



Deposited via The University of Leeds.

White Rose Research Online URL for this paper:

<https://eprints.whiterose.ac.uk/id/eprint/102058/>

Version: Accepted Version

Article:

Moore, JB and Fielding, BA (2016) Sugar and metabolic health: Is there still a debate? Current Opinion in Clinical Nutrition and Metabolic Care, 19 (4). pp. 303-309. ISSN: 1363-1950

<https://doi.org/10.1097/MCO.0000000000000289>

© 2016, Wolters Kluwer Health, Inc. This is an author produced version of a paper published in Current Opinion in Clinical Nutrition and Metabolic Care. This is a non-final version of an article published in final form in "Moore, JB and Fielding, BA (2016) Sugar and metabolic health: Is there still a debate? Current Opinion in Clinical Nutrition and Metabolic Care, 19 (4). pp. 303-309. ISSN 1363-1950". Uploaded in accordance with the publisher's self-archiving policy.

Reuse

Items deposited in White Rose Research Online are protected by copyright, with all rights reserved unless indicated otherwise. They may be downloaded and/or printed for private study, or other acts as permitted by national copyright laws. The publisher or other rights holders may allow further reproduction and re-use of the full text version. This is indicated by the licence information on the White Rose Research Online record for the item.

Takedown

If you consider content in White Rose Research Online to be in breach of UK law, please notify us by emailing eprints@whiterose.ac.uk including the URL of the record and the reason for the withdrawal request.

TITLE PAGE

Title

Sugar and metabolic health: is there still a debate?

Authors

J. Bernadette Moore^{a,b} and Barbara A. Fielding^a

Affiliations

^aDepartment of Nutritional Sciences, University of Surrey, Guildford, Surrey, GU2 7XH and ^bSchool of Food Science & Nutrition, University of Leeds, Leeds, LS2 9JT, United Kingdom

Corresponding author

Dr. Barbara A. Fielding

Faculty of Health and Medical Sciences

The Leggett Bldg;

University of Surrey

Guildford, Surrey GU2 7XH

b.fielding@surrey.ac.uk

+44 (0) 1483 68 8649

STRUCTURED ABSTRACT

Purpose of review

There is considerable political and public awareness of new recommendations to reduce sugars and sugar-sweetened beverages in our diets. It is therefore timely to review the most recent changes in guidelines, with a focus on evidence for metabolic health, recent research in the area and gaps in our knowledge.

Recent findings

Sufficient evidence links a high intake of sugar to dental caries and obesity, and high intakes of sugar-sweetened beverages in particular to increased risk of type 2 diabetes. This has led to the updating of dietary recommendations related to added sugars in the diet. The effects of specific sugars at usual intakes as part of an isoenergetic diet are less clear. The glycaemic response to food is complex and mediated by many factors, but sugar intake is not necessarily the major component.

Summary

There are many challenges faced by healthcare professionals and government bodies in order to improve the health of individuals and nations through evidence-based diets. Sufficiently powered long-term mechanistic studies are still required to provide evidence for the effects of reducing dietary sugars on metabolic health. However, there are many challenges for research scientists in the implementation of these studies.

Keywords

sugar, sugar-sweetened beverages, fructose, obesity, type 2 diabetes

INTRODUCTION

Dietary sugars have recently been the focus of considerable attention from scientists, public health officials, popular media and the public alike. In the face of the worldwide obesity crisis, an extensive body of evidence now demonstrates that increased consumption of sugar is associated with weight gain; and furthermore that reducing dietary sugar intakes leads to weight loss^[1]. These data, in combination with the evidence that reducing dietary sugar intakes will reduce global prevalence of dental caries^[2], prompted the World Health Organization (WHO) to review, update and strengthen their earlier guidance^[3] that dietary intakes of added/free sugars be reduced to less than 10% of energy intakes^[4]. Although public health advice continues to vary depending on the advising body, both the United Kingdom (UK) and the United States (US) also published significantly strengthened dietary guidelines for the consumption of added/free sugars in 2015 (Table 1). Whereas the US guidelines echoed the WHO in recommending that added sugars should be limited to no more than 10% of energy intakes^[6], the UK advice goes further in recommending the intake of free sugars should not exceed 5% of total dietary energy^[5]. While the trend for sugar consumption in the US did fall during 2005-2010, 71.4 % adults consumed 10% or more of calories from added sugar^[9]. The average free sugar consumption in the UK is also well over 10% in all age and gender categories, although there is a wide range of variation (Figure 1)^[10].

