Current opinion: Is added dietary sugar detrimental to health?

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Abstract

The intake of added dietary sugars has escalated dramatically since the 1970s, mostly due to a global increase in the consumption of sugar-sweetened beverages (SSBs). This trend has sparked concerns among scientists and consumers alike regarding the safety of added dietary sugars. The popular media often accuse dietary sugars of being detrimental to health. However, when consumed in moderate amounts, current research does not support the premise that added dietary sugar has a detrimental effect on dental health, mental health and behaviour, weight management, chronic diseases of lifestyle, or the intake of micronutrients. However, SSBs represent concentrated and energy-dense forms of dietary sugars, easily consumed in large amounts. Not surprisingly, excessive consumption of these beverages is associated with weight gain and obesity. Intake of more than one to two drinks per day is also linked to the metabolic syndrome (MS), insulin resistance, type 2 diabetes, cardiovascular diseases, hypertension, gout and non-alcoholic fatty liver disease. Experimental studies suggest that excessive fructose intake may play a direct role in the metabolic changes that manifest as the MS, but further research into this is required. Current recommendations state that added sugars may be enjoyed in moderation as part of a balanced diet, with an appropriate energy intake to achieve or maintain healthy weight. However, excessive intake of added sugars, particularly fructose-containing sugars, should be avoided, and the energy intake from SSBs should be limited to less than 400-600 kJ per day.

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Introduction

Since ancient times, human beings have displayed a preference for sweet foods like honey, berries and dates. In modern societies, sugar intake consists of about 50% natural sugars in fruits, vegetables, milk and grains, while the rest comprises refined sugars that are added to foods during processing, during preparation, or at the table.1 On food labels, added sugars are listed as various forms of sugar (as sucrose, molasses, or white or brown sugar), invert sugar, glucose (dextrose), fructose (levulose), maltose, malt syrup, corn sweeteners, high-fructose corn syrup (HFCS) and honey.² During food processing, sugars are added to enhance flavour, to improve taste and to provide fuel for fermentation when baking with yeast, or for alcohol production. Sugars also supply texture and colour to baked goods, act as a bulking agent in ice cream and confectionary, preserve jams and canned fruit, and balance acidity in tomato- and vinegar-based products.1

Since the 1970s, the use of added sugars has escalated globally, mainly due to the growing consumption of sugar-

sweetened beverages (SSBs), including soft drinks, sugared fruit drinks, iced teas and sport drinks.³ Over the last decade, there have been growing concerns regarding the safety of the consumption of added sugar. In the lay media, added dietary sugars are constantly accused of causing a wide variety of serious health problems. Several websites, popular magazine articles, and even advertising campaigns for alternative sweeteners, go so far as to liken sugar to a "poisonous" substance that "ruins health", leaving consumers very confused. The aim of this review is to briefly summarise the current scientific debate regarding the impact of added sugars on health, including dental and mental health, behaviour, weight management, chronic lifestyle diseases, and micronutrient dilution.

Dental caries

Added and natural dietary sugars, as well as sugars derived from the digestion of starch by amylase in saliva, may all contribute to tooth decay. During the first 20-30 minutes after exposure to food, *Streptococcus mutans* (normally present in the mouth) ferments these sugars and

produces an acid that erodes tooth enamel.¹ The extent of the damage to the enamel depends on the duration and frequency of exposure to the acid-generating food.^{4,5} Stimulation of saliva hastens clearance of the substrate from the mouth and buffers acidity. Experimental studies reveal that, while pure sucrose reduces mouth pH, it also stimulates salivary flow and is dispelled from the mouth relatively fast. In contrast, sticky foods, even those with low sugar content, such as cooked starch, also reduce oral pH, but are cleared slowly and therefore cause more tooth decay than high-sugar foods, which are consumed quickly.5 Similarly, prolonged exposure of enamel to any acid-producing food or beverage at a sustained $pH < 5.5^6$ (e.g. people who snack all day, babies put to bed with a bottle, or athletes constantly sipping sports drinks) is more erosive than brief exposure.¹ SSBs such as soft drinks, sport drinks and citric juices, are also quite acidic (pH 2.4-4.0)⁷⁻⁹ and can erode enamel independently of the sugar content.¹⁰ Non-sugary foods help remove sugar from the teeth, hence added sugar is best consumed with meals.1 Cheese and milk buffer mouth acids and may minimise the damage caused by these acids to tooth enamel.¹¹ A systematic review concluded that good oral hygiene and regular brushing with a fluoride-containing toothpaste is more effective for preventing cavities than avoiding sugary foods.12

