The Case to Ban Sugary Food and Drink from Schools: these products are addictive, and kids will learn best without them.

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ABSTRACT

In New Zealand, schools have been a battleground as a setting to address the obesity epidemic, with successive governments enforcing, and then repealing laws to ban junk food from canteen menus. Just what is considered 'junk food' remains controversial, but recently sugar sweetened beverages have become a target of public health groups. The adverse health consequences of their intake have been the principal arguments to reduce their availability. Here, we argue that the advantages of reducing intake of sugar in schools are very likely to lead to educational and other social benefits. Indices of sugar intake have been associated with aggressive behaviour, attention deficit, dysphoria and suicidal thoughts in cross-sectional studies. Longitudinal studies have also linked soft drink intake with impaired cognitive development. We believe that banning sugary drinks from schools will assist teachers and students to better achieve their learning goals, with a side effect of improving their health status.

COMPETING INTERESTS: Nil

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Introduction

Traditionally, sugary foods and drinks have been considered a source of 'empty calories',1 contributing to weight gain2 and the sequelae of obesity, such as diabetes,³⁻⁹ hypertension,¹⁰⁻¹² gout¹³ and other diseases. Added dietary sugar (here sucrose or high fructose corn syrups) also causes tooth decay,¹⁴ and this constitutes another reason to control intake, particularly in children.

Schools in New Zealand have been a battle ground for nutrition campaigns, with restrictions on what is available in these settings enforced, then repealed by successive governments.¹⁵ Social marketing campaigns, such as fuelled4life encourage these institutions to offer healthy options. The criteria for this program which are used to define a healthy from a less healthy food are vague, and no foods or drinks are deemed unhealthy. The only two categories available for foods or drinks to be classified include: "everyday" and "sometimes".

Here, we define sugary food and drink as that which is >5% added sugar by weight. This includes cakes, biscuits, sugar sweetened soft drinks, fruit juice (although it is technically not 'added sugar'), cordial and many other manufactured food items. Whole fruit is excluded, since the sugar content is generally low concentration, with high fibre content. In contrast, fruit juice is included since it generally has concentrated sugar content, with fibre content often excluded during the manufacturing.

In this paper, we propose that sugar intake not only leads

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to health problems but other behavioural consequences that are likely to disrupt child learning. It is hoped that drawing attention to this issue will increase the impetus to enforce policies and legislation that limit the supply and sale of sugary food and drinks in educational institutions. Here, we define educational institutions as early childhood centres, primary and secondary schools and tertiary institutions.

What is addiction?

We argue that high sugar intake impairs behaviour and learning in childhood principally due to its addictive properties, and that these effects have largely been overlooked when discussing healthy food policies. addictive behaviour. This 'negative re-inforcement', driven by the rapid relief of withdrawal symptoms becomes a powerful means by which the addict learns to automatically reproduce their substance taking behaviour.¹⁷

Official definitions of addiction, labelled 'substance use disorder' in the psychiatric 'bible', the DSM-V,¹⁸ emphasize the increased motivation associated with taking the substance which lead to social, work related and legal problems. Other features include withdrawal, craving, psychological and physical adverse effects, failure to control use and taking progressively greater amounts.

Addiction also influences thought.¹⁹ Denial and minimisation of the effects of the object of addiction, the drug or the

	Alcohol	Opioids	Sedatives	Cocaine	Nicotine	Stimulants
Sweating	Yes	Yes				
Nausea	Yes	Yes	Yes			
Change in heart rate	Increase		Increase		Decrease	
Sleep Disturbance	Yes	Yes	Yes	Yes	Yes	Yes
Anxiety			Yes		Yes	
Dysphoric mood		Yes			Yes	
Fever		Yes				
Increased appetite				Yes	Yes	
Irritability					Yes	Yes
Difficulty concentrating					Yes	
Time course						
Onset	6-12 hours	4-6 hours		1 week	2-12 hours	
Peak	3-7 days	2-3 days			2-3 days	
Duration	1-2 weeks	2 weeks		Up to 10 weeks	3-4 weeks	

TABLE 1. SIGNS AND SYMPTOMS OF SUBSTANCE WITHDRAWAL

Adapted from: American Psychiatric Association 2000; Hughes et al. 1994.