The consensus of both the WHO commissioned meta-analysis^[1] and the UK's Scientific Advisory Committee on Nutrition (SACN) report on Carbohydrates on health^[5], which contained the results of independently executed meta-analyses, is that the relationship between a high sugar diet and obesity is clear. The evidence for links between a high-sugar diet and obesity has recently been summarised by Jebb^[11] who also set out various population-level options that are available to reduce the intake of free sugars. Since obesity is a leading risk factor for cardio-metabolic disease, adherence to current guidelines, as set out in Table 1, should be beneficial in this respect. Equally, the evidence base for a causal relationship between dietary intakes of sugar-sweetened beverages specifically and type 2 diabetes is now unquestionable with the SACN meta-analysis concluding that 'prospective cohort studies indicate that greater consumption of sugars-sweetened beverages is associated with increased risk of type 2 diabetes mellitus'. However, what is less

clear are the metabolic effects of sugars outside the 'sugar-sweetened beverages' category in the context of an isoenergetic diet. This has led to some degree of controversy and uncertainty about the effects of individual dietary sugars, e.g. fructose, on metabolic health. A number of plausible mechanisms have been elucidated linking **high** sugar intakes with metabolic dysfunction, but evidence of adverse effects of sugars independent of associated body weight gain is still lacking. Human studies are expensive and difficult to perform in a free-living population. In this context, here we review some of the more recent, pertinent studies in the literature that have aimed to investigate either the metabolic effects of sugars per se, or the impact of sugar intakes on metabolic risk factors in humans. In addition, we discuss some of the challenges in translating evidence-based dietary recommendations to changes in consumer eating patterns.

Dietary sugars, energy intake and fatty liver

A recent study reported on two dietary interventions in which free-living individuals were provided with food that provided an excess of energy and were asked to consume the food ad libitum, and uneaten items were returned^[12]. The participants were also asked to consume sugar-sweetened test beverages (compulsory). The authors found that both glucose and fructose consumed in liquid form (mean energy intake per day 515 ± 92 and 509 ± 92 kcal) promoted excess energy intake during the study period compared with an aspartame control drink because the participants did not compensate with a decrease in energy from the solid foods. Another part of the work in older, more obese individuals compared fructose, glucose and high fructose corn syrup (HFCS) containing drinks in a similar fashion and found no significant difference in energy intake between the groups. The sugar consumed in the beverages equated to the sugar in approximately 1.25 L regular cola per day. The individuals did not gain weight in this short-term study but the results are in accord with the notion that glucose, fructose and HFCS are likely to be equally obesogenic when consumed in sugar-sweetened beverages.

Obesity is a risk factor for non-alcoholic fatty liver disease (NAFLD) and we have previously concluded that... 'whether or not dietary sugars, including fructose, at typically consumed population levels, effect hepatic lipogenesis and NAFLD pathogenesis in humans independently of excess energy remains unresolved'. The issue is still largely unresolved but an in-patient study by

Schwarz and colleagues^[13] attempted to address this in a short-term, 9-day, study in which the effect of a high-fructose diet (25% of total energy) was compared isoenergetically with an equivalent amount of complex carbohydrate in eight healthy men. The study found that liver fat and *de novo* lipogenesis were significantly higher after the high fructose intervention, notably in the absence of weight gain. To put the amount of fructose consumed into perspective however, this equates to the highest (95th percentile) reported consumption of total fructose in the US, in young men aged 19-22y (134 g = 536 kcal per day), but was supplied entirely as an experimental sugar-sweetened beverage.

Separately, a secondary analysis of a European study examined uric acid levels in overweight/obese individuals randomised to consume 1L of either a sucrose-sweetened soft drink (SSSD; coca-cola from Denmark), semi-skimmed milk, diet cola or water for 6 months^[14]. Uric acid levels increased significantly only in the SSSD group (0.31 ± 0.02 vs. 0.35 ± 0.03 nmol/l, $P=0.02$), and this increase correlated with the previously reported^[15] increases in liver fat ($r=0.44$) and plasma triglycerides ($r=0.34$). At a population level, Ma and colleagues^[16] examined the associations between sugar-sweetened beverage consumption in cohorts from the Framingham Study and: fatty liver (defined by computed tomography with a liver to phantom ratio cut off < 30) in 2634 participants; plus elevated serum alanine transaminase (ALT; >19U/L women, >30U/L for men) levels, in 5908 participants. Daily consumption of sugar-sweetened beverages, as assessed by food frequency questionnaire, was associated with odds ratios of 1.6 for fatty liver and 1.3 for elevated ALT levels. Interestingly, in multi-variate analyses, while these associations remained significant when BMI was adjusted for, they disappeared when visceral adipose tissue was incorporated into the model.

