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TE APĀRANGI

Sugar and Health

Evidence update

Sugar and health is a topic that is surrounded by mixed messages in the New Zealand media. This summary of the current evidence sets out to remove some of the confusion around sugars in food, sugar intake, how sugars are processed in the body, and health effects from sugar consumption.

Consumption of sugar and sweetening of food has steadily increased over time, and so has disease related to diet.[1 2 3] The increasing numbers of New Zealanders who are overweight and/or have diabetes[4 5 6] has spurred interest in the role of sugars in diet and their effects on health.[7 8 9 10 11]

Many studies in New Zealand and internationally have provided important information about the links between sugar and disease. How sugar influences health and physical conditions that in turn can lead to chronic disease is increasingly being clarified, and many linkages have been established.[12 13 14]

Summary – sugar and health

EVIDENCE

There is strong evidence linking sugar consumption with increased body weight as well as tooth decay.

HEALTH

Many studies also show an association between a high intake of added sugars and obesity, type-2 diabetes, heart disease and gout – however research to date has not yet fully established the exact mechanisms behind these relationships.

REDUCING

No study has shown any negative effects from reducing sugar intake in the diet.

ADDING

The current discussion on negative health effects from sugar is mainly concerned with sugars, such as sucrose, that have been added by food manufacturers.

DRINKS

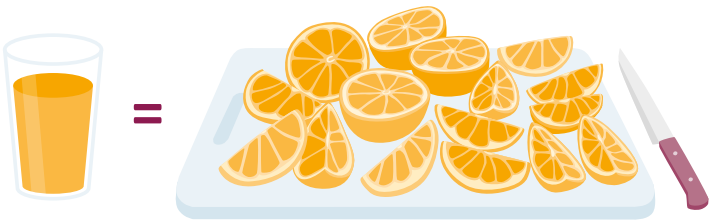
Beverages which are high in sugar are nutrient and fibre poor, and can readily lead to overconsumption because they do not lead to a sense of fullness.

WHO

The World Health Organization (WHO) recommends a maximum intake of sugars of less than 10% of total energy intake for adults and children, and less than 5% for better health, excluding sugars found in whole fruits, milk and vegetables.

LABELS

Current food labelling does not allow consumers to assess how much sugar has been added to food and drinks, making it difficult to follow dietary recommendations and guidelines.



Sugar in food

Sugar is a carbohydrate found in natural foods such as fruit, vegetables, honey and milk. Sugar is also used in refined form by manufacturers to sweeten foods and drinks, and for food preservation.

The most common sugars are sucrose, glucose, lactose and fructose. Sucrose is the naturally-occurring combined form of glucose and fructose, also called white sugar or table sugar. Lactose is found in dairy products. These sugars all appear in foods and are classified as added, free and intrinsic sugars:[15] [16]

- *Added sugars* are extracted, concentrated and refined from sources such as sugar cane, fruit, sugar beet, or corn.
- *Free sugars* include added sugars but also include sugars naturally found in honey, syrups and fruit juices – foods that are usually thought of as 'sweeteners'.[17]
- *Intrinsic sugars* are found naturally in the cell structure of foods such as fresh fruit, grains, vegetables and milk.

Foods with high levels of added sugars can greatly increase the energy (calorie) content of foods without providing any other nutritional benefits, as opposed to whole foods with intrinsic sugars that are accompanied by other nutrients, such as dietary fibre, vitamins, and minerals.[18]

Fruit juice and added sugars in a sweet drink may be more detrimental to your health compared with eating a whole fruit with its intrinsic sugars, for many reasons:

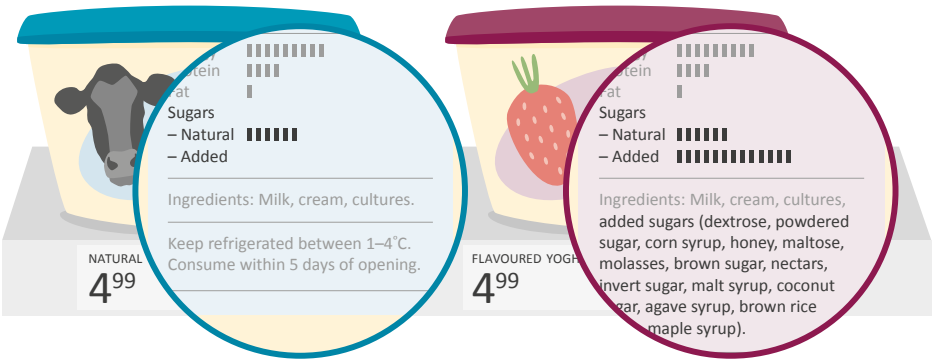
- While the whole fruit also contains minerals, vitamins and fibre with the sugar, the sugary drink mostly consists of water and added sugars.
- The nutritional content will be lower for the drink than for the fruit, while the concentration of sugar will be higher.
- The concentration of sugar can be illustrated by one glass of orange juice, which can contain sugar from as many as five oranges.
- You would be less likely to eat five oranges in one go, as the actual fruit will be more filling due to the fibre content and volume.[19] [20]
- If the sugar comes with its inherent fibre (as with whole fruit) then up to 30% of this sugar will not be absorbed. Instead, it will be metabolised by the microbes in the gut, which may improve microbial diversity and help prevent disease. The fibre will also mean a slower rise in blood glucose, which has shown to have positive health effects.[21]
- The sugary drink will be easier to consume in large amounts, which increases the overconsumption risk[22] – this is partly because the sugary drink does not offer the same sense of fullness as the equivalent amount of food.[23] [24]