Mental health

Sugar has been blamed for causing misbehaviour in hyperactive children and even criminal behaviour in adults.¹ However, studies, including a meta-analysis, have failed to confirm these relationships.^{1,13} However, many parents firmly believe that sugar is the cause of their children's hyperactive behaviour. (It is noteworthy that occasions such as children's parties, where children are prone to consuming large amounts of sugary foods, are also associated with plenty of excitement, which may overstimulate children independently of what they consume.²)

Media articles have linked sugar intake to Alzheimer's disease, schizophrenia and depression. However, a recent systematic review⁵ revealed no studies on Alzheimer's, but identified a single study showing an inverse association between sugar intake and the severity of schizophrenia symptoms.¹⁴ However, the reviewers concluded that the results of this study were confounded by medication.⁵ A single study demonstrated that depressed subjects consumed more carbohydrates and sugar than controls,¹⁵ which could suggest a causative role for sugar, or merely indicate a preference among depressed individuals for sweet, high-carbohydrate foods.

Sugar addiction

Animal models and a few human studies suggest that carbohydrates and sugary foods may have addictive potential, by triggering the release of opioids and dopamine.¹⁶ These studies suggest that, in certain genetically predisposed individuals, the consumption of sugar-rich foods or drinks may prime the release of euphoric endorphins and dopamine within the nucleus accumbens, in a manner similar to some drugs.¹⁶ Some evidence also suggests that cross-sensitisation between sugar addiction and narcotic dependence may occur in certain genetically predisposed individuals.¹⁶ However, controlled human trials to support the theory pertaining to the addictive potential of sugar are lacking, and some researchers strongly argue that there are currently human studies to truly support the theory.¹⁷ Notably, a controlled trial has found that self-described "sugar addicts" tend to eat more of everything when they indulge, without this resulting in an elevation in the energy percentage derived from sugar.18

People are often motivated to completely eliminate sugar from their diets due to their perception of sugar as a "bad" food. It is plausible that such self-imposed restriction may actually increase the desire for the "forbidden" substance, and trigger cravings for sweet foods.¹ Thus cravings may be more psychological than physiological,⁵ similar to the way in which a capsule containing the pharmacoactive substance commonly found in chocolate does not satisfy chocolate cravings.¹

Weight gain, overweight and obesity

The aetiology of obesity is a complex interplay of genetics and environmental factors, but in essence translates to excess energy intake (primarily attributed to energy-dense diets) that is not balanced by appropriate levels of energy expenditure (ascribed to inadequate levels of physical activity).^{18,19} Excessive intakes of any food or beverage that lead to energy intakes that exceed energy expenditure will therefore cause weight gain. Added sugar contributes to the palatability of products such as confectionary and desserts, and encourages over-consumption of these high fat, energy-dense foods, while further contributing to the overall energy density of these products. By itself, however, sugar, like all carbohydrates and proteins, is only half as energy dense (17 kJ/g) as fat (38 kJ/g).¹ When total energy intakes are controlled, sugar should therefore not cause weight gain any more than any other carbohydrate or protein food. Likewise, studies found that including moderate amounts of sugar in an energy-restricted diet did not negatively affect weight loss compared to diets that did not contain any added sugar. Indeed, a bit of added sweetness was found to improve compliance to energy-restricted diets.^{1,20-22} Epidemiological studies have reported an inverse association in certain populations between high sugar intakes on the one hand, and fat intake and weight gain on the other. This is called the sugar-fat see-saw effect.^{5,23} Research also does not support the notion that sugar increases appetite and stimulates overeating, but rather demonstrates that sucrose may contribute to satiety.²⁴

The global increase in the prevalence of obesity is paralleled by the escalating consumption of SSBs, hinting at a possible connection.^{25,26} Indeed, recent meta-analyses link high intakes of SSBs to weight gain, overweight and obesity.^{25,26} This is not surprising since these beverages are energy-dense (170-200 kJ/100 ml) and easily consumed in large amounts. In practice, dietitians often record diet histories that reveal intakes by clients of one to two litres of these beverages per day, which constitutes at least 1 700-3 400 kJ/day.

