The New Zealand sugar (fructose) fountain: time to turn the tide?
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In 2005 New Zealanders drank and ate, on average, about half a cup (138g) of added sucrose (sugar) per day. In contrast, less than 40g a day (about one and a half tablespoons) are recommended by the World Health Organization to prevent dental caries, obesity and chronic disease.\(^1\) The growth of global sugar demand has been well documented,\(^2,3\) but is it a major concern? Here we review the composition of sugar, its physiological properties, consumption trends, risks it poses to New Zealanders’ health, and finally, should we attempt to turn the tide?

By sugar we mean white table sugar, or crystalline sucrose, derived from sugar cane or beet. Sucrose is also found in less concentrated form in fruits and vegetables, and consists of a single molecule of naturally occurring glucose and fructose, chemically linked. In this article we restrict our discussion to “added sugar”, which are mixed in to food or drink during processing or preparation.

Trends in sugar intake

A national increase in demand for sugar has occurred in the last 40 years. In 1961 daily per capita consumption of added sugar was 126g, which increased to 138g in 2005; a 25% increase occurring mostly between 1980 and 2005.\(^4\) In New Zealand adults, between 8–10% of daily energy consumed came from sucrose consumption, whereas children (aged 5 to 14) consumed between 10–16% of daily energy from this source.\(^5\) In children, 25% came from beverages, with powdered drinks the main source (45%), followed by soft drinks (33%) and cordials and fruit drinks (15%).\(^6\) The other 75% was attributed to a wide variety of foods. Evidence suggests that soft drink consumption is a growing source of sugar in the New Zealand diet with sales increasing 4% per annum in the early 2000s.\(^7\)

What is wrong with sugar?

Traditional arguments mounted against added sugar focus on it as a source of “empty calories”.\(^8\) This statement is true, refined sugar lacks any micronutrients, present in less refined sugar cane derivatives (molasses). However, having “empty calories” does not distinguish sugar from other refined carbohydrates, such as starch. Sugar, like any other purified carbohydrate, has an energy density of 17kJ/g (equivalent to protein). However, fat, has over twice the energy per unit mass (34kJ/g). Therefore its caloric content may not be the main contribution of sugar to adverse health outcomes.

In exploring adverse effects on health, we consider sugar’s chemical constituents. One half of the sucrose disaccharide consists of glucose, commonly found in starch as long chains (present in flour and potatoes) and other disaccharides such as lactose (found in dairy products). Some diets are based on the physiological properties of glucose. The ‘glycaemic index’ (GI), for example, measures the physiological response of
Low glycaemic index diets have been shown, in meta-analyses, to both reduce risk of chronic disease risk and weight in obese people. Serum glucose is commonly measured in plasma to detect diabetes, and average serum glucose level over three months, measured by HbA1c correlates with increased risk of mortality. Sugar, however, has a moderate GI (68), mainly due to its high glucose component, so advocates of GI based diets downplay the role of sucrose in weight gain and chronic disease.

The other half of the sucrose molecule, fructose, has little effect on GI, but is it benign? Fructose receives scant attention in nutritional science or medical practice. While free fructose naturally occurs in honey and fruit the most common source is as a disaccharide, in sugar. What is unusual about sugar is its concentration of fructose. For example, banana typically contains 6% fructose by weight (from both free fructose and sucrose). Sugar, in contrast, has an equal ratio of fructose to glucose (50% by weight). In the United States, high fructose corn syrup often replaces sucrose in food manufacturing. It is simply fructose and sucrose in their elemental form, rather than as a disaccharide. In this article we draw a distinction between concentrated fructose present in refined sugar, and the lower concentrations in naturally occurring sources.

The sugar-refining process not only concentrates sucrose but removes substances which slow its digestion and absorption. Amongst these elements are polyphenols which inhibit digestive enzymes in the human gut. Cross over studies indicate that polyphenol rich meals reduce the glycaemic index of matched carbohydrate loads. Polyphenols therefore favourably slow the absorption of glucose, with likely similar effects on fructose.