Added sugars are hard to detect in food and drink

The current discussion on negative health effects from sugar is mainly concerned with sugars, such as sucrose, that have been added by food manufacturers. Sucrose (derived from refined sugar cane or sugar beet) and high-fructose corn syrup (derived from corn) are used by food manufacturers for sweetening foods and drinks, for food preservation and for enhancing the flavour of processed foods.[25] [26]

In New Zealand, sucrose is commonly used to sweeten fizzy drinks and beverages, whereas high-fructose corn syrup is more commonly used in the US food industry. In foods, added sugars are often found in packaged and processed foods, such as sugary and fizzy drinks, breakfast cereals, muesli bars, biscuits, baked products (cakes, tarts, puddings) and cooking sauces.[27]

Examples of other names for sugar that might be found in packaged foods are dextrose, powdered sugar, corn syrup, honey, maltose, molasses, brown sugar, nectars (e.g. peach nectar), invert sugar, malt syrup, coconut sugars, agave syrup, brown rice syrup and maple syrup.[28] [29]



How much sugar should we be eating?

The World Health Organization recommends that we eat less free sugar* but the guidelines do not apply to intrinsic sugars that are naturally present in fruit, milk and vegetables.[30]

For both adults and children, WHO states that the intake of free sugars should be reduced to less than 10% of total energy intake. That is about 50g, or around 12 teaspoons per day (one teaspoon = 4g of sugar). Further reductions to below 5% of the total energy intake (or around 6 teaspoons) could provide additional health benefits.

The infographic on the right illustrates some common everyday foods and their sugar content.[31] [32]

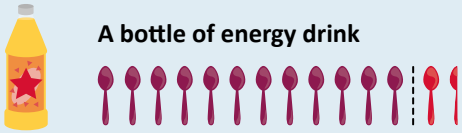
Food manufacturers in New Zealand do not normally need to declare how much sugar is added and how much sugar is naturally present in a particular food – only the total sugar content must be declared.[33] This makes it difficult for the consumer to recognise levels of added sugars in food; sugars which should be limited according to current dietary recommendations.[34]

The US Food and Drug Administration have made it compulsory to include added sugars in grams and as a percentage of the daily diet to food labels in the USA from year 2018.[35] At present there is no move to do the same in New Zealand.

The US food labels will state calorie content and level of added sugar

It is recommended to have no more than 12 teaspoons of added sugars each day

How many teaspoons of sugar are found in some common food and drinks?



* Definition: "free sugars [...] include monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, *and* sugars naturally present in honey, syrups, fruit juices and fruit juice concentrates" (WHO 2015).

What happens in the body when we eat sugar?

Sugar metabolism is the process by which energy contained in the foods that we eat is made available as fuel for the body.[36] The body's cells can use glucose directly for energy, and most cells can also use fatty acids for energy. Glucose and fructose are metabolised differently, and when they are consumed in excess they may have different implications for health.[37] [38]

Looking at glucose first – when food is consumed, there is a corresponding rise and subsequent fall in blood glucose level, as glucose is absorbed from the gastrointestinal tract into the blood and then taken up into the cells in the body.

Glucose in the blood stimulates the pancreas to release insulin, which then triggers uptake of glucose by cells in the body (e.g. muscle cells) causing blood glucose to return to base levels.[39]

Insulin will turn off fat burning and promote glucose burning as the body's primary fuel source.[40] Any excess glucose ends up being stored as glycogen in the muscles, and it can also be stored as lipid in the fat tissue.

Fructose is also taken up into the blood from the gut, but in this case, the liver serves as a pre-processing organ that can convert fructose to glucose or fat.[41] [42] The liver can release the glucose and fat into the blood or store it as glycogen or fat depots, which, if sugars are consumed in excess, may lead to fatty liver disease and also increase risk for diabetes and cardiovascular disease.[43]

There are also some noted interaction effects between glucose and fructose, in that glucose enables fructose absorption from the gut, while fructose can accelerate glucose uptake and storage in the liver.[44]

Excessive consumption of added sugars can lead to serious illness

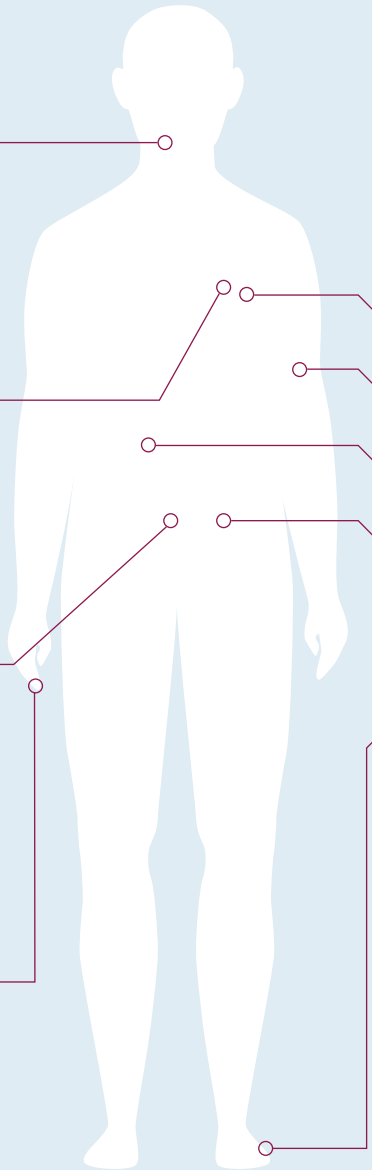
Researchers have found many negative health effects that are related to excessive sugar consumption:

Dental health
Tooth decay increases with sugar consumption, and high-sugar diets are associated with a higher dental caries risk.

