

Relation of total sugars, fructose and sucrose with incident type 2 diabetes: a systematic review and meta-analysis of prospective cohort studies

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ABSTRACT

BACKGROUND: Sugar-sweetened beverages are associated with type 2 diabetes. To assess whether this association holds for the fructose-containing sugars they contain, we conducted a systematic review and meta-analysis of prospective cohort studies.

METHODS: We searched MEDLINE, Embase, CINAHL and the Cochrane Library (through June 2016). We included prospective cohort studies that assessed the relation of fructose-containing sugars with incident type 2 diabetes. Two independent reviewers extracted relevant data and assessed risk of bias. We pooled

risk ratios (RRs) using random effects meta-analyses. The overall quality of the evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system.

RESULTS: Fifteen prospective cohort studies (251 261 unique participants, 16 416 cases) met the eligibility criteria, comparing the highest intake (median 137, 35.2 and 78 g/d) with the lowest intake (median 65, 9.7 and 25.8 g/d) of total sugars, fructose and sucrose, respectively. Although there was no association of total sugars (RR 0.91, 95% confidence interval [CI] 0.76–1.09) or fructose (RR 1.04, 95% CI 0.84–1.29) with

type 2 diabetes, sucrose was associated with a decreased risk of type 2 diabetes (RR 0.89, 95% CI 0.80–0.98). Our confidence in the estimates was limited by evidence of serious inconsistency between studies for total sugars and fructose, and serious imprecision in the pooled estimates for all 3 sugar categories.

INTERPRETATION: Current evidence does not allow us to conclude that fructose-containing sugars independent of food form are associated with increased risk of type 2 diabetes. Further research is likely to affect our estimates. **Trial registration:** ClinicalTrials.gov, no. NCT01608620

Sugars, particularly fructose-containing sugars, have been implicated as an important driver in the rise in incidence of type 2 diabetes.^{1,2} Sugar-sweetened beverages, which represent the greatest source of fructose-containing sugars in the diet,³ form most of the basis for this link.^{4,5} It remains unclear whether the association between beverages sweetened with sugars and type 2 diabetes can be explained by the fructose that these beverages contain. Several high-quality systematic reviews and meta-analyses have assessed the relation of sugar-sweetened beverages with incident type 2 diabetes. Our objective was to conduct a systematic review and meta-analysis of prospective cohort

studies to determine the role of fructose-containing sugars independent of food form in the development of type 2 diabetes.

Methods

Our systematic review and meta-analysis followed the Cochrane Handbook for Systematic Reviews and Interventions,⁶ and reported results according to Meta-analysis of Observational Studies in Epidemiology (MOOSE) guideline⁷ and PRISMA guideline (www.prisma-statement.org). The study protocol is registered (ClinicalTrials.gov identifier, NCT01608620).

Data sources and searches

We searched MEDLINE, Embase, CINAHL and the Cochrane Library databases through June 2016. The search strategy is presented in supplementary Table 1 (Appendix 1, available at www.cmaj.ca/lookup/suppl/doi:10.1503/cmaj.160706/-/DC1). The search was restricted to human studies without language restrictions. Manual searches of the reference lists of included studies supplemented electronic searches.

Study selection

We included prospective cohort studies that assessed intake of fructose-containing sugars (total sugars, fructose, sucrose, high-fructose corn syrup or added sugars) and incident type 2 diabetes in participants who did not have diabetes.

Data extraction

Two reviewers (C.T. and R.T.) independently extracted relevant data from included studies. The main outcome was type 2 diabetes risk expressed as risk ratios (RRs) with 95% confidence intervals (CI). Authors were contacted for missing data.

Risk of bias

The Newcastle-Ottawa Scale (NOS) was used to assess the risk of bias in included studies, where up to 9 points were awarded based on cohort selection, comparability (adjustments) and

ascertainment of the outcome.⁸ Owing to concerns regarding the use of cut-off scores,⁶ we did not use our prespecified cut-off score for NOS.⁹ Differences were reconciled by consensus.

Grading of the evidence

The quality and strength of the evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) system.¹⁰⁻²² Included observational studies started at low-quality evidence by default and then were downgraded or upgraded based on prespecified criteria. Criteria to downgrade included study limitations (weight of studies showed risk of bias by NOS), inconsistency (substantial unexplained inter-study heterogeneity, $I^2 > 50\%$ and $p < 0.10$), indirectness (presence of factors relating to the population, exposures and outcomes that limit generalizability), imprecision (95% CIs were wide or crossed a minimally important difference of 10% [RR 0.9–1.1]) and publication bias (significant evidence of small-study effects). Criteria to upgrade included a large size effect (RR > 2 or RR < 0.5 in the absence of plausible confounders), a dose–response gradient and attenuation by plausible confounding effects.