To consider the question of whether sugar is addictive, it is first necessary to define addiction. Some debate surrounds the use of the term, however, it is most often recognised as a failure to give-up or stop the use of a substance of behaviour.¹⁶ It is a disorder of motivation, or failure of the will. According to commonly accepted definitions, the object of addiction leads to some harmful consequences, which may or may not be recognised by the subject.¹⁷ The presence of a withdrawal syndrome, when the substance or behaviour is stopped is also considered a pathognomonic sign of the disorder.¹⁶ Such withdrawal symptoms are thought to be the reason that addicts find it hard to stop using their addictive substance. The unpleasant symptoms are subconsciously known by the addict to be relieved by taking the substance or performing the behaviour, is a feature of many addictions. Psychological tests show that addiction is associated with automatic behaviour and subversion of conscious thought processes.¹⁹ When addicts encounter sensory stimuli associated with use of the drug, known as cues, impulsive behaviour often follows. Impaired concentration, a symptom of withdrawal, is another feature of the syndrome. This may be mediated by intrusive, unwanted and involuntary urges and cravings to use the substance. Exposure to cues, such as others undertaking the behaviour or images of the addictive substance, can increase the intensity of craving and urges.¹⁹

A range of substances, if taken regularly, are known to promote addiction syndromes with stereotyped withdrawal syndromes. The withdrawal symptoms are provoked by abstinence from the substance and commonly the unpleasant symptoms last from three weeks to several months (table 1). The symptoms are usually mild, but enough to be unpleasant, and often include craving the substance, difficulty concentrating, irritability, and restlessness.

Beyond the clinical manifestations of addiction is the physiology that underlies addiction. The identification of the part of the brain activated by drugs of abuse has contributed to our understanding of what makes some substances addictive over others. The origin of addiction has been linked to a part of the human brain responsible for subconscious control of behaviour and motivation, associated with survival functions, such as eating, drinking and sexual behaviour. One can readily appreciate that drug addiction may be viewed as a 'hunger' for drugs, such that some substances become as important, or even more important than eating food and consuming water and other drinks. This anatomic site, the dopaminergic mesocorticolimbic projection or reward centre, present in the midbrain, is most often implicated in the biology of addiction.¹⁷ In human and animal studies, administration of substances of abuse increases concentrations of the chemical messenger or neurotransmitter, dopamine, in this centre, considered the main component of the brain reward system. For example, an intravenous dose of cocaine results in increased release of dopamine by blocking re-uptake by nerve terminals in a part of the brain called the nucleus accumbens. Other substances, such as opioids, nicotine and alcohol act in a different area of the reward pathway, stimulating nerve cells in other regions, which ultimately influence the nucleus accumbens, increasing dopamine release. This common anatomic site, along with the linked neurotransmitter, dopamine, has, therefore, linked the biology of drug addiction with its clinical features.

Addiction and mental health

Although dopamine has been linked to addiction, the neurotransmitter also plays a role in people with mental health disorders. Dopamine has been labelled the "wind of the psychotic fire", when describing its role in the symptoms of disorders such as schizophrenia.²⁰ Evidence for dopamine's importance emerges from clinical practice - for example, treatment of patients with Parkinson's disease using therapeutic doses of levo-dopa, a dopamine like substance, can rarely cause a drug-induced psychosis.²¹ Conversely, drugs used to treat psychoses such as schizophrenia interfere with dopamine pathways, and may result in unwanted Parkinsonism, manifested by expressionless, blank expressions and a characteristic pill-rolling tremor.

If addiction and psychosis share the same biological pathway and a common neurotransmitter (dopamine), then we might expect that mental disorders and addiction commonly coexist in individuals. For workers in the mental health field, such disorders are all too frequently associated, with the term "dual-diagnosis" used to summarise the occurrence of the two disorders in the same patient. In one summary, the prevalence of smoking was between 80 and 90% in people treated in hospital with schizophrenia.²² Numerous epidemiological studies describe the co-occurrence of schizophrenia and other forms of addiction, such as to alcohol, metamphetamine and opiates.²² Further, the presence of illicit drug use in people with schizophrenia predicts relapse, treatment resistance and need for further hospital treatment.²²

Sugar and food addiction

Although addiction to various drugs and behaviours has become widely accepted, food addiction is not similarly widely recognised. Descriptions of addiction, however, frequently use terms usually reserved for food, such as drug addiction is like a "hunger for drugs". Also, support groups such as Overeater's anonymous use an addiction model, similar to that used for other addictions to help members control their appetites and eating. Terms such as "craving" and "hit" are often used in advertising for sugary products, however, the idea of food as addictive is not widely recognised.