Heart disease
People who consume high amounts of sugary foods or drinks are more likely to be at risk of heart disease.

Weight gain (especially abdominal fat)
Increased sugar intake and consumption of added sugars in sugar-sweetened beverages have been linked to body weight gain.

Diabetes
Evidence shows that people who consume high amounts of sugary foods or drinks are more likely to be at risk of type-2 diabetes.



There are a number of scientific studies proposing that fructose may have particularly negative effects on health.

If very high doses of fructose are consumed, fat can accumulate in the liver. This, and other mechanisms, could lead to a range of health conditions.

Fat in the blood

Increased blood pressure

Fatty liver disease

Insulin resistance

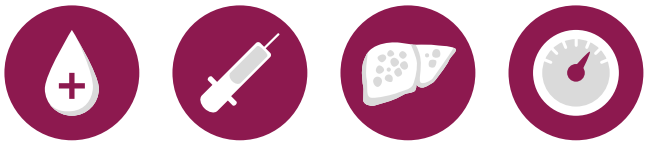
Gout

Strong evidence for weight gain and dental health

The WHO guidelines to reduce free sugars in the diet are based on evidence showing that adults who consume less free sugar have a lower body weight, and that increasing the amount of free sugars in the diet is associated with a comparable weight increase.[45]

A link between consumption of added sugars in sugar-sweetened beverages and body weight gain in adults and children has been reported, and these studies have also informed the WHO guidelines.[46] Furthermore, there are a large number of studies showing that beverages in any form are not compensated for by reduced food intake – hence, they add excessive calories to the overall diet and promote weight gain.[47 48 49]

Consistent evidence is also found for dental health, where it has been shown that tooth decay increases with sugar consumption.[50 51 52 53 54 55] A review of international evidence that contributed to the WHO guidelines on sugar consumption shows that high-sugar diets are associated with a higher dental caries risk.[56] Studies also support that intake of free sugars below the WHO recommendations is associated with a lower risk for dental caries.[57]



Sugar and other non-communicable diseases

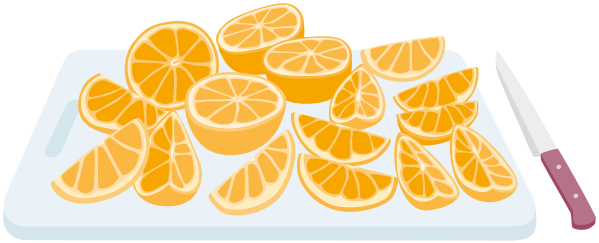
Evidence shows that people who consume high amounts of sugary foods or drinks are more likely to have high levels of fat in the blood,[58 59] insulin resistance,[60 61] fatty liver,[62 63 64] metabolic syndrome,[65 66 67 68] risk of type-2 diabetes and heart disease, [69 70 71 72 73 74 75 76 77 78 79 80] and abdominal fat gain[81 82] than people who do not consume much sugar at all. In some studies, this association continues to hold true even after accounting for the fact that people who consume more sugar may also have higher body weight and energy intake (which are risk factors for the above mentioned diseases).[83 84 85 86 87]

Because of their high amounts of added sucrose, and the often large quantities consumed on single occasions, sugar-sweetened beverages in particular have been suggested as increasing the risk of chronic disease, independently of obesity.[88 89 90]

Some population studies have claimed that our increased consumption of added sugars per se is a factor driving the obesity and diabetes pandemic.[91 92 93] However, one limitation with this type of study which is using data on very large groups and not on individuals, is that the outcome (diabetes) is measured on population level, and so is the exposure (added sugars consumption). Because the outcome and the exposure data are not linked to the same individuals, these studies can only show general associations, and they are identifying only one component of what is likely to be a multi-factorial problem.

A comprehensive review of different dietary intervention trials compared the effects of high and low consumption of added sugars on risk factors for cardiovascular disease, where the only difference was the amount of sugars and non-sugar carbohydrates that the participants had consumed.[94] The review found that sugars seem to contribute to risk of heart disease, independent of the effect that sugar has on body weight.

Studies in rodents have provided some insights into the underlying mechanisms involved, including reports that high levels of sugar intake are linked with arrhythmias, heart muscle cell death and disturbed metabolism in heart cells.[95 96 97] These conditions appeared even in the absence of changes in body weight or diabetic blood glucose levels, suggesting that excess sugar consumption may be dangerous for the heart even when someone is slim and seemingly healthy. These 'cause-and-effect' data are preliminary and require validation in human tissues.