Statistical analysis

To obtain summary estimates, we natural log-transformed and pooled the RRs using the generic inverse variance method with random-effects models. We used RRs comparing extreme quan-

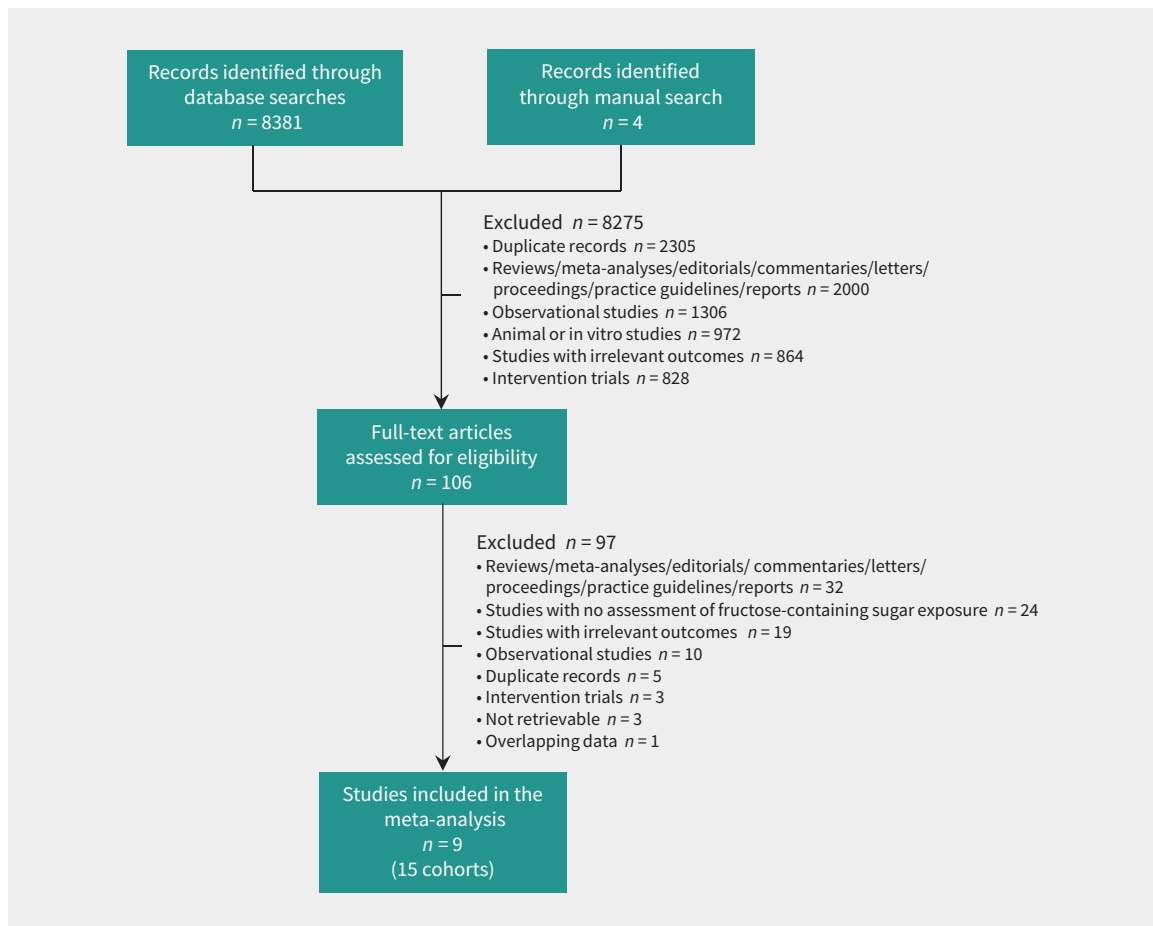


Figure 1: Summary of evidence search and selection.

tiles and scaled RRs per 100 g/d for total sugars, 50 g/d for fructose and 50 g/d for sucrose to standardize the doses based on estimated average intakes in Canada.²³ Heterogeneity was assessed (Cochran Q statistic) and quantified (I^2 statistic). If I^2 was greater than or equal to 50%, we interpreted this as indicating substantial heterogeneity.^{6,16} We investigated possible sources of heterogeneity. To assess whether any single study exerted an undue influence on the summary estimates, we performed sensitivity analyses by systematically removing each study with recalculation of the summary estimates. We performed additional sensitivity analyses by restricting pooled analyses to studies using validated measures of sugars intake to assess any influence of how the exposures were assessed. Prespecified subgroup analyses were done by sex, follow-up, NOS and individual domains of NOS using meta-regression analyses. Linear and nonlinear dose-response analyses were assessed by using generalized least squares trend (GLST) estimation models and spline curve model-

ling (MKSPLINE procedure). If 10 or more cohort comparisons were available, we investigated publication bias by visual inspection of funnel plots and using the Begg and Egger tests. Data were analyzed using Review Manager (RevMan) version 5.2 (The Nordic Cochrane Centre) and Stata version 12 (StataCorp).