Metabolic syndrome and chronic diseases of lifestyle

Recent systematic reviews and meta-analyses demonstrate positive associations between SSB intake and the cluster of chronic lifestyle diseases, referred to as the metabolic syndrome (MS).^{3,27}

The current model of the MS identifies insulin resistance (IR) as the pivotal pathophysiological change.27-29 Although not all patients with IR are overweight, excess body fat, particularly visceral fat stored around the vital organs, dramatically raises the risk for MS and associated chronic lifestyle diseases. As body size increases, expanding lipocytes produce more proinflammatory cytokines such as TNF-alpha, and less anti-inflammatory cytokines such as adiponectin. These, and other metabolic changes, contribute to increasing IR, which triggers compensatory insulin secretion by the pancreas and gives rise to progressive hyperinsulinaemia, which in turn may increase IR even further.²⁷⁻²⁹ Chronic stimulation of insulin secretion by the pancreas eventually destroys the beta cells, leading to postprandial hyperglycaemia, which, in some patients, may progress to type 2 diabetes.²⁷⁻²⁹ Hyperinsulinaemia also inhibits sodium and uric acid excretion by the kidneys, thus contributing to hypertension and gout. IR and hyperinsulinaemia are further associated with increased levels of circulating triglycerides and lowdensity lipoprotein (LDL), particularly small, dense LDL particles, which are vulnerable to oxidation in the proinflammatory environment associated with obesity.

Oxidised LDL (ox-LDL) is intimately involved in the aetiology of atherosclerotic plaque formation and subsequent cardiovascular diseases.²⁷⁻²⁹ High levels of circulating triglycerides also become trapped in the liver, causing hepatic inflammation and fibroblastic changes in the liver, some level of which is evident in most obese people. This is referred to as non-alcoholic fatty liver disease, and may eventually progress to liver cirrhosis.^{28,29}

Agrowing body of evidence, including results of experimental, large observational, and long-term prospective cohort studies, which followed large populations over extended periods of time, links SSB intake to type 2 diabetes,³ cardiovascular diseases,^{30,31} hypertension³⁰ and gout.³² A recent meta-analysis concluded that a regular SSB intake of one to two drinks per day, versus intakes of none or one drink per month, increased the risk for MS by 20% and that for type 2 diabetes by 26%.³ Given the energy density and easily consumable nature of SSBs, an association between high levels of SSB intake and chronic metabolic diseases linked to IR is expected.

Animal studies, as well as several small short-term controlled clinical trials (< 40 participants, < 10 weeks), implicate a direct role of fructose in the development of IR and the manifestations of the MS, at least in obese subjects.33 In the USA, soft drinks and most SSBs are sweetened with HFCS,²⁵ while in South Africa these beverages are sweetened with sucrose (information provided by the South African Sugar Association). HFCS is usually a 55%:45% mixture of fructose and glucose as monosaccharides, while sucrose is a disaccharide comprising glucose and fructose. Therefore, both HFCS and sucrose constitute roughly 1:1 mixtures of fructose and glucose. In a recent trial,³⁴ overweight adults consumed three glasses of either a glucose-sweetened beverage (n = 15), or a fructose-sweetened beverage (n = 17), per day, over a period of 10 weeks without any changes to their normal diets. Both groups gained on average of 1.4 kg in weight and 0.8 kg in fat mass over the 10 weeks. Interestingly, however, the fat gain in the glucose group was subcutaneous, while that in the fructose groups was visceral. Contrary to the glucose group, the fructose group also developed increased fasting glucose and insulin levels, decreased hepatic insulin sensitivity, increased 24-hour postprandial triglyceride levels, as well as increased blood levels of apo-B, LDL, small, dense LDL and ox-LDL.³⁴

In light of these concerns, the American Heart Association recommends that energy from SSB should be limited to 400-600 kJ/day, or one drink per day.³⁵