Is fructose more threatening to health than glucose?

There are a number of studies proposing that, compared to glucose, fructose may have particularly adverse effects, leading to fat accumulation, atherosclerosis, raised blood pressure, appetite disruption, and insulin sensitivity.[98] [99]

The adverse health effects of fructose would not simply be due to the excess energy or total sugar intake – it is suggested that fructose could have an independent effect on health because of how the fructose is affecting and disrupting the metabolic process, but more research is required to establish the exact mechanisms. [100] [101] [102] [103] [104]

If very high doses of fructose are consumed, fat will accumulate in the liver, and glucose will not be metabolised as efficiently anymore.[105] This could lead to insulin resistance (impaired glucose uptake) with increased glucose levels in the blood to follow. This in turn could lead to excessive insulin release or what is called hyperinsulinemia,[106] [107] increased levels of fats in the blood that can lead to heart disease, and increased uric acid levels or hyperuricaemia that in turn can lead to gout.[108] [109] [110]

The latter condition may have particular relevance to New Zealand, as Pacific Island populations (particularly those of Polynesian descent) have showed a high prevalence of hyperuricaemia and gout.[111] This could be due to an inherently higher uric acid level among these populations, with a demonstrated genetic predisposition.[112] Fructose has been shown to increase uric acid levels, so the suggested chain of events is that the fructose causes heightened uric acid levels – of already (inherently) high levels – which can then lead to gout.[113]

There is an emerging body of evidence showing that very high fructose doses (more than 100 g/day, which you would have to eat 200 g of sucrose to achieve, or four times the WHO recommended level)** may promote weight gain and fatty liver disease, which are known risk factors for diabetes.[114] [115] [116] However, whether this occurs with low to moderate intakes is still debated.

The few studies that have investigated low and moderate levels of fructose consumption in humans have not been able to identify consistent adverse effects from fructose, compared to other sugars.[117] [118] [119] [120] [121] So – whether fructose has a particularly significant negative impact on health compared to glucose, and whether it can cause direct negative health effects independently, remains an active area of research.[122]

** To consume 100 g fructose, you would need to consume about 200 g of sucrose (which is made up by 50% fructose and 50% glucose). To achieve a total of 100 g fructose you would need to consume the equivalent of about 2 L of a sugary soft drink, or 3 bottles of a sports energy drink, or 12 pieces of whole fruit.

Where are the gaps in the research on sugar and health?

More research is needed to understand the exact mechanisms linking sugars to poor health outcomes, compared with other macronutrients – fats, proteins and other carbohydrates – in our diet, and there is especially a need for intervention studies that can provide high-grade evidence.

WHO has outlined some gaps where more research is needed in order to revise the WHO sugar consumption recommendations in 2020:

- How the intake of free sugars affects metabolism.
- Longer-term studies showing how changes in free sugars intake affect health.
- Thresholds above which the consumption of free sugars increases weight gain.
- The effectiveness of behavioural changes in reducing the intake of free sugars.