Results

Figure 1 shows the flow of the literature search. Out of 8381 reports, we included 9 reports of 15 cohort studies involving 251 261 unique participants and 16 416 cases of type 2 diabetes:^{24–32} 12 cohort comparisons ($n = 105 846$, 13 727 cases) for total sugars, 6 cohort comparisons ($n = 107 972$, 3833 cases) for fructose and 8 cohort comparisons ($n = 192 332$, 4535 cases) for sucrose. There were no cohort comparisons available for high-fructose corn syrup or added sugars.

Table 1 shows the characteristics of the included studies. Participants were from 11 countries and had a median age of

Table 1 (part 1 of 2): Characteristics of prospective cohort studies investigating the dietary intake of total sugars, fructose and sucrose and incident type 2 diabetes

Study, year	Cohort	Country	No. of participants	No. of incident cases	Age, yr	Duration of study, yr	Dietary intake assessment (at baseline)	Sugars exposure,† g/d	Method of outcome assessment	Funding source‡
Colditz et al., 1992 ²⁴	Nurses Health Study	United States	84 360 (F)	702	34–59	6	Validated SFFQ	Sucrose exposure unknown	Self-report	Agency
Meyer et al., 2000 ²⁵	Iowa Women's Health Study	United States	35 988 (F)	1141	55–69	6	Validated FFQ	Sucrose 25.8–57.7, fructose 12.5–35.5	Self-report	Agency
Janket et al., 2003 ²⁶	Women's Health Study	United States	38 480 (F)	918	46–61	6	Validated SFFQ	Sucrose 25.8–57.2, fructose 25.8–57.2, total sugars 25.8–57.2	Self-report	Agency
Hodge et al., 2004 ²⁷	Melbourne Collaborative Cohort Study	Australia	31 641	365	27–75	4	FFQ	Total sugars 63.6–194.4	Physician diagnosis	Agency
Barclay et al., 2007 ²⁸	Blue Mountains Eye Study	Australia	1833	138	≥ 49	10	Validated SFFQ	Total sugars 100	Self-report	NA
Montonen et al., 2007 ²⁹	Finnish Mobile Health Clinic Examination Survey	Finland	4284	175	40–69	12	Interview with questionnaire	Sucrose 28.5–79.5, fructose 6.0–28.8, total sugars 92.0–171	Medical record linkage	NA
Schulze et al., 2008 ³⁰	EPIC-Potsdam	Germany	9702 (M), 15 365 (F)	491 (M), 355 (F)	35–65	7–11	Validated SFFQ	Sucrose 22.5–102 (M), 28.2–83.4 (F); fructose 8.4–40.6 (M), 11–34.8 (F)	Physician diagnosis	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	The Netherlands	2290	828	20–70	12	Quantitative Dietary Questionnaire	Total sugars 65–137	Medical record linkage	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	Denmark	4037	2055	50–64	12	Validated SFFQ	Total sugars 65–137	Medical record linkage	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	France	867 (F)	288	40–65	12	Quantitative Dietary Questionnaire	Total sugars 65–137	Medical record linkage	Agency

Table 1 (part 2 of 2): Characteristics of prospective cohort studies investigating the dietary intake of total sugars, fructose and sucrose and incident type 2 diabetes

Study, year	Cohort	Country	No. of participants	No. of incident cases	Age, yr	Duration of study, yr	Dietary intake assessment (at baseline)	Sugars exposure,† g/d	Method of outcome assessment	Funding source‡
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	Germany	3578	1584	40–65 (M), 35–65 (F)	12	Quantitative Dietary Questionnaire	Total sugars 65–137	Medical record linkage	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	Italy	3393	1437	35–74	12	Quantitative Dietary Questionnaire; Naples, Validated SFFQ; Ragusa, Dietary Interview	Total sugars 65–137	Medical record linkage	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	Spain	5889	2564	40–65 (M), 35–65 (F)	12	Quantitative Dietary Questionnaire, Dietary Interview	Total sugars 65–137	Medical record linkage	Agency
Sluijs et al., 2013 ³¹	EPIC-InterAct Study	Sweden	5401	2622	30–72	12	Validated SFFQ, 14-d record	Total sugars 65–137	Medical record linkage	Agency
Ahmadi-Abhari et al., 2014 ³²	EPIC-Norfolk Study	England	4153	753	40–79	6.3	Validated SFFQ, 7-d food diary	Sucrose 25–76.5, fructose 8–32, total sugars 71.5–149.5	Medical record linkage	Agency

Note: F = females, FFQ = Food-Frequency Questionnaire, HR = hazard ratio, IQR = interquartile range, M = males, NA = not available, SD = standard deviation, SFFQ = Semiquantitative Food-Frequency Questionnaire.

*Durations reported as mean ± SD, median (IQR) or as a range.

†Sugars exposure reported as median (IQR) or as a range.