Dietary Sugars and Metabolic Health

One of the most tangible effects of dietary glucose on the body is the postprandial rise in blood glucose concentration and the glycaemic index is commonly used as a measure of the blood glucose-raising potential of food^[5]. However, a recent report has quantified the remarkably high, between-person variability in post-meal blood glucose responses in response to both specific foods and mixed meals^[17], which serves to highlight the significant limitations of these measures.

In this impressive study, 800 people without diabetes were monitored by continuous glucose monitoring for 7 days, while food intake and other lifestyle factors were recorded. While replicate measurements of the post-prandial glucose response (PPGR) to the same food were reproducible within the same individual, the study demonstrated tremendous interpersonal variability with low and high responders noted to both standardised and real-life meals. Using a machine learning approach, the authors developed and independently validated a prediction algorithm based on 137 different features related to person-specific factors such as gut microbiome, anthropometric data, physical activity and blood lipids. The algorithm-predicted PPGRs correlated much better with the measured PPGRs as opposed to the current “carbohydrate counting” model ($R=0.71$ vs. 0.38) or caloric counting ($R=0.33$).

The National Health and Nutrition Examination Survey (NHANES) provided evidence from cross-sectional data in the US regarding the effect of added sugars on risk of CVD mortality^[7]. Twenty-four hour dietary recalls from 11 733 adults were used to estimate dietary intake of added sugar, which included sugar-sweetened beverages and all sugars in processed or prepared foods, but not naturally occurring sugar, such as in fruits and fruit juices. The study found that, ‘compared with those who consumed approximately 8.0% of calories from added sugar, participants who consumed approximately 17% to 21% of calories from added sugar had a 38% higher risk of CVD mortality’. This did not seem to be related to a generally unhealthy diet. Interestingly, there appeared to be no appreciable difference in adjusted hazard ratio between 5 and 10 % calories from added sugars. Another point to consider is that the risk is relative and many other important risk factors for CVD have, by necessity, been corrected for in the analysis. This study was included in a recent review that also focussed the relationship between sugar-sweetened beverages and CVD^[18].

To a certain extent, the deleterious metabolic effects of dietary sugars can be mitigated by exercise and previously, Tappy and colleagues showed that an acute, very high-fructose diet (30 % energy) that leads to hypertriglyceridaemia can be normalised with moderate aerobic exercise^[19]. The energy intake was balanced to account for the extra energy expenditure. This work has been extended in a new study^[20] where participants consumed a high-fructose diet before participating in one of 3 metabolic studies in random order. A high fructose drink was given

either before or after an exercise load or in combination with no exercise. It was found that moderate exercise (1h cycling at 100 W), only when performed after fructose ingestion was beneficial in leading to higher fructose oxidation (80 % of ingested fructose) and less storage of metabolites. It might be thought that the choice of a pure fructose drink would compromise the postprandial response to a certain extent because of the low insulin-potential of fructose. However, because of conversion of fructose to glucose (calculated to be about 15 % of the fructose load), there were small increases in plasma glucose and insulin, and a robust decrease in plasma non-esterified fatty acid concentrations. The latter occurring presumably because adipose tissue is sensitive to small changes in plasma insulin concentrations in healthy people.

Sugar-sweetened beverages and metabolic health

In separate work, a dose response study was carried out in healthy young men and women to examine cardiovascular disease (CVD) risk factors in response to 2 weeks consumption of HFCS (55% fructose, 45% glucose) drinks at 0% (aspartame sweetened), 10%, 17.5% or 25% of energy intake, and postprandial responses to the study food over a 24 h period^[21]. The 10% HFCS equates to the sugar content of approximately 550 mL regular cola. Risk factors for CVD including LDL-cholesterol, uric acid and postprandial but not fasting triglyceride concentrations increased in response to the sugar sweetened beverages, statistically independent of body weight^[22]. More evidence is needed concerning the metabolic effects of a longer term diet (using high-sugar foods regularly consumed on a Western diet), and a recent trial may help to answer this ^[23].

