Consumption of sugar-sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: a systematic review, meta-analysis, and estimation of population attributable fraction

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Abstract

Background Current evidence is insufficient for prospective associations of consuming sugar-sweetened beverages (SSB), artificially-sweetened beverages (ASB), and fruit juice with type 2 diabetes (T2D) for which adiposity is adequately controlled. The population attributable fraction (PAF) for T2D remains unknown.

Objectives To examine these prospective associations before and after adjustment for adiposity; and to estimate PAF for T2D due to SSB consumption in the United States and the United Kingdom.

Data sources and eligibility Prospective studies of non-diabetic adults, published until February 2014, were identified by searching PubMed, EMBASE, OVID, and Web-of-Knowledge. The PAF was estimated in national surveys in the US, 2009-2010 (n=4,729 representing 189.1 million non-diabetic adults) and the UK, 2008-2012 (n=1,932 representing 44.7 million).

Synthesis methods Random-effects meta-analysis and survey analysis for PAF due to SSB consumption.

Results Prespecified information was extracted from seventeen cohorts (38,253 cases/10,126,754 person-years). Higher SSB consumption was associated with higher incidence of T2D by 18% per one serving/day (95% confidence interval=8.8 to 28%, I^2 for heterogeneity=89%) and 13% (5.8 to 21%, I^2=79%) before and after adjustment for adiposity, respectively; for ASB, 25% (18 to 33%, I^2=70%) and 8% (2.1 to 15%, I^2=64%); and for fruit juice, 5% (-1.0 to 11%, I^2=58%) and 7% (0.8 to 14%, I^2=51%). Potential sources of heterogeneity or bias were not evident for SSB. For ASB, publication bias and residual confounding were indicated. For fruit juice, the finding was non-significant in studies ascertaining T2D objectively ($P_{heterogeneity}$=0.008). Under specified assumptions for PAF, of 20.9 million events of T2D predicted to occur over 10 years in the US (absolute event rate=11.0%), 1.8 million would be attributable to SSB consumption (PAF=8.7%, 95% confidence interval=8.3 to 9.2%); and of 2.6 million events in the UK (absolute event rate=5.8%), 79 thousand would be attributable to SSB consumption (PAF=3.6%, 3.3 to 4.0%).

Limitations Residual confounding and limited evidence of causality.

Conclusions Habitual SSB consumption was associated with greater T2D incidence, independently of adiposity. Although ASB and fruit juice also showed positive associations with T2D incidence, the findings were likely to involve bias. Nonetheless, both ASB and fruit juice were unlikely to be healthy alternatives to SSB for the prevention of T2D. Under assumption of causality, SSB consumption over years may be related to a substantial number of cases of new-onset diabetes.

INTRODUCTION

Health effects of sugar-sweetened beverages (SSB), artificially sweetened beverages (ASB) and fruit juice have received considerable attention from scientific and public communities. SSB consumption is likely to contribute to the obesity epidemic and development of type 2 diabetes (T2D).[1–5] ASB and fruit juice are candidate alternatives to SSB, but their prospective associations with T2D have not been well established yet, because only a few studies have examined the associations, of which potential bias has been debated.[5–9]

Each of these beverages has been investigated and reviewed for prospective associations with incident T2D.[4–9] However, some reviews assessed evidence qualitatively.[3,6–8] A few meta-analyses were available, but one aggregated studies that adjusted for obesity status and studies that did not[4,9], and the other separated such studies *ad hoc* only for SSB, but not for ASB or fruit juice.[5] The influence of adiposity is crucial to better characterise, because obesity can be a mediator by directly causing T2D and, thus, mediating an association of SSB consumption with T2D; and because obesity can be a confounder by altering dietary habits and confounding an association of beverage consumption with incident T2D.[8,10] Previous studies indeed reported that obese individuals tend to consume more SSB, more ASB, and less fruit juice than leaner individuals.[6,10,11] Moreover, despite the growing interest in a policy intervention to reduce SSB consumption at a population level[12–14], no study has translated a prospective association of SSB consumption with T2D into a measure of its population-level impact, including population attributable fraction (PAF), in a contemporary population.

To fill the gaps in knowledge, we first conducted a systematic review and meta-analysis of prospective studies to test whether or not habitual consumption of each of SSB, ASB, and fruit juice would be associated with T2D incidence. We specifically aimed to meta-analyze the associations with and without adjustment for adiposity, because the association may be both mediated and also confounded by adiposity. Second, to provide policy-relevant measures, we used the meta-analysis result for SSB to estimate PAF for 10-year risk of T2D due to SSB consumption in contemporary populations of the United States (US) and the United Kingdom (UK), where approximately half of each population consumed SSB in recent years.[1,10]