‡ Agency funding is that from government, university or not-for-profit health agency sources.

52.6 years (interquartile range [IQR] 20–79 yr). There were more female than male participants, with 4 large female cohorts and 11 smaller mixed cohorts. Median follow-up was 12 years (IQR 4–12 yr), 6.3 years (IQR 6–12 yr) and 6.2 years (IQR 6–12 yr) for total sugars, fructose and sucrose, respectively. Ascertainment of incident cases was done by medical record linkage (60%), self-report (27%) and physician diagnosis (13%).

Median intakes for total sugars, fructose and sucrose were 65 g/d (IQR 25.8–100 g/d), 9.7 g/d (IQR 6–25.8 g/d) and 25.8 g/d (IQR 22.5–28.5 g/d), respectively, in the lowest quantile of intake. In the highest quantile of intake, median intakes for total sugars, fructose and sucrose were 137 g/d (IQR 57.2–194.4 g/d), 35.2 g/d (IQR 28.8–57.2 g/d) and 78 g/d (IQR 57.2–102 g/d), respectively. Dietary intake was assessed by food frequency questionnaires, semiquantitative food frequency questionnaires (47%), quantitative dietary questionnaires (20%) or mixed methods (33%). No studies differentiated between added sugars and naturally occurring sugars.

Funding sources did not include industry funding. Thirteen studies reported funding from agency alone, whereas the other 2 studies did not report funding sources.

Supplementary Table 2 (Appendix 1) shows the statistical adjustments performed in the included studies. All studies adjusted for the prespecified primary confounding variable (age) and adjusted for at least 4 of 5 secondary confounding variables

(markers of overweight/obesity, family history of diabetes, energy intake, physical activity, sex).

Supplementary Table 3 (Appendix 1) shows the NOS scores for the included studies. Although several studies lost points for selection and outcome assessment, there was no evidence of serious risk of bias across the included studies.

Visual inspection of funnel plots (Supplementary Figure 18, Appendix 1), and formal testing with the Begg ($p = 0.7$) and Egger tests ($p = 0.4$) did not show evidence of publication bias for total sugars. Publication bias was not assessed for fructose and sucrose because there were less than 10 cohort comparisons.

Supplementary Table 4 (Appendix 1) shows a summary of the GRADE assessments for the association of total sugars, fructose and sucrose intake with incident type 2 diabetes. The evidence for a lack of harm was rated as very low quality for total sugars and fructose because of downgrades for serious inconsistency and imprecision, and low quality for sucrose because of a downgrade for serious imprecision and an upgrade for a significant inverse dose–response gradient.

Intake of sugars and type 2 diabetes

Figure 2 and supplementary Figure 1 (Appendix 1) show the relation between intake of total sugars and incident type 2 diabetes. There was no association (RR 0.91, 95% CI 0.76–1.09) with evidence of substantial heterogeneity ($I^2 = 76%$, $p < 0.001$) when we

compared the highest and the lowest levels of intake. Risk ratio per 100 g/d intake was 0.92 (95% CI 0.77–1.08), with evidence of substantial heterogeneity ($I^2 = 79\%$, $p < 0.001$).

Figure 3 and supplementary Figure 2 (Appendix 1) show the relation between fructose intake and incident type 2 diabetes. We found no association (RR 1.04, 95% CI 0.84–1.29) with evidence of substantial heterogeneity among studies

($I^2 = 71\%$, $p < 0.01$) when we compared the highest and the lowest levels of intake. Risk ratio per 50 g/d intake was 1.09 (95% CI 0.73–1.63), with evidence of substantial heterogeneity ($I^2 = 75\%$, $p < 0.01$).

Figure 4 and supplementary Figure 3 (Appendix 1) show the relation between sucrose intake and incident type 2 diabetes. We found a significant protective association (RR 0.89, 95% CI 0.80–