After ingestion, fructose is absorbed from the mid to distal small bowel and almost completely metabolised by the liver, independent of the hormone insulin. Unlike glucose, fructose does not stimulate insulin release. Metabolism of fructose depletes intracellular energy stores (ATP), and induces uric acid production. The principal products of hepatic fructose metabolism are triglycerides, which are then released into the circulation.

While fructose is processed, conversion of glucose to glycogen (glycogenesis) in the liver is blocked. The reduction in glucose metabolism, in turn, causes insulin levels to rise so that glucose is taken up in alternative sites, such as muscle tissue. Such high insulin levels leads to compensatory insulin resistance in muscle tissue. This mechanism may explain how fructose has little acute effect on serum glucose levels, but importantly, impairs glycaemic control after long-term exposure to high doses. Further details of fructose physiology are presented elsewhere.

Using animal studies, researchers have documented adverse metabolic effects of refined fructose consumption. Rodents fed on high fructose and sucrose diets, but not high glucose diets develop features of the metabolic syndrome, such as hyperinsulinaemia, hyperuricaemia and hypertriglyceridaemia.

In contrast to animal data, links between fructose and adverse health outcomes have not been so convincingly demonstrated in humans, although longer exposure studies and higher doses of fructose (>200g/day or the equivalent of two cups of sugar) tend to produceclarer adverse health outcomes. For example, small intervention studies
have shown that high doses of fructose provoke insulin resistance within one week, whereas smaller doses (<100g/day) may conversely improve glycaemic control. Whilst 200g is greater than the average New Zealand daily intake (70g/day), variation in consumption means that a substantial proportion of New Zealanders are likely to ingest more than 100g/day. A rise in systolic blood pressure of 7mmHg was observed after intake of 200g of fructose per day for 14 days in a randomised trial (n=74). This study also found adverse effects on triglycerides, fasting insulin and metabolic syndrome outcomes. Other effects include modest weight gain in some short term studies. The health effects of long term, high dose exposure of fructose, which occurs in some subsets of the population have not been studied in experimental trials.

Perhaps the best described effect of fructose consumption is deterioration in lipid profiles. A meta-analysis indicated that fructose worsens serum triglycerides in experimental studies of patients with diabetes, compared with control diets. Increasing evidence supports the association between triglycerides and coronary heart disease, although abnormal triglyceride rich lipoproteins are commonly associated with other adverse lipid abnormalities. Uncertainty still exists over which of these fractions is causally associated with coronary disease.

The best known ill-effect of excess sugar intake is dental caries. The British Nutrition Foundation stated that “the evidence establishing sugars as an aetiological factor in dental caries is overwhelming. The foundation of this lies in the multiplicity of studies rather than the power of any one”. Starch, and other nutrients, in contrast, show little effect. If sugar causes dental decay, and is linked to coronary risk factors, we expect and have indeed found published associations with disease outcomes (after controlling for established risk factors). Although explanations for this relationship have focused on the putative pro-inflammatory role of oral bacteria, the association may be explained by sugar intake (Figure 1).

**Figure 1. A plausible causal diagram explaining nature of the association between dental caries and coronary artery disease. Solid arrows indicate proposed direction of causation while dashed arrow shows apparent association**
Given that sugar-sweetened soft drinks make up a large proportion of added sugar in modern diets, intake of such beverages may be a proxy for sugar exposure. Systematic reviews of the effect of these drinks consistently show associations with adverse outcomes. For example, a meta-analysis of longitudinal studies investigating the correlation between such drinks and increased body weight, showed an $r$-value of 0.09 ($P=0.001$). One randomised study (considered a more compelling design for assessing causation than observational studies) in which obese adolescents were given either supplemental diet soft drinks or no intervention found a beneficial effect, reducing body mass index.

**Sucrose and addiction**

Why is sugar consumption rising? Evidence points to sugar possessing rewarding qualities similar to drugs of abuse. Addiction is defined as a loss of control, usually associated with the intake of specific drugs that induce consumption of increasing amounts of the substance after initial exposure. Alcohol and opiate dependence, or addiction, are often perceived by society as ‘serious addictions’ usually because impaired social relationships and work performance coexist. Conversely, dependence on nicotine and caffeine, for example, are considered ‘lesser addictions’ as they do not necessarily dominate the addict’s life. These ‘lesser’ addictions do, nevertheless, share many of the other clinical features of more severe syndromes. For example, the repeated quenching of unpleasant withdrawal symptoms from substance use leads to strong negative re-inforcement of such behaviour, shifting drug ingestion from consciously initiated to automated actions. Some readers may be familiar with the relief of mild caffeine withdrawal symptoms; such as irritability and reduced concentration; which often follow the drinking of a cup of coffee.