Our experts

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References

1. Popkin, BM and Hawkes, C. 2015. Sweetening of the global diet, particularly beverages: patterns, trends, and policy responses. The Lancet Diabetes & Endocrinology 4:2, pp 174–86
2. OECD/Food and Agriculture Organization of the United Nations 2015. Sugar projections – consumption per capita. OECD-FAO Agricultural Outlook 2015, OECD Publishing, Paris
3. Butler CD, Dixon J, Capon AG (Eds.). 2015. Health of people, places, and planet. Canberra: Australian National University Press
4. Ministry of Health. 2015. New Zealand Health Survey. Annual update of key findings 2014/15. Wellington: Ministry of Health
5. World Health Organization (WHO). 2012. Global burden of disease estimates. Geneva: WHO
6. Coppell KJ, Mann JI, Williams SM et al. 2013. Prevalence of diagnosed and undiagnosed diabetes and pre-diabetes in New Zealand: findings from the 2008/09 adult nutrition survey. New Zealand Medical Journal. 1:126 (1370) pp 23–42
7. Karter AJ, Schillinger D, Adams AS et al. 2012. Elevated rates of diabetes in Pacific Islanders and Asian subgroups. Diabetes Care. Oct 36:3, pp 574–9
8. Lal A, Moodie M, Ashton T et al. 2012. Health care and lost productivity costs of overweight and obesity in New Zealand. Australian and New Zealand Journal of Public Health. 36:6, pp 550–6
9. American Diabetes Association. 2016. The cost of diabetes. http://www.diabetes.org/advocacy/news-events/cost-of-diabetes.html (accessed 22 June 2016)
10. The Guardian. 2016. The Sugar Conspiracy. Guardian. 7 April 2016
11. Washington Post. 2013. Is sugar a toxin? Experts debate the role of fructose in our obesity epidemic. Washington Post. 1 September 2013
12. Stanhope, KL. 2016. Sugar consumption, metabolic disease and obesity: The state of the controversy, Critical Reviews in Clinical Laboratory Sciences. 53:1, pp 52–67
13. Bray GA, Popkin BM. 2014. Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes? Health be damned! Pour on the sugar. Diabetes Care. 37, pp 950–956
14. Kahn R, Sievenpiper JL. 2014. Dietary sugar and body weight: have we reached a crisis in the epidemic of obesity and diabetes? We have, but the pox on sugar is overwrought and overworked. Diabetes Care. 37, pp 957–962
15. Mann J, Cummings J. 2012. Carbohydrates. In: Mann, J. and Trustwell, S. Essentials of Human Nutrition, 4th ed. Oxford: Oxford University Press
16. Cummings, HJ, Stephen, AM. 2007. Carbohydrate terminology and classification. European Journal of Clinical Nutrition. 61, pp 15–18
17. World Health Organization (WHO). 2015. Sugars intake for adults and children. Geneva: WHO
18. Raben A, Hermansen L. 2006. Health aspects of mono- and disaccharides. In: Eliasson, Carbohydrates in food. New York: Taylor and Francis
19. Te Morenga L, Mallard S, Mann J. 2012. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. British Medical Journal 15:346, p 7492
20. Anderson GH, Woodend D. 2003. Effect of glycemic carbohydrates on short-term satiety and food intake. Nutrition Reviews 61, pp 17–26
21. Augustin LSA, Kendall CWC, Jenkins DJA et al. 2015. Glycemic index, glycemic load and glycemic response: An international scientific consensus summit from the international carbohydrate quality consortium (ICQC). Nutrition, Metabolism & Cardiovascular diseases. 25:795–815
22. Pan A, Hu FB. 2011. Effects of carbohydrates on satiety: differences between liquid and solid food. Current Opinion in Clinical and Nutritional Metabolic Care. 14:4, pp 385–390
23. Brand-Miller JC, Holt SHA, Pawlak DB, McMillan J. 2002. Glycemic index and obesity. The American Journal of Clinical Nutrition. 76:281S–285S
24. Raben A, Hermansen L. 2006. Health aspects of mono- and disaccharides. In: Eliasson, Carbohydrates in food. New York: Taylor and Francis
25. Food Standards Australia New Zealand. 2016. Food Standards Code, revised version. 1 March 2016. URL: http://www.foodstandards.gov.au/consumer/labelling/Pages/default.aspx. Accessed on 29 August 2016
26. Nishida C, Nocito FM, Mann J. 2007. Joint FAO/WHO Scientific Update on Carbohydrates in Human Nutrition. European Journal of Clinical Nutrition. 61, Suppl 1, pp 1–137
27. Ministry of Health, University of Otago. 2011. A focus on nutrition: Key findings of the 2008/09 New Zealand adult nutrition survey. Wellington: Ministry of Health
28. Popkin, BM, Hawkes, C. 2016. Sweetening of the global diet, particularly beverages: patterns, trends, and policy responses. Lancet Diabetes Endocrinology. 4, 174–186
29. United States Department of Agriculture Food Composition Database. 2016. https://ndb.nal.usda.gov/. Accessed on 29 August 2016
30. World Health Organization. 2015. Sugars intake for adults and children. Geneva: WHO
31. Eyles H, Neal B, Jiang Y, Ni Mhurchu C. 2016.Estimating population food and nutrient exposure:comparison of store survey data with household panel food purchases. British Journal for Nutrition. May 28:115(10):1835–42
32. Ni Mhurchu C, Eyles H. 2014. Sweetened and unsweetened non-alcoholic beverages in New Zealand: assessment of relative availability, price, serve size, and sugar content. Pacific Health Dialog. 20:1
33. Food Standards Australia New Zealand. 2016. Food Standards Code, revised version. 1 March 2016. https://www.legislation.gov.au/Details/F2015L00392
34. World Health Organization. 2015. Sugars intake for adults and children. Geneva: WHO
35. Food and Drug Administration 27 May 2015. https://federalregister.gov/a/2016-11867
36. Bronk JR. 1999. Human Metabolism. Harlow, England: Addison Wesley Longman Ltd
37. Sun SZ, Empie MW. 2012. Fructose Metabolism in Humans. Nutrition and Metabolism. 9:89
38. Mann J, Cummings J. 2012. Carbohydrates. In: Mann, J. and Trustwell, S. Essentials of Human Nutrition, 4th ed. Oxford: Oxford University Press
39. Raben A, Hermansen L. 2006. Health aspects of mono- and disaccharides. In: Eliasson 2006. Carbohydrates in food. New York: Taylor and Francis
40. Saris, WH. 2003. Sugars, energy metabolism, and body weight control. American Journal of Clinical Nutrition. Oct 78:4, pp 850S–857S
41. Wolever TMS. 2006. Mechanisms by which different carbohydrates elicit different glycaemic responses. In: The glycaemic index: A physiological classification. Cambridge: CABI
42. Mayes PA. 1993. Intermediary metabolism of fructose. American Journal of Clinical Nutrition. Nov 58 (5 Suppl), pp 754S–765S