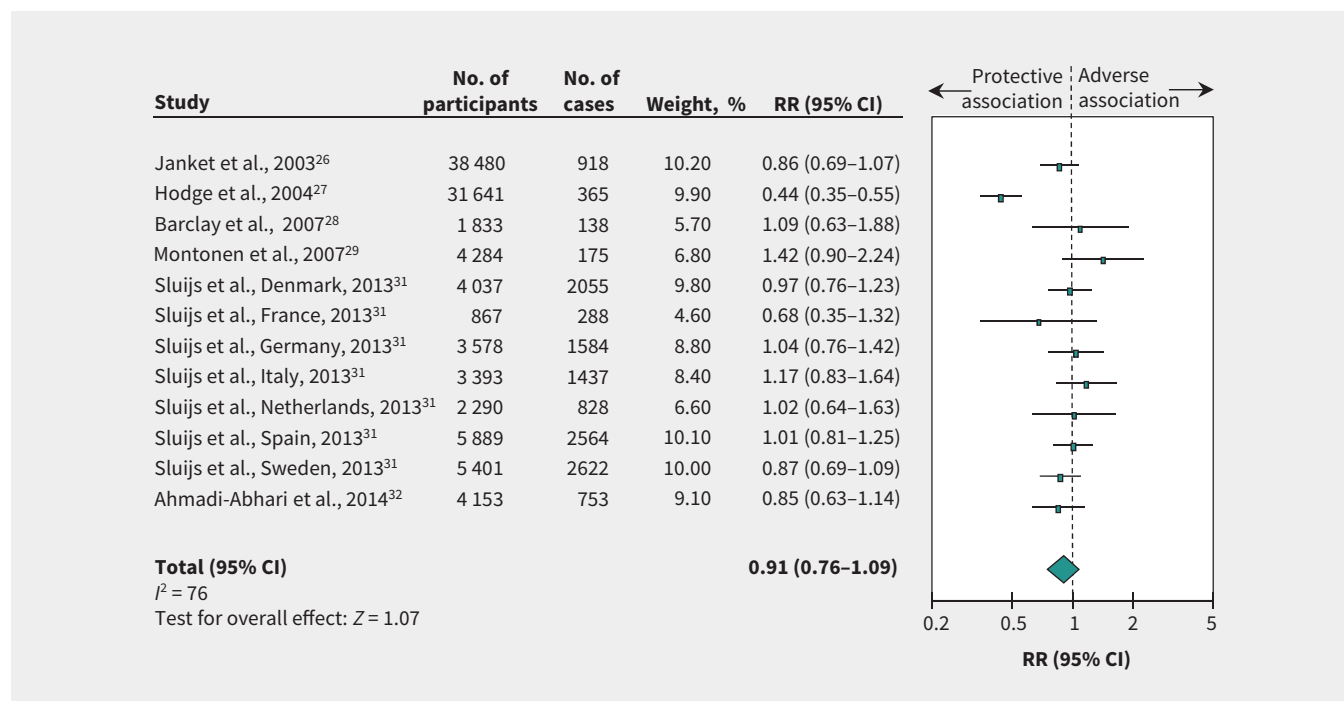


Figure 2: Relation between intake of total sugars and incident type 2 diabetes (highest v. lowest level of intake). Pooled risk estimate is represented by the blue diamond. Values of $I^2 \geq 50\%$ indicate substantial heterogeneity.^{6,16} Values greater than 1.0 indicate an adverse association. CI = confidence interval, RR = risk ratio.

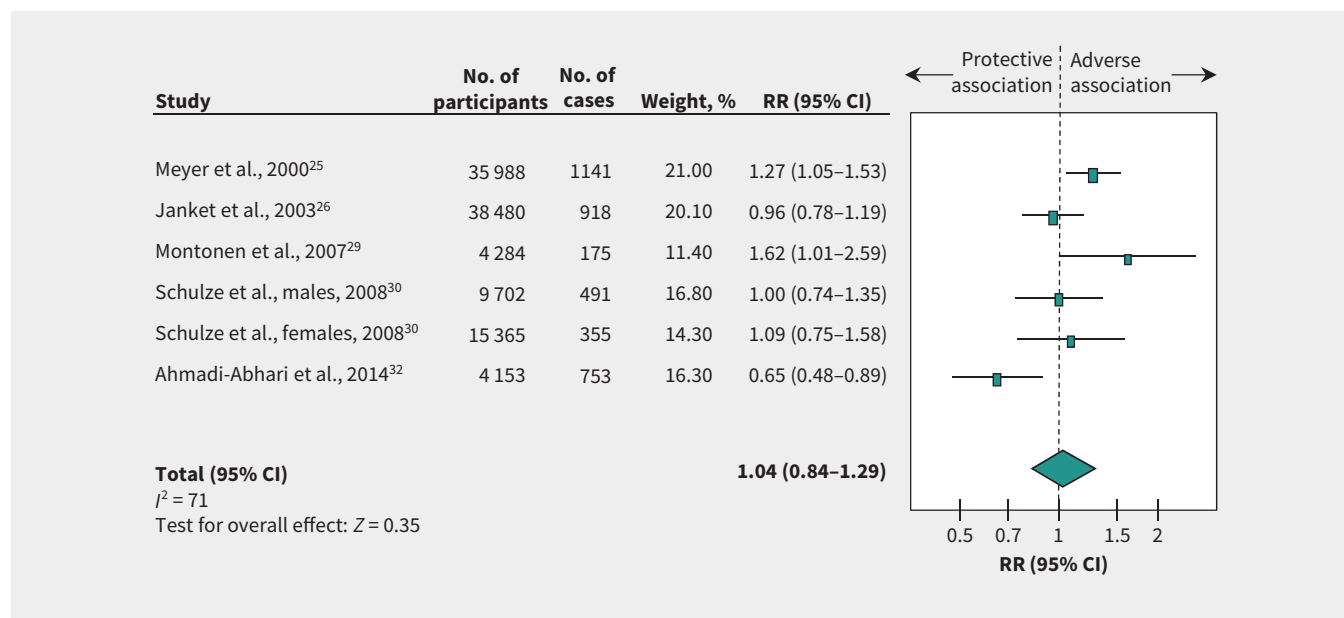


Figure 3: Relation between intake of fructose and incident type 2 diabetes (highest v. lowest level of intake). Pooled risk estimate is represented by the blue diamond. Values of $I^2 \geq 50\%$ indicate substantial heterogeneity.^{6,16} Values greater than 1.0 indicate an adverse association. CI = confidence interval, RR = risk ratio.