A recent systematic review and meta-analysis is in accord with the previous body of evidence linking consumption of sugar-sweetened beverages with the risk of type 2 diabetes, but perhaps goes further in significantly demonstrating an association that is independent of adiposity^[24]. In this work, the current consumption of sugar-sweetened beverages was estimated to cause approximately two million excess events of type 2 diabetes in the US and 80,000 in the UK over 10 years. This could cost nearly £12.0bn in the US and £206m in the UK. Conversely, independent modelling exercises have demonstrated that reducing free sugars (40% over 5 years) added to sugar-sweetened beverages, presumably through reformulation, could prevent a significant number (~300,000 over 20 years) of incident cases of type 2 diabetes^[25]. In a similar

vein of modelling the benefit to a reduction in sugar consumption, a very recent report from a public health policy agency in the UK has modelled the effect of a 20% sugar tax in the UK. The analysis concluded this would lead to a reduction of 18,000 incident cases of diabetes over 10 years and reduction of ~25,500 cases of obesity-related disease in total^[26]. These data are in line with previous work that showed a 20% sugar tax would reduce the number of obese adults in the UK by 1.3% (180,000 people) and the number who are overweight by 0.9% (285,000 people)^[27]. Similar modelling exercises recently published for the US also estimate that a tax on sugar-sweetened beverages would reduce consumption by 20%, with a concomitant reduction in mean BMI by 0.16 and significant financial savings in the cost of healthcare over ten years (\$23.6 billion)^[28]. While taxes on sugar-sweetened beverages have been adopted in several countries and have been demonstrated to be both acceptable to the public^[29] and, importantly, to reduce consumption^[30]; the effect of a sugar tax on obesity and health remains to be proven^[31] and even advocates argue that this would be but a small step in the right direction^[32].

Hidden dietary sugars and the challenge for Public Health

One of the challenges for Nutritional Scientists in communicating to an often confused public^[33], in the face of changing guidelines, is the terminology used to describe dietary sugars (Table 2); and the adoption of the WHO term 'free sugars' by the UK, replacing the previous 'non-milk extrinsic sugars', is considered beneficial^[36]. The terms 'free sugars'^[4,5] and 'added sugars'^[6-8] are essentially equivalent in describing sugars (mono- and disaccharides) added to foods and beverages by the manufacturer, cook or consumer, and sugars found in honey, syrups, fruit juices and juice concentrates (Table 2). Sugars naturally present in whole fruit or milk (lactose) are excluded. However, while these definitions are unambiguous, we count at least 25 different examples of sugars that may be listed as ingredients (Table 2) and these can be difficult for even the most conscientious of consumers to recognize^[37].

Syrups are particularly confusing, the demonizing of fructose in the form of HFCS consumption in the US^[38], may lead the European public to perhaps believe they are safer. Restrictions on trade mean that sugar sweetened beverages in the European Union are largely sweetened by sucrose and commonly used syrups in baked goods ('glucose-fructose syrup' or

'fructose-glucose syrup'; Table 2) are largely derived from beet or cane sugar^[36]. Furthermore there is a perception that 'natural' sugars and syrups are more healthful and/or less processed. Agave syrup in particular is marketed as a natural sweetener, but is processed in a fashion almost identical to HFCS and contains approximately 84% fructose^[35]. Although antioxidants are detectable in some alternatives to white sugar these are largely found in amounts much smaller than in whole food such as blueberries or walnuts^[39]. While honey and maple syrups do contain a myriad of compounds in addition to glucose and fructose, these vary enormously depending on where and how they were harvested and processed^[40,41], and any health benefits of these have yet to be established. By far and large the dominant compounds are mono- and disaccharides contributing to their significant energy density (Table 3).

The dialogue regarding the role of dietary sugars on human health and disease has reached a fervent pitch in recent years, not least regarding the contentious issue of a 'sugar tax'. Indeed, the subjects of either reformulation or sugar tax as policy interventions to reduce obesity and the burden of obesity-related metabolic disease illicit fairly polarised discussions. However, as highlighted by both the McKinsey Global Institute in their economic analysis of interventions to reduce obesity^[44], and Public Health England's assessment of the evidence-based actions to reduce sugar consumption^[43], these are only two of many possible interventions required. We would concur with the conclusions that any single intervention is likely to have only a small overall impact on its own and a systemic, sustained group of initiatives, delivered at a population level, is needed to both reduce sugar consumption and address the health burden associated with obesity.