The question that arises is whether high levels of fruit juice consumption may have a similar effect to high consumption of SSBs, since pure fruit juice has a high fructose content and is as energy dense as SSBs. However, pure fruit juice contains high levels of beneficial micronutrients and phytochemicals, which SSBs do not. Studies linking SSB intake to metabolic outcomes generally did not report on fruit juice intakes. However, a large cohort linking SSBs to gout did reveal that total fruit juice intake was also associated with a higher risk of gout,³² but in the Framingham Offspring Study, fruit juice consumption was inversely associated with fasting glucose levels, although not with fasting insulin.³⁶ Pending further studies, it may be prudent at this stage to advocate the consumption of whole fruits rather than fruit juice, since experimental studies suggest that whole fruits (which are far less energy dense than fruit juice) do not exert the same detrimental metabolic effects as shown with the SSBs.^{37,38}

Glycaemic response

The rate, magnitude and duration of the rise in blood glucose that occurs after a particular food or meal is consumed is referred to as the glycaemic response. The glycaemic response is expressed as the glycaemic index (GI), which quantifies the rate and duration of the response, and the glycaemic load (GL), which quantifies the magnitude of the response.² An exaggerated glycaemic response following a meal or a snack triggers an exaggerated insulin response,² which, on a chronic basis, may contribute to the negative metabolic manifestations of hyperinsulinaemia.³⁵

Different types of carbohydrates are digested at different rates, and thus have different GIs. Many factors influence GI, but fibre, proteins and fat generally slow down stomach emptying and digestion and reduce glycaemic responses after a carbohydrate-containing meal.² Sucrose contains equal amounts of glucose (high GI) and fructose (low GI) and therefore has an intermediate GI when consumed on its own.^{1,39} When consumed as a part of a low-GI meal, moderate amounts of sucrose do not cause exaggerated insulin responses.³⁹ Despite the inherent intermediate GIs of sucrose or HFCS, refined carbohydrates such as SSBs, when consumed alone, should be more rapidly digested than added sugars in meals. Also, since SSBs are very energy dense, they should result in larger GLs (the product of GI and the absolute amount of carbohydrate per serving). SSBs, particularly when consumed in large volumes, may therefore cause high glycaemic responses that trigger exaggerated insulin responses.35

Nutrient dilution

Since all refined sugar, even honey and molasses (contrary to popular belief), is pure carbohydrate, and devoid of micronutrients, it may be viewed as "empty energy". A major health concern is that the overconsumption of sugary foods may displace more nutritious food from the diet and lead to vitamin and mineral deficiencies in vulnerable groups like children, adolescents and the elderly.¹ Although it seems plausible that added sugar intake at high levels will cause nutrient dilution, studies are as yet inconclusive on the maximum level of added sugar consumption to prevent such a dilution effect.^{40,41} Currently, most countries and the World Health Organization (WHO) recommend that an added sugar intake up to 10% of total daily energy is compatible with adequate micronutrient intake, provided that a variety of foods is consumed from all the food groups.^{40,41} Nutrient dilution studies have also found that added sugars improve the taste of more nutrient-dense foods, such as enriched breakfast cereals, making it more likely that children will eat these products, thus contributing to adequate micronutrient intakes. Recent systematic reviews40,41 have failed to demonstrate a conclusive link between sugar consumption per se and nutrient dilution, but did highlight several methodological problems that make comparison between studies difficult, and which need to be addressed in further studies.⁴² On the other hand, a meta-analysis of SSB intake and health did find inverse relationships with intakes of milk and calcium.25

Conclusion and recommendations

The current body of evidence does not support the premise that added dietary sugar, when used moderately as part of a balanced diet, results in detrimental effects on health. This is based on the presumption that the balanced diet supplies appropriate energy levels to achieve and maintain a healthy body weight, which will differ among individuals, depending on height, lean muscle mass and activity levels.^{1,2,39} A wellbalanced diet may be achieved by eating a wide variety of foods from each of the different food groups depicted in the Food Guide Pyramid¹ and by following the South African Food-Based Dietary Guidelines.⁴³