METHODS

Study searches and selection

Following the PRISMA guidelines[15] and the protocol (not registered, available upon request), relevant studies were identified through hand searches and systematic searches of four databases on May 31, 2013 (updated on 10 February 2014): PubMed, EMBASE, OVID, and Web-of-Knowledge. Search terms included those related to types of beverages, diabetes, and prospective-study design (Supplementary Text for details). Time and language of publications were not restricted. After duplicates were removed, articles were screened based on titles and abstracts by one author (FI) and independently reviewed in duplicate by the authors (FI, LO'C, and ZY). Eligibility criteria were a prospective design, assessment of beverage consumption and incident T2D, and recruitment of adults free of diabetes and aged 18 years or older. Follow-up of at least two years on average was also considered, because diabetes incidence could alter approximately two years after lifestyle modification.[16,17]

Data extraction and quality assessment

Information was extracted in a standardised manner in duplicate, including baseline demographics, body-mass index, duration of follow-up, exclusion criteria, sample sizes, loss to follow-up, assessments of beverage consumption and incident T2D, types of beverage consumption, measures of prospective associations and the 95% confidence intervals (CI), covariates evaluated, and sources of funding. We extracted measures of associations that were the most adjusted for sociodemographic and lifestyle factors, with and without further adjustment for adiposity measures. Influence of adjustment for total energy intake would be interesting,[4,6] but this meta-analysis used estimates adjusted for total energy whenever possible, for parsimony and possible reduction of confounding and measurement errors by energy adjustment.[18] We extracted estimates stratified by age, sex or adiposity measures, if reported, to use in analysis of heterogeneity. We extracted information from relevant articles of identified cohorts to obtain additional information related to study design and quality.

We contacted authors of identified articles to request additional information. We requested additional information if an article did not report two types of estimates before and after adjustment for adiposity, based on either categorical or continuous analysis for SSB, ASB, and fruit juice separately. When we contacted authors, we requested estimates before and after adjustment for adiposity based on both continuous and categorical variables of each beverage consumption; and requested estimates based on longer follow-up if available. In addition, we

contacted authors of cohorts that did not meet eligibility criteria but could be eligible by providing additional information (Table S1).

Risks of bias were examined in concordance with the Cochrane Collaboration's tools, including A Cochrane Risk Of Bias Assessment Tool: for Non-Randomized Studies of Interventions, ACROBAT-NRSI).[19–21] The following seven domains of bias were rated *post hoc* as 'high', 'low', or 'unknown' risk of bias (see Supplementary Text for considerations)[20,21]: confounding, selection, exposure measurement, misclassification over time, missing data, outcome measurement, and selective reporting. As an example, bias specific to this meta-analysis included likelihood of misclassifying sugar-sweetened fruit drink to fruit juice (e.g. fruit punch). We tested influence of these sources of bias in sensitivity analyses. Bias related to exposure and outcome measurements were incorporated quantitatively to meta-analysis (see below and Supplementary Text). Overall quality of evidence was assessed based on study quality, results from sensitivity analysis, and principles of the Grades of Recommendation, Assessment, Development and Evaluation (GRADE).[22]

Meta-analysis

We used Stata13.1 (Stata Inc., Texas, US) for analyses (α two-side=0.05, unless indicated). Statistical details are described in Supplementary Text. Each of SSB, ASB and fruit juice was considered as the main exposure. SSB was defined as any sweetened beverages not presented as diet or non-caloric beverages, including sugar-sweetened fruit juice. ASB included low-caloric soft drinks as reported in each study. Fruit juice was defined as 100% fruit juice or fruit juice assessed separately from fruit drinks. Measures of associations were standardised to relative risk (RR) per one serving/day of beverage consumption, after we confirmed this unit was used most frequently across studies. Because volume per serving was specific to a population, ranging from 237 ml (1 cup) to 355 ml (12 oz) (median across publications=250 ml/day), we repeated meta-analysis to estimate RR per 250 ml/day. Odds ratios, if reported, were converted to RR.[23] If a study reported categorical estimates only, RRs for categories were combined in a single dose-response estimate.[24] If only stratified estimates were reported, they were merged by fixed-effects meta-analysis to derive a cohort-specific estimate assuming consistency of associations within a cohort.

We performed random-effects meta-analysis as pre-specified, assuming that biological effects of beverages in different populations would vary randomly at least by processing and composition of beverages. The heterogeneity of associations was expressed by I^2.[25] For each of SSB, ASB, and fruit juice, we estimated RRs before

and after adjustment for adiposity measures. We additionally estimated crude RR without any adjustment to assess a magnitude of overall confounding. Non-linear associations were evaluated by cubic spline meta-regression for which we used available categorical estimates.[24]

In observational studies, within-person variability of exposure can cause bias.[26–30] As performed previously[26–32], we compiled within-person variation of beverage consumption in each study and adjusted for them to estimates of each study. Uncertainty in self-reported diagnosis of T2D was also calibrated for estimates from studies without objective information on T2D incidence.[33] Estimates after study-specific calibration were pooled to compute RR adjusted for within-person dietary variation and uncertainty of T2D ascertainment.

We assessed potential sources of heterogeneity of associations across studies, using meta-regression. Publication status (published or not) was included *post hoc* as a potential source of heterogeneity. Stratified meta-analysis was performed by each variable identified as a significant source of heterogeneity ($P<0.01$) and by pre-specified variables: age, sex, body-mass index (BMI), and location of study.