43. Tappy L, Lê KA. 2015. Health effects of fructose and fructose-containing caloric sweeteners: where do we stand 10 years after the initial whistle-blowings? Current Diabetes Reports 15:8, p 54
44. Laughlin MR. 2014. Normal Roles for Dietary Fructose in Carbohydrate Metabolism. Nutrients. 6:8, pp 3117–3129.
45. World Health Organization. 2015. Sugars intake for adults and children. Geneva: WHO
46. Ebbeling CB et al. 2012. A randomized trial of sugar-sweetened beverages and adolescent body weight. New England Journal of Medicine. 367:15, pp 1407–1416
47. DellaValle, DM, Roe LS, Rolls BJ. 2005. Does the consumption of caloric and non-caloric beverages with a meal affect energy intake? Appetite. 44:2, pp 187–193
48. Flood J, Roe LS, Rolls BJ. 2006. The effect of increased beverage portion size on energy intake at a meal. Journal of the American Dietetic Association. 106:12, pp 1984–1990
49. Mourao DM, Bressan J, Campbell WW et al. 2007. Effects of food form on appetite and energy intake in lean and obese young adults. International Journal of Obesity (London). 31:11, pp 1688–1695
50. Sheiham A. 2001. Dietary effects on dental diseases. Public Health Nutrition. 4:2b, pp 569–591
51. Burt BA, Pai S. 2001. Sugar consumption and caries risk: a systematic review. Journal of Dental Education 65:10, pp 1017–1023
52. Moynihan PJ, Kelly SAM. 2014. Effect on caries of restricting sugars intake. Systematic review to inform WHO guidelines. Journal of Dental Research. 1, pp 8–18
53. Gussy MG, Waters EG, Walsh O et al. 2006. Early childhood caries: Current evidence for aetiology and prevention. Journal of Paediatrics and Child Health. 42:1–2, pp 37–43
54. Meyer BD, Lee JY. 2015. The confluence of sugar, dental caries, and health policy. Journal of Dental Research. 94:10, pp 1338–40
55. Dhurandhar NV, Thomas D. 2015. The link between dietary sugar intake and cardiovascular disease mortality – an unresolved question. JAMA. 313:9, pp 959–960
56. World Health Organization. 2015. Sugars intake for adults and children. Geneva: WHO
57. Moynihan P, Peterson PE. 2004. Diet, nutrition and the prevention of dental diseases. Public Health Nutrition. 7:1A, pp 201–226
58. Duffey KJ, Gordon-Larsen P, Steffen LM et al. 2010. Drinking caloric beverages increases the risk of adverse cardiometabolic outcomes in the Coronary Artery Risk Development in Young Adults (CARDIA) Study. The American Journal of Clinical Nutrition 92:954–959
59. Welsh JA, Sharma A, Cunningham SA et al. 2011. Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. Circulation. 123:249–57
60. Bremer AA, Auinger P, Byrd RS. 2010. Sugar-sweetened beverage intake trends in US adolescents and their association with insulin resistance-related parameters. Journal of Nutrition and Metabolism. Article ID: 196476
61. Yoshida M, McKeown NM, Rogers G et al. 2007. Surrogate markers of insulin resistance are associated with consumption of sugar-sweetened drinks and fruit juice in middle and older-aged adults. Journal of Nutrition. 137, pp 2121–2127
62. Assy N, Nasser G, Kamayse I et al. 2008. Soft drink consumption linked with fatty liver in the absence of traditional risk factors. Canadian Journal of Gastroenterology. 22, pp 811–816
63. Ouyang X, Cirillo P, Sautin Y et al. 2008. Fructose consumption as a risk factor for non-alcoholic fatty liver disease. Journal of Hepatology. 48, pp 993–999
64. Stanhope KL. 2012. Role of fructose-containing sugars in the epidemics of obesity and metabolic syndrome. Annual Review of Medicine. 63, pp 329–343
65. Lustig RH, Mulligan K, Noworolski S et al. 2016. Isocaloric fructose restriction and metabolic improvement in children with obesity and metabolic syndrome. Obesity. 24:453–460
66. Chan TF, Lin WT, Huang HL et al. 2014. Consumption of sugar-sweetened beverages is associated with components of the metabolic syndrome in adolescents. Nutrients. 6:2088–2103
67. Dhingra R, Sullivan L, Jacques PF et al. 2007. Soft drink consumption and risk of developing cardiometabolic risk factors and the metabolic syndrome in middle-aged adults in the community. Circulation. 116:480–488
68. Rodríguez LA, Madsen KA, Cotterman C, Lustig RH. 2016. Added sugar intake and metabolic syndrome in US adolescents: cross-sectional analysis of the National Health and Nutrition Examination Survey 2005–2012. Public Health and Nutrition. 2, pp 1–11
69. New Zealand Nutrition Foundation. 2014. The role of sugars in the diet of New Zealanders. Auckland 2014
70. Heart Foundation of New Zealand. 2013. Sugar and the heart – a snapshot of the evidence in relation to sugar and heart health. Auckland 2013
71. Public Health England. 2015. Sugar reduction – the evidence for action. London: Public Health England. PHE publications gateway number 2015391
72. Fazeli Farsani S, van der Aa MP, van der Vorst MM. 2013. Consumption of sweet beverages and type 2 diabetes incidence in European adults: results from EPIC-InterAct. 2013. Diabetologia. 56:7, pp 1520–1530
73. Australian Dietary Guidelines. National Health and Medical Research Council 2013. Canberra: National Health and Medical Research Council
74. Malik VS, Popkin BM, Bray GA et al. 2010. Sugar-sweetened beverages, obesity, type 2 diabetes, and cardiovascular disease risk. Circulation. 121, pp 1356–1364
75. de Koning L, Malik VS, Rimm EB et al. 2011. Sugar-sweetened and artificially sweetened beverage consumption and risk of type 2 diabetes in men. American Journal of Clinical Nutrition. 93:1321–1327
76. Montonen J, Jarvinen R, Knekt P et al. 2007. Consumption of sweetened beverages and intakes of fructose and glucose predict type 2 diabetes occurrence. Journal of Nutrition. 137, pp 1447–1454
77. Yang Q, Zhang Z, Gregg EW et al. 2014. Added sugar intake and cardiovascular diseases mortality among US adults. Journal of the American Medical Association Internal Medicine. 174:516–524
78. Imamura F, O'Connor L, Ye Z et al. 2015. Consumption of sugar sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: systematic review, meta-analysis, and estimation of population attributable fraction. BMJ. 351: h3576
79. Malik, VS, Hu FB. 2015. Fructose and Cardiometabolic Health: What the Evidence From Sugar-Sweetened Beverages Tells Us. Journal of the American College of Cardiology. 66:14, pp 1615–1624
80. Johnson RJ, Nakagawa T, Sanchez-Lozada TG et al. 2013. Sugar, Uric Acid, and the Etiology of Diabetes and Obesity. Diabetes. 62:10, pp 3307–3315
81. Odegaard AO, Choh AC, Czerwinski SA et al. 2012. Sugar-sweetened and diet beverages in relation to visceral adipose tissue. In: Obesity. Silver Spring 2012, 20:3, pp 689–91
82. Pollock NK, Bundy V, Kanto W et al. 2012. Greater fructose consumption is associated with cardiometabolic risk markers and visceral adiposity in adolescents. Journal of Nutrition. 142, pp 251–257
83. de Koning L, Malik VS, Kellogg MD et al. 2012. Sweetened beverage consumption, incident coronary heart disease, and biomarkers of risk in men. Circulation. 125:1735–1741, S1
84. Malik VS, Popkin BM, Bray GA et al. 2010. Sugar-sweetened beverages, obesity, type 2 diabetes mellitus, and cardiovascular disease risk. Circulation. 121:1356–1364
85. Malik VS, Popkin BM, Bray GA et al. 2010. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes: a meta-analysis. Diabetes Care. 33:2477–2483