Added sugar is described as "discretionary energy" that can be used in the diet after all the other food groups are included in the recommended amounts.35 Few people can eat large amounts of sugary treats and still meet all their nutrition requirements without exceeding their energy allowance.1 Current research indicates that excessive intake of sugar, particularly in the form of energy-dense and easy-to-consume sugar sweetened beverages, may lead to weight gain and obesity-related health problems, such as the MS. Moreover, some evidence suggests that excessive amounts of fructose may be metabolised differently in obese subjects than it would be in normal-weight subjects, and may directly contribute to insulin resistance and metabolic disorders. There is a need for randomised controlled human studies to clarify the issues raised in this review. Meanwhile, it seems prudent to at least restrict the consumption of sugar-sweetened beverages to 400-600 kJ or less per day.35

References

- Rolfes SR, Pinna K, Whitney E. Understanding normal and clinical nutrition. 8th ed. Canada: Wadsworth Gengage Learning; 2008;117.
- Smolin LA, Grosvenor MB. Nutrition: science and application, 4th ed. USA: John Wiley & Sons, Inc; 2007:145.
- Malik VS, Popkin BM, Bray GA, et al. Sugar sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diab Care. 2010. (Epub ahead of print).
- Anderson CA, Curzon ME, Van Loveren C, et al. Sucrose and dental caries: a review of the evidence. Obes Rev. 2009;10(1):S41-54.
- Ruxton CHS, Gardner EJ, McNulty HM. Is sugar consumption detrimental to health? A review of the evidence: 1995-2006. Crit Rev Food Sci Nutr. 2010;50(1);1-19.
- Tahmassebi JF, Duggai MS, Malik-Kotru G, Curzon MEJ. Soft drinks and dental health: A review of the current literature. J Dent. 2006;34:2-11.
- Jain P, Nihill P, Sobkowsk J, Agustin MZ. Commercial soft drinks: pH and in vitro dissolution of enamel, Gen Dent. 2007;55(2):167-7.
- Rees J, Loyn T, McAndrew R. The acidic and erosive potential of five sports drinks. Eur J Prosthodont Restor Dent. 2005;13(4):186-90.
- Aliping-McKenzie M, Linden RWA, Nicholson JW. The effect of Coca-Cola and fruit juices on the surface hardness of glass-ionomers and "compomers." J Oral Rehab. 2004;31:1046-1052.
- Wongkhantee S, Patanapiradej V, Maneenut C, Tantbirojn D. Effects of acidic foods and drinks on surface hardness of enamel, dentine, and tooth-coloured filling materials. J Dent. 2006;34:214-220.
- Kashket S, DePaola DP. Cheese consumption and the development and progression of dental caries. Nutr Rev. 2002;60(4):97-103.
- Burt BA, Pai S. Sugar consumption and caries risk: a systematic review. J Dent Edu. 2001;65(10):1017-1023.
- Wolraich ML, Wilson DB, White JW. The effect of sugar on behaviour or cognation in children: a meta-analysis. JAMA. 1995;274(20):1617-1621.
- Stokes C, Pete M. Dietary sugar and polyunsaturated fatty acid consumption as predictors of severity of schizophrenia symptoms. Nutr Neuroscience. 2004;7:247-9.
- Christensen L, Somers S. Comparison of nutrient intake between depressed and non-depressed individuals. Int J Eating Dis. 1996;20:5-9.
- Fortuna JL. Sweet preference, sugar addiction and the familial history of alcohol dependence: shared neural pathways and genes. J Psychoactive Drugs. 2010;42(2):147-151.
- Burton D. The plausibility of sugar addiction and its role in obesity and eating disorders. Clin Nutr. 2010;29(3):288-303.
- Gee M, Mahan LK, Escott-Stump S. Weight management. In: LK Mahan & S Escott-Stump, eds. Krause's food & nutrition therapy. 12th ed, Saunders Elsevier. 2008:540-542.
- Saris WHM. Sugars, energy metabolism and weight control. Am J Clin Nutr. 2003;78:S850-7.
- White C, Drummond, De Looy A. Comparing advice to decrease both dietary fat and sucrose, or dietary fat only, on weight loss, weight maintenance and perceived quality of life. Int J Food Sci Nutr. 2010;61(3):282-294.
- Kirkwood L, Aldujaili E, Drummond S. Effects of advice on dietary intake and/ or physical activity on body composition, blood lipids and insulin resistance following a low-fat, sucrose-containing, high-carbohydrate, energy-restricted diet. Int J Food Sci Nutr. 2007;58(5):383-397.
- Black RN, Spence M, McMahon RO, et al. Effect of eucaloric high- and lowsucrose diets with identical macronutrient profile on insulin resistance and vascular risk: a randomised controlled trial. Diabetes. 2006;55(12):3566-3572.
- Gibney M, Sigman-Grant M, Stanton JL, Keast DR. Consumption of sugars. Am J Clin Nutr. 1995;62:S178-94.
- 24. Anderson GH, Woodend D. Consumption of sugars and the regulation of short-term satiety and food intake. Am J Clin Nutr. 2003;78:S843-9.
- Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systemic review. Am J Clin Nutr. 2006;84:274-288.
- Vartanian LR, Schwartz MB, Brownell KD. Effects of sugar consumption on nutrition and health: a systemic review and meta-analysis. Am J Pub Health. 2007;97(4):667-675.
- 27. Alberti KGMM, Eckel RH, Grundy SM, et al. Harmonising the metabolic