Does sugar intake show evidence of addictive patterns of behaviour? Although by no means widely accepted in nutrition circles,²³ both human and animal laboratory studies show evidence that addiction to sugar occurs.^{24,25} Of all the food groups, carbohydrate is commonly ascribed addictive properties, and within this food group, sugar (sucrose).^{25,26} At a clinical level, in humans, carbohydrate craving has often been reported,²⁶ although a full withdrawal syndrome has not yet been described. Anatomical changes found following positron emission tomography of people who suffer from drug addiction show evidence of adaptation, with increased concentration of dopamine receptors compared to controls. Such midbrain changes also occur in obese individuals.²⁷

Some clues of addiction may be found in popular books. For example, in the book which popularised the Atkins diet,²⁸ the author described obese clients he had helped lose weight who reported symptoms of a possible food withdrawal syndrome, similar to a tobacco withdrawal syndrome. One such patient recounted unsuccessful trials of varied weight loss techniques such as laxatives and drugs that provoked vomiting. He even underwent surgery intended to effect weight loss. Nothing worked. He described:

"...often I would shake until I could put some sugar in my mouth."

Cues were also reported:

"I had an hour's drive from my office to my home, and I knew every restaurant, every candy machine and every soft drink dispenser along the whole route."

In more commonly accepted addictions, such as smoking cigarettes, those elements of the individual's environment that precede drug taking or reward become focal points of attention (cues) for substance abusers. In smokers, an example of cues includes seeing others light up, images of cigarettes or a pack of their favourite brand present in advertising or simply sniffing tobacco vapours in the air. Atkins suggested that a similar syndrome occurred in his patient's relationship with food, although he never mentioned the term addiction. During his evening commute, the patient's attention was diverted, seemingly, against his will, in the direction of any likely source of food, particularly sugar. Case studies of individuals reporting withdrawal syndromes after abstaining from refined starch and sugar have also been published.²⁹

Some clues to the linkage of food and addiction are

observed in clinical settings. Logically, we might expect that if obesity and weight gain are also related to the neurotransmitter, dopamine, then when the effects of dopamine are blocked in some way, for example by drugs, then people that take these drugs would put on weight. Drugs used to treat psychosis, all, to a greater or lesser extent, block the action of dopamine, and all are known to cause weight gain, with some drugs having more potent side effects than others.³⁰⁻³³ Even in people with psychosis, many are overweight before they start treatment, compared to the frequency in the general population, so that overeating may play a causal role in the aetiology of psychotic disorders.³⁴

Research undertaken on rodents also supports the idea of sugar addiction. Rodents have been observed to exhibit features of addiction, such as tolerance and withdrawal after being fed with high sugar diets.²⁴ Similar withdrawal syndromes were not encountered after high fat feeding.

The influence of sugar on behaviour and learning

If we accept the idea that sugar has addictive properties, the presence of withdrawal symptoms, such as craving, irritability and difficulty concentrating, are likely to impair learning in educational settings. Some evidence from observational studies supports this view.

Diet and behavioural outcomes show strong epidemiological associations in cross-sectional studies. One Australian cross-sectional study (n=1,779) with participants assessed at 14 years of age, divided their subjects into a 'Western style' diet, high in cakes, biscuits, confectionary and soft drink intake, and a 'healthy pattern' using a factor analysis. Participants were rated as high or low for these categories, depending on their responses in a food frequency questionnaire. Individuals who scored high for Western diet had a two-fold increased risk (adjusted odds ratio = 2.21, 95% confidence interval = 1.18, 4.13) of a diagnosis of attention deficit hyperactivity disorder, compared to those who scored low for a Western style diet. Gender, physical activity, maternal stress and family income were other significant risk factors identified.³⁵