CONCLUSION

Lowering dietary sugar could be a way to reverse current alarming trends in obesity prevalence and metabolic disease. New guidelines that suggest reducing added/free sugars in the diet to 5-10% total energy could help to reverse this trend but will be difficult to achieve for many individuals. Challenges include consumer misconceptions about types of added sugars that can be found as ingredients and the obfuscation of sugars on package labels. The best measures to realize the reduced intake of dietary sugars at a population level is a matter of ongoing debate.

KEY POINTS

- There is a causal link between a high-sugar diet and obesity
- Sugar-sweetened beverage consumption is linked to the development of type 2 diabetes
- A very high-sugar diet is associated with an increase in CVD mortality
- The effect of specific sugars as part of an isocaloric diet on metabolism diet is not clear
- The glycemic response to carbohydrate in free-living individuals is dictated by many more factors than diet alone.

Acknowledgements

None.

Financial Support and Sponsorship

None.

Conflicts of Interest

There are no conflicts of interest.

REFERENCES

1. Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *Bmj* 2013; 346:e7492.
2. Moynihan PJ, Kelly SA. Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. *J Dent Res* 2014; 93(1):8-18.
3. World Health Organization. Diet, nutrition and the prevention of chronic diseases: report of a joint WHO/FAO expert consultation. Geneva2003. Available from: http://www.who.int/nutrition/publications/obesity/WHO_TRS_916/en/.
4. World Health Organization. Guideline: Sugars intake for adults and children. Geneva 2015. Available from: http://www.who.int/nutrition/publications/guidelines/sugars_intake/en/.
5. ** Scientific Advisory Committee on Nutrition. Carbohydrates and Health. London: The Stationery Office; 2015. Available from: <https://www.gov.uk/government/publications/sacn-carbohydrates-and-health-report>.

This is an important resource which details the findings of the committee regarding the evidence base for carbohydrates and health, outlining many areas where insufficient data are available.

6. U.S. Department of Health and Human Services and U.S. Department of Agriculture. 2015 – 2020 Dietary Guidelines for Americans 2015. Available from: <http://health.gov/dietaryguidelines/2015/guidelines/>.
7. National Health and Medical Research Council. Australian Dietary Guidelines. Canberra2013. Available from: https://www.eatforhealth.gov.au/sites/default/files/files/the_guidelines/n55_australian_dietary_guidelines.pdf.
8. European Food Safety Authority. Scientific Opinion on Dietary Reference Values for carbohydrates and dietary fibre. *EFSA J* 2010; 8(3):1462.
9. ** Yang Q, Zhang Z, Gregg EW, et al. Added sugar intake and cardiovascular diseases mortality among US adults. *JAMA Intern Med* 2014; 174(4):516-24.

This cohort study from The National Health and Nutrition Examination Survey (NHANES) is important because of a paucity of data relating sugar intake to cardiovascular disease. The study reports a detrimental effect of a very high sugar intake on CVD mortality when correcting for confounders.

10. Bates B, Lennox A, Prentice A, et al. National Diet and Nutrition Survey: Results from Years 1, 2, 3 and 4 (combined) of the Rolling Programme (2008/2009 – 2011/2012)2014. Available from: <https://www.gov.uk/government/publications/national-diet-and-nutrition-survey-headline-results-from-years-1-2-and-3-combined-of-the-rolling-programme-200809-201011>.
11. Jebb SA. Carbohydrates and obesity: from evidence to policy in the UK. *Proc Nutr Soc* 2015; 74(3):215-20.

There are various options open to policy makers in the face of an unacceptably average intake of free sugars in the UK diet. This conference paper clearly sets out options for policy makers to consider, and the implications if interventions are implemented, if they wish to achieve reductions at a population level.

12. Kuzma JN, Cromer G, Hagman DK, et al. No difference in ad libitum energy intake in healthy men and women consuming beverages sweetened with fructose, glucose, or high-fructose corn syrup: a randomized trial. *Am J Clin Nutr* 2015; 102(6):1373-80.
13. * Schwarz JM, Noworolski SM, Wen MJ, et al. Effect of a High-Fructose Weight-Maintaining Diet on Lipogenesis and Liver Fat. *J Clin Endocrinol Metab* 2015; 100(6):2434-42.

Studies that investigate the effect of diet on complex metabolic pathways are expensive and difficult, and compliance is always an issue. Moreover, pathways may have to be ‘pushed’ with interventions at the high end of normal. In this study the issue of compliance was minimised by the use of ‘in-patient’ facilities but the metabolic effects of fructose were studied in response to an experimental high-dose sugar-sweetened beverage. The study found that liver fat and *de novo* lipogenesis were significantly higher after a high fructose intervention notably in the absence of weight gain. This study illustrates the capabilities of a high-fructose dose to affect pathways of fatty acid accumulation in the liver.