The biological basis of addiction offers clues to why some substances are rewarding. Symptoms of addiction are linked to part of the brain responsible for subconscious control of behaviour and motivation. The dopaminergic mesocorticolimbic projection, present in the midbrain, is most often implicated. Human and animal studies show changes in this region after exposure to addictive substances. For example, an intravenous bolus of cocaine results in a spike in extracellular dopamine by blocking re-uptake by nerve terminals in the nucleus accumbens. Also, drug induced dopamine release in this projection is associated with “feeling high”.

Is sugar consumption similar to other addictive behaviours? Although by no means widely accepted in nutritional circles, evidence supports such a link. Other articles more fully evaluate the evidence for sugar consumption and obesity sharing features of addiction, so we only briefly discuss the salient evidence. Of all the food groups, carbohydrate is commonly ascribed addictive properties, and within this food group, sugar. In humans, carbohydrate craving has often been reported in obese people, although a full withdrawal syndrome has not been described. We portrayed one case of an obese woman who recounted a likely food withdrawal syndrome after abstinence from sugar and white flour, whose symptoms resolved after about one month. This pattern is similar to the temporality of symptoms observed in other addiction syndromes after abstinence. Obese people also show anatomical changes similar to people who suffer from drug addiction, with increased density of dopamine receptors in reward centres compared to controls.
Is action justified?

Observational studies document a range of adverse associations with sugar or soft drink consumption, and limited numbers of experimental studies indicate that such associations are likely to be causal. Unlike other addictions with adverse health effects (such as tobacco), no regulation discourages consumption, and in medical circles, little appreciation of such adverse health effects have surfaced. Other authorities noted similar evidence of adverse outcomes from sugar consumption, yet conclude their advice with no restrictions.31 Our opinion is that sugar is contributing to obesity, diabetes and associated cardiovascular disease, and by its addictive nature will resist restraint.

Other counter arguments may suggest that sugar is ubiquitous and unlikely to pose a significant health threat, because it only consists of naturally occurring sugars, albeit in a more concentrated form. Sir Richard Doll reflected on a similar point, when in 1947, a cause for an epidemic of lung cancer was sought. Several exposures were mooted - from pollution to arsenic - however smoking was discounted because it “...was such a normal thing and had been for such a long time it was difficult to think that it could be associated with any disease.”32

From a public health viewpoint, we must consider possible negative consequences of taking action. Restricting sugar intake is unlikely to cause unintended adverse nutritional effects because sugar is devoid of trace micronutrients. At worst, reducing sugar consumption is likely to improve oral health; at best, it will lower rates of obesity, diabetes and cardiovascular disease.

For the clinician, patients with risk factors for, or established, coronary artery disease are likely to benefit from advice to severely limit sugar intake, noting common sources to avoid. Doctors may warn patients that symptoms such as craving, irritability, and limited concentration may peak in the first days after abstinence, but wane after about one month. Careful monitoring for improvement and change in drug requirement (for oral hypoglycaemics and antihypertensives particularly) is prudent in the early stages. Simple quit techniques may also help such as removing the substance from easy access to reduce the likelihood of relapse.

Experience from public health initiatives to reduce smoking prevalence indicates that individual treatment has only a weak effect compared with a more comprehensive population approach such as food reformulation and economic incentives to change behaviour. We agree with arguments to consider incentives to reduce sugar in manufactured foods, such as taxation or legislation, or directly taxing high-sugar beverages themselves.33 Revenues from such a strategy can be directed to promote healthier food and drinks.

Other population level measures, drawn from tobacco control, include restricting sales and marketing of sugar sweetened products, particularly to younger consumers. Whilst we acknowledge that such a move may be unpopular, the negative externalities that accrue from escalating health care costs of obesity and diabetes, require bold and assertive action if we are to reverse this tide.

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