86. Stanhope KL, Medici V, Bremer AA et al. 2011. A dose-response study of consuming high-fructose corn syrup-sweetened beverages on lipid/lipoprotein risk factors for cardiovascular disease in young adults. American Journal of Clinical Nutrition. 101:6, pp 1144–1154

87. Yang Q, Zhang Z, Gregg EW, et al. 2014. Added sugar intake and cardiovascular diseases mortality among US adults. JAMA Internal Medicine. 174:4, pp 516–524

88. Malik V, Popkin BM, Bray GA et al. 2010. Sugar-sweetened beverages and risk of metabolic syndrome and type 2 diabetes. Diabetes Care. 33:11, pp 2477–83

89. Malik VS, Hu FB. 2015. Fructose and cardiometabolic health: What the evidence from sugar-sweetened beverages tells us. Journal of the American College of Cardiology. 66:14, pp 1615–1624

90. Stanhope, KL. 2016. Sugar consumption, metabolic disease and obesity: The state of the controversy. Critical Reviews in Clinical Laboratory Sciences. 53:1, pp 52–67

91. Basu S, Yoffe P, Hills N et al. 2013. The relationship of sugar to population-level diabetes prevalence: an econometric analysis of repeated cross-sectional data. PLoS One. 8:2, e57873

92. Yang Q, Zhang Z, Gregg EW et al. 2014. Added sugar intake and cardiovascular diseases mortality among US adults. JAMA Internal Medicine 174:516–524

93. Popkin BM, Adair LS, Ng SW. 2012. The global nutrition transition: the pandemic of obesity in developing countries. Nutritional Reviews. 2012. 70:1, pp 3–21

94. Te Morenga L, Howatson, Jones et al. 2014. Dietary sugars and cardiometabolic risk: systematic review and meta-analyses of randomized controlled trials of the effects on blood pressure and lipids. American Journal of Clinical Nutrition. 100:1, pp 65–79

95. Mellor KM, Bell JR, Young MJ et al. 2011. Myocardial autophagy activation and suppressed survival signaling is associated with insulin resistance in fructose-fed mice. Journal of Molecular and Cellular Cardiology. 50:1035–1043

96. Mellor K, Ritchie RH, Meredith G et al. 2010. High-fructose diet elevates myocardial superoxide generation in mice in the absence of cardiac hypertrophy. Nutrition. 26:842–848

97. Sommese L, Valverde CA, Blanco P et al. 2016. Ryanodine receptor phosphorylation by CaMKII promotes spontaneous Ca(2+) release events in a rodent model of early stage diabetes: The arrhythmogenic substrate. International Journal of Cardiology. 202, pp 394–406

98. Malik VS, Popkin BM, Bray GA et al. 2010. Sugar-sweetened beverages, obesity, type 2 diabetes, and cardiovascular disease risk. Circulation. 121:1356–1364

99. Johnson RJ, Nakagawa T, Sanchez-Lozada LG et al. 2013. Sugar, Uric Acid, and the Etiology of Diabetes and Obesity. Diabetes. 62:10, pp 3307–3315