14. Bruun JM, Maersk M, Belza A, et al. Consumption of sucrose-sweetened soft drinks increases plasma levels of uric acid in overweight and obese subjects: a 6-month randomised controlled trial. *Eur J Clin Nutr* 2015; 69(8):949-53.
15. Maersk M, Belza A, Stodkilde-Jorgensen H, et al. Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: a 6-mo randomized intervention study. *Am J Clin Nutr* 2012; 95(2):283-9.
16. Ma J, Fox CS, Jacques PF, et al. Sugar-sweetened beverage, diet soda, and fatty liver disease in the Framingham Heart Study cohorts. *J Hepatol* 2015; 63(2):462-9.
17. ** Zeevi D, Korem T, Zmora N, et al. Personalized Nutrition by Prediction of Glycemic Responses. *Cell* 2015; 163(5):1079-94.

This novel study aimed to develop a prediction algorithm for postprandial glycaemic excursions, in healthy people, in response to everyday meals. What emerges from the work is the high level of variability between people in response to the same meals.

18. Malik VS, Hu FB. Fructose and Cardiometabolic Health: What the Evidence From Sugar-Sweetened Beverages Tells Us. *J Am Coll Cardiol* 2015; 66(14):1615-24.
19. Egli L, Lecoultre V, Theytaz F, et al. Exercise prevents fructose-induced hypertriglyceridemia in healthy young subjects. *Diabetes* 2013; 62(7):2259-65.
20. Egli L, Lecoultre V, Cros J, et al. Exercise performed immediately after fructose ingestion enhances fructose oxidation and suppresses fructose storage. *Am J Clin Nutr* 2016; 103(2):348-55.
21. Stanhope KL. Sugar consumption, metabolic disease and obesity: The state of the controversy. *Crit Rev Clin Lab Sci* 2016; 53(1):52-67.
22. ** Stanhope KL, Medici V, Bremer AA, et al. A dose-response study of consuming high-fructose corn syrup-sweetened beverages on lipid/lipoprotein risk factors for cardiovascular disease in young adults. *Am J Clin Nutr* 2015; 101(6):1144-54.

This is an important mechanistic study demonstrating the principal of a dose dependent increase in cardiovascular risk factors when high fructose corn syrup beverages are given to healthy individuals.

23. Umpleby M, Shojaee-Moradie F, Fielding B, et al. A diet low in sugar reduces the production of atherogenic lipoproteins in men with high liver fat. *Atherosclerosis* 2015; 241:e46.
24. * Imamura F, O'Connor L, Ye Z, et al. 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* 2015; 351:h3576.

This work confirms the notion that habitual consumption of sugar sweetened beverages is associated with a greater incidence of type 2 diabetes. Although not without statistical issues, the study concludes that artificially sweetened beverages and fruit juice were not likely to be healthy alternatives to sugar sweetened beverages for the prevention of type 2 diabetes.

25. Ma Y, He FJ, Yin Y, et al. Gradual reduction of sugar in soft drinks without substitution as a strategy to reduce overweight, obesity, and type 2 diabetes: a modelling study. *Lancet Diabetes Endocrinol* 2016; 4(2):105-14.
26. UK Health Forum. Short and sweet: Why the Government should introduce a sugary drinks tax. 2016. Available from: <http://nhfshare.heartforum.org.uk/RMAssets/Modelling/CRUKSSB/Short%20and%20Sweet%20Tech%20Sum%20LIVE.pdf>.
27. Briggs AD, Mytton OT, Kehlbacher A, et al. Overall and income specific effect on prevalence of overweight and obesity of 20% sugar sweetened drink tax in UK: econometric and comparative risk assessment modelling study. *BMJ* 2013; 347:f6189.
28. Long MW, Gortmaker SL, Ward ZJ, et al. Cost Effectiveness of a Sugar-Sweetened Beverage Excise Tax in the U.S. *Am J Prev Med* 2015; 49(1):112-23.
29. Julia C, Mejean C, Vicari F, et al. Public perception and characteristics related to acceptance of the sugar-sweetened beverage taxation launched in France in 2012. *Public Health Nutr* 2015; 18(14):2679-88.
30. Colchero MA, Popkin BM, Rivera JA, Ng SW. Beverage purchases from stores in Mexico under the excise tax on sugar sweetened beverages: observational study. *BMJ* 2016; 352:h6704.

