of selected articles is available upon request to the authors.

sucrose-motivated behaviors. When the effect of each specific intervention is considered individually, it is generally interpreted as evidence that a given gene, brain region or neurotransmitter system is specifically involved in cocaine reward or motivation but not in sucrose reward or motivation. However, the big picture emerging from Table 2 clearly shows that many different specific interventions can profoundly affect cocaine reward or motivation while sparing sucrose reward or motivation. This discovery strongly suggests that the neurobiological substrate of sugar reward and motivation is more robust than that of cocaine (i.e., more resistance to individual biological failures or dysfunctions). Such robustness probably results from the evolution of multiple neural mechanisms for seeking and taking sugar and other foods high in calories [24²²] and further underscores the exceptional motivational power of sugar sweetness [14].

CONCLUSION

The extreme intensity of reward and craving produced by addictive drugs, like cocaine, largely explains, together with their toxic effects, why these drugs tend to be universally prohibited. The danger to be avoided by prohibition is that drug reward will turn people away from more socially valued, healthy, and productive life activities and occupations. Most of the current medical diagnostic criteria of drug addictive disorders reflect this harmful shift in behavioral preference and priority [44]. As reviewed here, there is now strong evidence supporting the notion that hyperpalatable foods, notably those high in added sugar, can induce reward and craving that are at least comparable to addictive drugs. Although more research in humans is clearly needed to confirm this conclusion, there is now solid evidence in nonhuman animals showing that sugar and sweet reward can even be more rewarding and attractive than addictive drugs, probably owing to an underlying robust neural substrate. Such biological robustness may be sufficient to explain why people can have difficulty to control the consumption of foods high in sugar when continuously exposed to them.

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Conflicts of interest

There are no conflicts of interest.

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Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 492).

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