100. Gugliucci A, Lustig RH, Caccavello R et al. 2016. Short-term isocaloric fructose restriction lowers apoC-III levels and yields less atherogenic lipoprotein profiles in children with obesity and metabolic syndrome. Atherosclerosis. DOI: http://dx.doi.org/10.1016/j.atherosclerosis.2016.06.048

101. Basciano H, Federico L, Khosrow A. 2005. Fructose, insulin resistance, and metabolic dyslipidemia, Nutrition & Metabolism. DOI: 10.1186/1743-7075-2-5

102. Lustig RH. 2016. Sickeningly Sweet: Does Sugar Cause Type 2-Diabetes? Yes. Canadian Journal of Diabetes. 2016. August. 40:4, pp 282–6

103. Cox CL, Stanhope KL, Schwarz KM et al. 2012. Consumption of fructose-but not glucose-sweetened beverages for 10 weeks increases circulating concentrations of uric acid, retinol binding protein-4, and gamma-glutamyl transferase activity in overweight/obese humans. Nutrition & Metabolism. 9:68

104. Sievenpiper JL, de Souza RJ, Cozma AI et al. 2014. Fructose vs. glucose and metabolism: do the metabolic differences matter? Current Opinion in Lipidology. 25, pp 8–19

105. Crofts CAP, Zinn C, Wheldon MC et al. 2015. Hyperinsulinemia: A unifying theory of chronic disease? Diabesity. 1:4, pp 34–43

106. Zavaroni I, Bonini L, Gasparini P, Barilli A, Zuccarelli A, Dall'Aglio E, et al. 1999. Hyperinsulinemia in a normal population as a predictor of non-insulin-dependent diabetes mellitus, hypertension, and coronary heart disease: The Barilla factory revisited. Metabolism. 48:8, pp 989–994

107. Vuorinen-Markkola H, Koivisto VA, Yki-Jarvinen H. 1992. Mechanisms of hyperglycemia-induced insulin resistance in whole body and skeletal muscle of type I diabetic patients. Diabetes. 41:5, pp 571–580

108. Johnson RJ, Perez-Pozo SE, Sautin YY, Manitius J, Sanchez-Lozada LG, Feig DI et al. 2009. Hypothesis: could excessive fructose intake and uric acid cause type 2 diabetes? Endocrinological Review. 30:1, pp 96–116

109. Batt C, Phipps-Green AJ, Black MA et al. 2014. Sugar-sweetened beverage consumption: a risk factor for prevalent gout with SLC2A9 genotype-specific effects on serum urate and risk of gout. Annals of the Rheumatic Diseases. 73, pp 2101–2106

110. Merriman TR, Dalbeth N. 2011. The genetic basis of hyperuricaemia and gout. In: Joint Bone Spine, 2011, Elsevier

111. Batt C, Phipps-Green A, Black MA et al. 2014. Sugar-sweetened beverage consumption: A risk factor for prevalent gout with SLC2A9 genotype-specific effects on serum urate and risk of gout. Annals of the Rheumatic Diseases. 73:12, pp 2101–6

112. Merriman TR, Dalbeth N, Johnson RJ. 2014. Sugar-sweetened beverages, urate, gout and genetic interaction. Pacific Health Dialog, March 20:1

113. Choi HK, Curhan G. 2008. Soft drinks, fructose consumption, and the risk of gout in men: prospective cohort study. British Medical Journal. 336, pp 309–12

114. Sievenpiper JL, Carleton AJ, Chatha S et al. 2009. Heterogeneous effects of fructose on blood lipids in individuals with type 2 diabetes: systematic review and meta-analysis of experimental trials in humans. Diabetes Care 32:1930–37

115. Sievenpiper JL, de Souza RJ, Mirrahimi A et al. 2012. Effect of fructose on body weight in controlled feeding trials: a systematic review and meta-analysis. Annals of Internal Medicine. 156, pp 291–304

116. Livesey G, Taylor R. 2008. Fructose consumption and consequences for glycation, plasma triacylglycerol, and body weight: meta-analyses and meta-regression models of intervention studies. American Journal of Clinical Nutrition 88:5, pp 1419–1437

117. Sievenpiper JL, de Souza RJ, Beyene J. 2012. Fructose: where does the truth lie? The Journal of the American College of Nutrition. 31:149–51

118. Ha V, Jayalath VH, Cozma AI et al. 2013. Fructose-containing sugars, blood pressure, and cardiometabolic risk: a critical review. Current Hypertension Reports. Aug 15:4, pp 281–97

119. Kolderup A, Svihus B. 2015. Fructose metabolism and relation to atherosclerosis, type 2 diabetes, and obesity. Journal of Nutrition and Metabolism. Article ID: 823081

120. Sievenpiper JL, de Souza RJ, Kendall CW et al. 2011. Is fructose a story of mice but not men? Journal of the American Dietetic Association. 111:2, pp 219–20

121. Tappy L, Lê KA. 2010. Metabolic effects of fructose and the worldwide increase in obesity. Physiological Review. 90:1, pp 23–46

122. Stanhope, KL. 2016. Sugar consumption, metabolic disease and obesity: the state of the controversy, Critical Reviews in Clinical Laboratory Sciences. 53:1, pp 52–67

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