31. Duhaney T, Campbell N, Niebylski ML, et al. Death by diet: the role of food pricing interventions as a public policy response and health advocacy opportunity. *Can J Cardiol* 2015; 31(2):112-6.
32. Obesity: we need to move beyond sugar. *Lancet* 2016; 387(10015):199.
33. Vanderlee L, White CM, Bordes I, et al. The efficacy of sugar labeling formats: Implications for labeling policy. *Obesity (Silver Spring)* 2015; 23(12):2406-13.
34. Hobbs L. Sweeteners from Starch: Production, Properties and Uses. *Starch: Chemistry and Technology*, Third Edition: Elsevier Inc.; 2009. p. 797-8312.
35. Willems JL, Low NH. Major carbohydrate, polyol, and oligosaccharide profiles of agave syrup. Application of this data to authenticity analysis. *J Agric Food Chem* 2012; 60(35):8745-54.
36. Moore JB, Gunn PJ, Fielding BA. The role of dietary sugars and de novo lipogenesis in non-alcoholic fatty liver disease. *Nutrients* 2014; 6(12):5679-703.
37. Munsell CR, Harris JL, Sarda V, Schwartz MB. Parents' beliefs about the healthfulness of sugary drink options: opportunities to address misperceptions. *Public Health Nutr* 2016; 19(1):46-54.
38. Griffin BA. Relevance of liver fat to the impact of dietary extrinsic sugars on lipid metabolism. *Proc Nutr Soc* 2015; 74(3):208-14.
39. Phillips KM, Carlsen MH, Blomhoff R. Total antioxidant content of alternatives to refined sugar. *J Am Diet Assoc* 2009; 109(1):64-71.
40. Perkins TD, van den Berg AK. Maple syrup-production, composition, chemistry, and sensory characteristics. *Adv Food Nutr Res* 2009; 56:101-43.
41. da Silva PM, Gauche C, Gonzaga LV, et al. Honey: Chemical composition, stability and authenticity. *Food Chem* 2016; 196:309-23.
42. US Department of Agriculture ARS, Nutrient Data Laboratory. USDA National Nutrient Database for Standard Reference, Release 28. Version Current: September 2015. Available from: <http://www.ars.usda.gov/nea/bhnrc/ndl>.
43. Public Health England. McCance and Widdowson's The Composition of Foods Integrated Dataset 2015. Available from: <https://www.gov.uk/government/publications/composition-of-foods-integrated-dataset-cofid>.
44. Dobbs R, Sawers C, Thompson F, et al. Overcoming obesity: An initial economic analysis. McKinsey Global Institution, 2014.

Table 1 Recently published guidance on consumption of dietary sugars

Organisation (Year)	Recommendations
World Health Organisation (2015) ^[4]	The intake of free sugars should be reduced to less than 10% of total energy intake In both adults and children
UK Scientific Advisory Committee on Nutrition (2015) ^[5]	The average population intake of free sugars should not exceed 5% of total dietary energy for age groups from 2 years upwards
U.S. Department of Health and Human Services (2015) ^[6]	Consume less than 10% of calories per day from added sugars
Australian National Health and Medical Research Council (2013) ^[7]	Limit intake of foods and drinks containing added sugars such as confectionary, sugar-sweetened soft drinks and cordials, fruit drinks, vitamin waters, energy and sports drinks
European Food Safety Authority (2010) ^[8]	The available evidence is insufficient to set an upper limit for intake of (added) sugars based on their effects on the basis of body weight or a risk reduction for dental caries