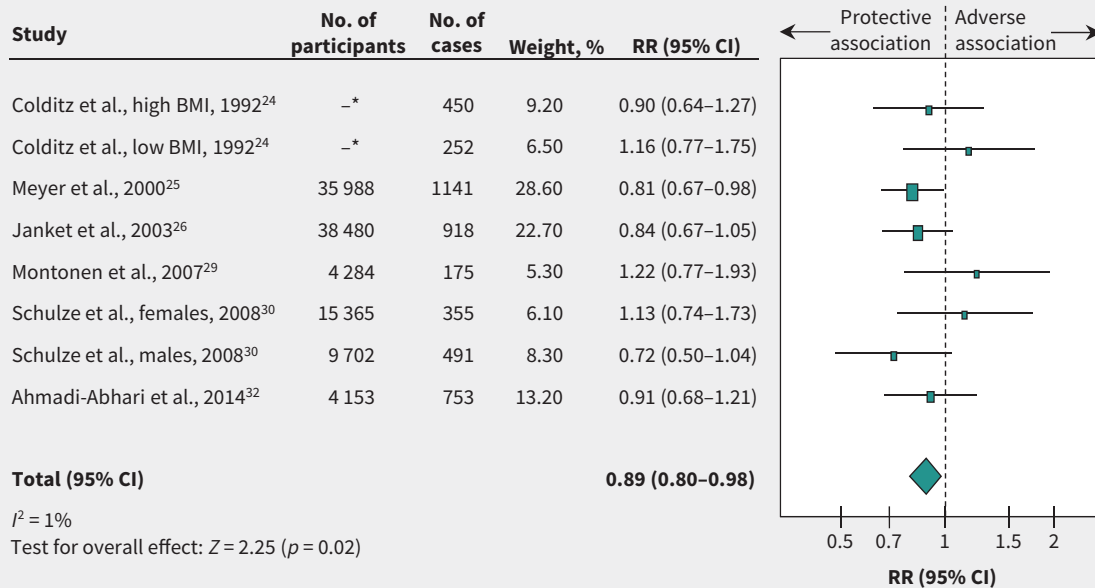


Figure 4: Relation between intake of sucrose and incident type 2 diabetes (highest v. lowest level of intake). Pooled risk estimate is represented by the blue diamond. Values of $I^2 \geq 50\%$ indicate substantial heterogeneity.^{6,16} Values greater than 1.0 indicate an adverse association. BMI = body mass index, CI = confidence interval, RR = risk ratio. *For Colditz and colleagues,²⁴ the total number of participants with high and low BMI was 84 360.

0.98) with no evidence of heterogeneity ($I^2 = 1\%$, $p = 0.4$) when we compared the highest and the lowest levels of intake. Risk ratio per 50 g/d intake was 0.91 (95% CI 0.83–1.00), with no evidence of heterogeneity ($I^2 = 12\%$, $p = 0.34$).

Additional analyses

The systematic removal of each study did not modify the lack of an association for total sugars or fructose. However, the removal of the study by Hodge and colleagues²⁷ ($I^2 = 9\%$, $p = 0.4$) in the analysis of total sugars and the study by Ahmadi-Abhari and colleagues³² ($I^2 = 40\%$, $p = 0.2$) in the fructose analysis did explain away most of the evidence of heterogeneity. The inverse association for sucrose was lost through the systematic removal of the studies by Colditz and colleagues²⁴ (high body mass index cohort comparison; RR 0.89, 95% CI 0.79–1.01), Meyer and colleagues²⁵ (RR 0.92, 95% CI 0.81–1.04), Janket and colleagues²⁶ (RR 0.91, 95% CI 0.80–1.03), Schulze and colleagues³⁰ (cohort comparison of males; RR 0.90, 95% CI 0.81–1.01) or Ahmadi-Abhari and colleagues³² (RR 0.89, 95% CI 0.79–1.01). However, the recalculated 95% CI did not include evidence of clinically important harm.

Restricting analyses to studies in which sugars were assessed using validated measures neither modified the associations nor the evidence of heterogeneity for total sugars, fructose or sucrose (supplementary Figures 4–6, Appendix 1).