Consistent findings with the Australian study have been returned from studies in very different cultural settings. Another similar cross-sectional study (n=375), carried out in Iran, among children (mean age of 8 years) provided evidence of a link between sugar intake and attention deficit hyperactivity disorder. Factor analysis, which identified a sweet dietary pattern, highlighted that those in the top compared to the lowest quintile for this characteristic had an almost four fold increase in risk of the disorder (odds ratio 3.95, 95% CI 1.16 to 15.31).³⁶

As well as attention disorders, intake of sugar is linked to violent behaviour. After adjusting for socio-demographic factors, a cross-sectional study of almost 3,000 5 year olds in the U. S.³⁷ linked diet with reported behaviour from their care-givers. The authors reported that:

"Children who consumed four or more servings of soda per day were more than twice as likely to destroy things belonging to others ([adjusted] OR, 2.54; 95% CI, 1.7-3.8), to get into fights (OR, 2.12; 95% CI, 1.3-3.5), and to physically attack people (OR, 2.28; 95% CI, 1.3-3.9) compared with children who did not drink soda". A dose-response relationship was noted between soda intake and aggressive behaviour scores. Similarly, soda intake was also associated with withdrawn behaviour in a dose-dependent fashion.

In adolescents, similar correlations were observed between reported soda intake and dysfunctional behaviour. In a large North American study, statistical analysis of high school student data (n = 16,188; mean age 16 years) showed dose-response relationships between soda intake and fighting, dysphoria, and suicidal thoughts and actions.³⁸ Other studies have replicated these associations in high school students.³⁹

While many of these adverse behaviours may adversely affect educational attainment, an Australian longitudinal study has directly linked sugar sweetened drink intake with poorer cognitive development in 2868 children. Diet quality was assessed at three years of age, whilst cognitive outcomes were measured using psychological tests at the age of 10 years.⁴⁰

Some studies have concluded that there is no relationship between sugar intake and cognitive function.⁴¹ Unlike the other studies that have been discussed, this meta-analysis looked at the effects of sugar, compared to non-caloric sweeteners in feeding studies which only considered short term intake and its cognitive effects. Subjects were generally fed a high sugar meal and asked to perform various tasks, and had this compared to the same situation after eating a non-calorically sweetened meal. The study concluded that sugar did not affect the behaviour of children. We argue that, keeping in mind the addiction model, this is to be expected. Given the high levels of sugar in Western diets, it is likely that in the short term, children will perform better after eating sugar, as their withdrawal symptoms will be temporarily relieved after a sugar-laden meal. In contrast to other observational studies, this analysis did not consider subjects' usual intake sugar.

Conclusion

If sugar is addictive, as we have proposed, and its effects on mental health are mediated through its addictive properties, long term exposure to sugar is likely to be statistically associated with adverse behavioural measurements. This mechanism explains the epidemiological associations we have reported. Addiction is itself thought to cause impulsivity⁴² and this mechanism is likely to explain the associations found in observational studies between sugar intake and violent behaviour.

Knowledge gained from the therapeutic treatment and public policy changes to control other addictions may be used to justify similar measures to control intake of sugar. Exposure to images of addictive products and easy access to addictive substances are likely to act as cues and triggers, and control of the environment to relieve these stimuli may reduce substance use. Control of cues to consume sugar, through legal control of marketing and advertising to children especially is likely be an important strategy to reduce intake. Educational institutions provide an environment in which such restrictions may be enacted, without the necessity of legal means. Our hope is that the evidence presented here will encourage education leaders to consider the role of food, and their food environment to their students' learning goals.

We have argued that evidence from biology and epidemiological sources shows consistent support for the idea that restricting sugar intake will improve educational attainment and reduce the frequency of problem behaviours. Although we do not have intervention data to support our hypothesis, we see features of the epidemiological data that support a causal link. These include the relatively strong associations (odds ratios between 2 and 4), dose-response effects are consistently described, and some study designs incorporate temporal separation of cause and effect. Further, it is likely that addiction and impulsivity is the underlying plausible biological explanation. Although policies to reduce sugar intake may be justified from a health perspective, it is very likely that these policies will improve what schools are most concerned with: teaching their students. Control of the supply of sugar in and around schools is likely to improve the outlook of both teachers and students alike. Healthier children would only be a fringe benefit.

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