Table 2 Definitions

Term	Definition
Sugars*	Conventionally describes chemically the monosaccharides (glucose, fructose, galactose) and disaccharides (sucrose, lactose, maltose). Sugars include those occurring naturally in foods and beverages or added during processing and preparation.
Free Sugars	'Monosaccharides and disaccharides added to foods and beverages by the manufacturer, cook or consumer, and sugars naturally present in honey, syrups fruit juices and juice concentrates' ^[4] . Under this definition lactose when naturally present in milk and milk products is excluded.
Added Sugars	'Syrups and other caloric sweeteners used as a sweetener in other food products. Naturally occurring sugars such as those in fruit or milk are not added sugars' ^[6]
Monosaccharides	The most simple form of carbohydrates including the three primary hexoses (six-carbon simple sugars): glucose, fructose and galactose; which are the monomers that make up naturally occurring di-, oligo- and poly-saccharides
Disaccharides	Product of condensation reaction between two monosaccharides; includes sucrose, lactose, maltose
Sucrose	A crystalline disaccharide of fructose and glucose found in many plants, predominantly sugar cane and sugar beets. It is extracted and refined and used widely as table sugar
Dextrose	Dextrorotatory-glucose or D-glucose; the most common isomer of glucose.
Levulose	Dextrorotatory-fructose or D-fructose; origin in dextrorotatory form describing molecule that rotates light to the left.
Glucose syrups (<i>corn syrup in U.S.</i>)	Purified aqueous solutions of nutritive saccharides obtained from edible starch, typically corn in U.S. and potato and wheat in the EU ^[34]
Glucose-fructose or Fructose-glucose syrup (<i>high-fructose corn syrup in U.S.</i>)	Aqueous solutions of nutritive saccharides obtained from edible starch in which a portion (at least 42%) of the dextrose (D-glucose) has been isomerized to fructose ^[34] ; Directive 2001/111/EC requires glucose syrups containing more than 5 % of fructose (dry matter) must be labelled as glucose-fructose syrup or fructose-glucose syrup and dried glucose-fructose syrup or dried fructose-glucose syrup, to reflect whether the glucose component or the fructose component is in greater proportion
Agave Syrup	Sweetener commercially produced from several species of agave plant; processing very similar to that of glucose-fructose syrups, but fructose content much higher at ~84% ^[35]

* Examples of sugars commonly found as ingredients: Sucrose, fructose, glucose, dextrose, maltose, lactose, trehalose, brown sugar, turbinado sugar, demerara sugar, raw sugar, cane sugar, fruit sugar, invert sugar, corn sweetener, corn syrup, high-fructose corn syrup, malt syrup, glucose syrup, glucose-fructose syrup, fructose-glucose syrup, honey, molasses, date syrup, agave syrup

Table 3: Energy and sugars found per 100g in common dietary sweeteners¹

Sweetener	Energy (kcal)	Energy (kJ)	Water (g)	Total Sugars (g)	Sucrose (g)	Glucose (g)	Fructose (g)
White sugar	387	1618	0.02	99.8	99.8	0.0	0.0
Brown sugar	380	1590	1.34	97.0	94.6	1.4	1.1
Turbinado sugar	399	1670	0.03	99.2	99.2	0.0	0.0
Honey	304	1272	17.1	82.1	0.9	35.8	40.9
Molasses	290	1213	21.9	74.7	29.4	11.9	12.8
Maple syrup	260	1088	32.4	60.5	58.3	1.6	0.5
Agave syrup	310	1297	22.9	68.0	0.0	12.4	55.6
Golden syrup	298	1269	20.0	79.0	32.8	23.1	23.0
HFCS	281	1176	24.0				

¹Data taken from the USDA National Nutrient Database^[42] except data for golden syrup, which was extracted from the Public Health England Composition of Foods Integrated Dataset^[43].

Figure 1

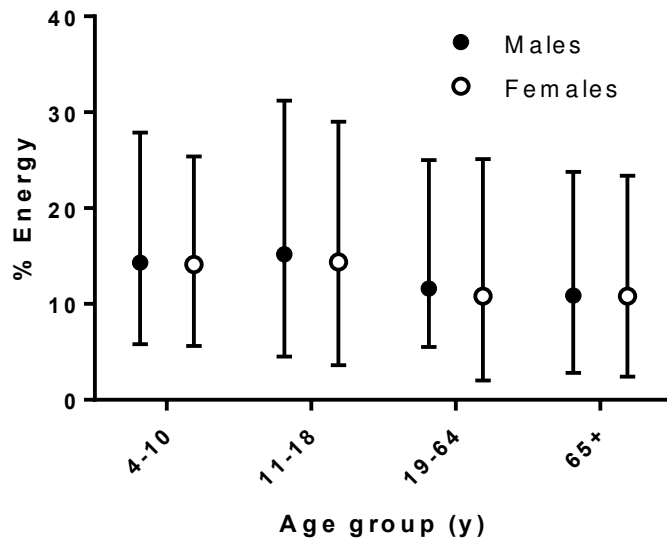


Figure 1. The percentage of energy intake from non-milk extrinsic sugars in the UK National Diet and Nutrition Survey (median, upper and lower 25 percentiles). Results from Years 1,2,3 and 4 (combined) of the Rolling Programme (2008/2009 – 2011/12)^[10].