syndrome: a joint interim statement of the International Diabetes Federation task force on epidemiology and prevention; National Heart, Lung and Blood Institute; American Heart Association; World Heart Federation; International Atherosclerosis Society; and International Association for the Study of Obesity. Circulation. 2009;120:1640-1645.

- Eckel RH, Grundy SM, Zimmet PZ. The metabolic syndrome. Lancet 2005;365:1415-1428.
- Monteiro R, Azevedo I. Chronic inflammation in obesity and the metabolic syndrome: mediators of inflammation. [homepage on the Internet]. c2010. Available from www.hindwi.com/journals/mi/2010/si.miom.html
- Dhingra R, Sullivan L, Jacques PF, et al. Cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 2007;116:480-488.
- Fung TT, Malik V, Rexrode KM, et al. Sweetened beverage consumption and risk of coronary heart disease in women. Am J Clin Nutr. 2009;89:1037-1042.
- Choi HK, Curhan G. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. BMJ. 2008;336(7639)309-312.
- Stanhope KL, Havel PJ. Fructose consumption: recent results and their potential implications. Ann NY Acad Sci. 2010;1190:15-24.
- Stanhope KL, Schwarz JM, Keim NL, et al. Effects of consuming fructose- or glucose-sweetened beverages for 10 weeks on lipids, insulin sensitivity and adiposity. J Clin Invest. 2009;119:1322-1334.
- Johnson RK, Appel LJ, Brands M, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation. 2009;120:1011-1020.
- 36. Yoshida M, McKeown NM, Rogers G, et al. Surrogate markers of insulin resistance is associated with consumption of sugar-sweetened drinks and fruit juice in middle and older-aged adults. J Nutr. 2007;137(9):2121-2127.
- Busserolles J, Gueux E, Rock E. Substituting honey for refined carbohydrates protects rats from hypertriglyceridemic and prooxidative effects of fructose. Br J Nutr. 2002;132:3379-3382.
- Busserolles J, Gueux E, Rock E. Oligofructose protects against the hypertriglyceridemic and pro-oxidative effects of a high fructose diet in rats. Br J Nutr. 2003;133:1903-1908.
- American Dietetic Association. Nutrition recommendations and interventions for diabetes (position atatement), Diab Care. 2007;30:S48.
- Rennie KL, Livingstone MBE. Associations between dietary added sugar intake and micronutrient intake: a systematic review. Br J Nutr. 2007;97:832-841.
- Gibson SA. Dietary sugars and micronutrient adequacy: a systematic review of the evidence. Nutr Res Rev 2007;20:121–31.
- 42. Livingstone MBE, Rennie KL. Added sugars and micronutrient dilution. Obesity Rev 2009;10:S34–40.
- South African Food Based Dietary Guidelines. Available from http://www. sahealthinfo.org/nutrition/foodbasedsept2001.pdf (Accessed: 10/09/2010).