Supplementary Figures 7–12 (Appendix 1) provide the a priori subgroup analyses. There was no evidence of effect modification for the associations of total sugars, fructose and sucrose with incident type 2 diabetes in a priori subgroup analyses. Any evidence of significant interstudy heterogeneity was not explained by a priori subgroup analyses.

There was no evidence of a dose–response gradient for total sugars or fructose using GLST estimation (supplementary Figures 13 and 14, Appendix 1). No dose–response gradient or dose thresholds for fructose was seen using the MKSPLINE procedure (supplementary Figure 15, Appendix 1), whereas total sugars could not be modelled because of insufficient data. Results of GLST estimation for sucrose showed evidence of a significant inverse relationship with incident type 2 diabetes per 25 g/d intake (RR 0.92, 95% CI 0.85–0.99, $p = 0.03$) (supplementary Figure 16, Appendix 1), but this relation was not seen for results using the MKSPLINE procedure (supplementary Figure 17, Appendix 1).

Interpretation

We conducted a systematic review and meta-analysis of prospective cohort studies of the relation between intake of sugars and incident type 2 diabetes. Pooled analyses showed that intakes of total sugars and fructose were not associated with type 2 diabetes, whereas intake of sucrose was associated with an 11% decrease in type 2 diabetes.

Our results do not support a hypothesis that the positive association seen between sugar-sweetened beverages and diabetes is mediated by the fructose-containing sugars they contain. Systematic reviews and meta-analyses have shown that sugar-sweetened beverages are associated with an increase in the risk of type 2 diabetes.^{4,5,33} Our pooled analyses failed to show a similar increase despite the inclusion of data from mostly the same set of cohorts.

The lack of an adverse association is difficult to reconcile with the biological mechanisms and ecological observations linking fructose-

containing sugars to type 2 diabetes.^{34–39} It is also difficult to reconcile with our earlier systematic review and meta-analysis showing a positive association between fructose intake and gout,⁹ given the emerging links between uric acid and diabetes.³⁷ One possible explanation for the lack of agreement is residual confounding from reverse causality. People at high risk of type 2 diabetes may avoid sugars as a preventive strategy, which decreases the risk associated with intake of total sugars, fructose or sucrose. Another explanation may relate to the increased intake of healthier food sources of sugars other than sugar-sweetened beverages, which themselves show null or protective associations with type 2 diabetes.

Although sugar-sweetened beverages are sources of most fructose-containing sugars in Canadian and American diets, other sources contribute meaningfully to overall intake^{23,40} (e.g., grains and grain products, fruit and fruit products, and dairy and dairy products).^{23,40} Many of these other food sources, which tend to be sweetened with sucrose, have either shown no association (e.g., cakes and cookies,⁴¹ and sherbert⁴²) or a protective association (e.g., whole-grain cereals, fruit, yogurt and even ice cream^{42–44}) with type 2 diabetes (Appendix 2, available at www.cmaj.ca/lookup/suppl/doi:10.1503/cmaj.160706/-/DC1). An inverse dose-response gradient, similar to that for sucrose, has even been found for whole-grain cereals, fruit and yogurt.^{42–44} Taken together, lack of an adverse association between intakes of total sugars, fructose or sucrose and diabetes may reflect important contributions from these other food sources.

In the absence of a particular adverse association between fructose-containing sugars and incident diabetes, one must consider alternative explanations for the observed association between sugar-sweetened beverages and diabetes. One explanation may relate to uncompensated energy. Systematic reviews and meta-analyses of controlled feeding trials^{45–51} have shown that the adverse effect of sugars on cardiometabolic risk factors is mediated by excess energy, with a signal for harm largely restricted to comparisons in which sugars supplement background diets with excess energy. It is possible that the food form of sugar-sweetened beverages promotes the excess energy intake. Evidence from systematic reviews and meta-analyses of acute preload trials have shown that sugars in liquid form elicit a weaker satiety response and are less compensated by a decrease in energy intake at subsequent meals than sugars in solid form,⁵² a mechanism that might contribute to weight gain and type 2 diabetes.^{53–55} Another possibility is that sugar-sweetened beverages are a marker of an unhealthy lifestyle.^{56–59} High consumers of sugar-sweetened beverages consume more energy, take less physical activity and smoke more,^{60–62} all of which may be difficult to measure and adjust for in observational studies.⁶³

Strengths and limitations

The strengths of our study are that we identified all available prospective cohorts through a systematic search strategy, performed quantitative syntheses and conducted an assessment of the quality and strength of the evidence by using the GRADE assessment.

Despite the inclusion of several large, high-quality cohorts, the inability to rule out residual confounding is a limitation inherent in all observational studies, and a reason that observational

studies start at low quality by GRADE. Sources of residual confounding include reverse causality, the reliability of self-report intake⁶⁴ and measurement of the exposure to sugars, measured and unmeasured confounders included in statistical models, and important collinearity effects from related dietary and lifestyle patterns. Another important limitation is inconsistency between studies. Although the evidence for heterogeneity was partially explained by the removal of several individual studies during sensitivity analyses, residual inconsistency could not be ruled out for total sugars and fructose. A final limitation is the imprecision in the estimates of pooled risk. The 95% CIs were wide and could not rule out clinically important benefit or harm for total sugars and fructose. In addition, there was some instability in the precision of the summary estimates for sucrose.

Balancing the strengths and weaknesses, the evidence was assessed as very low quality for total sugars and fructose, which was based on downgrades for inconsistency and imprecision, and low quality for sucrose, because of the combination of a downgrade for imprecision and an upgrade for an inverse dose-response gradient. In comparison, the evidence for the association between intake of sugar-sweetened beverages and type 2 diabetes^{4,5} would similarly be assessed by GRADE as low quality based on the combination of a downgrade for inconsistency and an upgrade for a positive dose-response gradient.

Conclusion

Our systematic review and meta-analysis of available prospective cohort studies does not support an adverse association between intake of fructose-containing sugars independent of food form and risk of type 2 diabetes. Our confidence in the evidence for this conclusion is generally weak. Sources of uncertainty include the risk of residual confounding in observational studies that prevent causal inferences from being drawn, serious inconsistency between studies and imprecision in estimates of pooled risk for total sugars and fructose, and serious imprecision in estimates of pooled risk for sucrose. Although our observation of a negative dose-response gradient between sucrose and incident diabetes might strengthen our confidence in the lack of harm associated with sucrose, more research is likely to have an important effect on our estimates. In the absence of a clear signal for harm, sugars alone do not appear to explain the relation between sugar-sweetened beverages and type 2 diabetes. More “food-based” research is needed to assess whether the same relation holds for other important food sources of sugars, such as grain and grain-based products, fruit and fruit products, and dairy and dairy products.

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Competing interests: Russell de Souza has served as an external resource person to the World Health Organization (WHO) Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health (guidelines for trans fats and saturated fats), and received remuneration from WHO for travel and accommodation. He also received compensation for contract research conducted for the Institute of Nutrition, Metabolism, and Diabetes at the Canadian Institutes of Health Research (CIHR), Health Canada and WHO. He has received research grants from the Canadian Foundation for Dietetic Research and CIHR, and lecture fees from McMaster Children's Hospital. Vanessa Ha has received research support from WHO. She has received a travel award and doctoral scholarship from CIHR. Alexandra Jenkins is part owner, Vice-President and Director of Research for Glycemic Index Laboratories, Toronto, Ont. She has received research support from the Canadian Diabetes Association (CDA). Thomas Wolever is part owner and President of Glycemic Index Laboratories. Cyril Kendall has received research support from the Advanced Foods and Materials Network, Agricultural Bioproducts Innovation Program through the Pulse Research Network, Agriculture and Agri-Food Canada, Almond Board of California, Barilla, Calorie Control Council, CIHR, Canola Council of Canada, The Coca-Cola Company, The International Tree Nut Council Nutrition Research & Education Foundation, Kellogg, Loblaw Companies Ltd., Pulse Canada, Saskatchewan Pulse Growers and Unilever. He has received consultant fees from American Pistachio Growers; speaker fees from American Peanut Council, Tate & Lyle and The WhiteWave Foods Company; and travel funding from Sabra Dipping Company, Tate & Lyle, International Tree Nut Council Research & Education Foundation, California Walnut Commission, Sun-Maid, The Peanut Institute, General Mills, Oldways Foundation and International Nut and Dried Fruit Council Foundation. He is a member of the Clinical Practice Guidelines Expert Committee for Nutrition Therapy of the European Association for the Study of Diabetes (EASD), the Diabetes and Nutrition Study Group of the EASD and the International Carbohydrate Quality Consortium, and is the Director for the Toronto 3D Knowledge Synthesis and Clinical Trials Foundation. David Jenkins has received research grants from Saskatchewan Pulse Growers, Agricultural Bioproducts Innovation Program through the Pulse Research Network, Advanced Foods and Materials Network, Loblaw Companies Ltd., Unilever, Barilla, Almond Board of California, Agriculture and Agri-Food Canada, Pulse Canada, Kellogg's Canada, Quaker Oats Canada, Procter & Gamble Technical Centres, Bayer Consumer Care (Springfield, New Jersey), PepsiCo/Quaker, International Nut and Dried Fruit Council Foundation, Soyfoods Association of North America, The Coca-Cola Company (investigator initiated, unrestricted grant), Solae, Hain Celestial, Sanitarium Company, Orafiti, International Tree Nut Council Nutrition Research & Education Foundation, The Peanut Institute, Canola Council of Canada, Flax Council of Canada, Calorie Control Council, CIHR, Canada Foundation for Innovation (CFI) and Ontario Research Fund. He has been on the speaker's panel, served on the scientific advisory

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