Publication bias was assessed by Egger's test, with a contour-enhanced funnel plot, and trim-and-fill analysis.[34] If trim-and-fill indicated publication bias, we adjusted summary estimates for the bias.[34] Robustness of summary findings were examined by sensitivity analyses: influence analysis,[32] fixed-effects meta-analysis, analysis using ml/day as a unit, analysis without studies with a high overall risk of bias, and analysis incorporating measures of uncertainty in adjustment for within-person dietary variations and T2D diagnosis.[35]

Adiposity is likely to confound an association of beverage consumption with T2D, particularly in research on ASB.[6,8,10,11] Because measurement of adiposity is imperfect in a large cohort study,[36] an association of beverage consumption with T2D incidence is subject to residual confounding, as discussed previously.[3,6,32,37–41] Therefore, to assess if such confounding would lead to a false-positive conclusion, we performed *post hoc* simulation analysis to examine influence of the bias.[42]

Type 2 diabetes risk attributable to SSB consumption in the United States and the United Kingdom

We estimated T2D risks attributable to SSB consumption over ten years in the US and the UK.[22,43] These countries contributed to the meta-analysis to the large extent and provided publically-available national data on diets and risk factors for T2D: the US National Health and Nutrition Examination Survey, 2009-2010[44]; and the UK National Diet and Nutrition Survey, 2008-2012[45]. The recent cycle was selected for greater generalisability to recent

populations. Selecting adults aged ≥20 years and without diabetes based on medical history, drug use, and biological measures (glucose and haemoglobin A1c), we analyzed 4,729 US adults who represented 189.1 million; and 1,932 UK adults who represented 44.7 million.

PAF was estimated, by applying an algorithm of the Cochrane Collaboration to survey data.[22,43] We first estimated habitual SSB consumption based on 24-hour recalls in US and 4-day food records in UK. Then, we estimated a 10-year T2D risk based on a risk-prediction algorithm developed and validated in each country.[46,47] The predicted T2D risk for each individual was considered as a 'assumed control risk' (ACR)[22] if the current SSB consumption would remain constant. Then, we calculated an alternative T2D risk for each individual if the SSB consumption would become zero, calculating ACR×(1/RR per serving/day)×observed SSB servings/day. The difference between the two risk estimates represented a risk attributable to SSB consumption. Using the risk estimates, sampling weights, and a population size, we estimated the absolute numbers of events over 10 years, events attributable to SSB consumption ('absolute risk reduction'[22]), and PAF (the proportion of events attributable to SSB consumption). The estimation assumed causality and no change in individuals' characteristics over time. Validation of 10-year risk prediction was performed in the US survey, predicting diabetes prevalence in 2009-2010 by using data collected in 1999-2000. Further details are presented in Supplementary Text.

RESULTS

We identified 1937 articles after removing duplicates, reviewed 33 articles in full text, and identified 21 articles from 16 cohorts as eligible for this meta-analysis (Figure S1). From one cohort which publication did not meet eligibility criteria, we obtained unpublished information (Table S1).[22] Finally, data from 17 cohorts were evaluated (Table 1)[11,39–41,48–64] comprising 38253 T2D cases over 10,126,756 person-years in total. No study or publication was funded by industry.

Quality of the studies has been examined (Table S2; Supplementary Text). Methods of assessing diets, methods of ascertaining T2D, and validity of these measurements varied across studies (Table 1, Tables S3). We considered six cohorts having potential bias in quantitative results based on at least one of the following reasons: publication of a conference abstract only[61]; exclusion of participants lost in follow-up[56,62]; likelihood of substantial residual confounding[40]; and no separation between fruit juice and SSB (fruit drinks) or between SSB and ASB[56,63]. Subtypes of each of SSB, ASB, and fruit juice were not assessed in any studies, except separating SSB and ASB by caffeine content.[48] Selective reporting might exist in some studies[39,51,54,57,61,63], but not considered as a source of bias, for example, reporting only non-quantitative results for SSB in a study mainly on ASB.[39]

Confounding was likely to exist in all of the studies. As would be expected, consumers of ASB tended to be overweight or obese or hypertensive.[11,39,49,58,65] In longitudinal analysis, all studies statistically adjusted for potential confounders including socio-demographic variables, clinical factors (family history of diabetes or prevalent diseases), and lifestyle factors including a diet (Table S4). None of these factors was identified as a single cause of confounding, according to studies assessing influence of potential confounding in different regression models.[11,41,48–50,53,58,60,62–64] However, a combination of multiple factors was likely to cause confounding (Table 2, Table S4). After adjustment for multiple potential confounders, RR for SSB was attenuated from 1.25 to 1.18 (32% change); and for ASB, 1.48 to 1.25 (43%). By contrast, the point estimate for fruit juice was shifted upward, from 0.97 to 1.05.

Beverage consumption and type 2 diabetes

Findings from meta-analysis are summarised in Table 2. Higher consumption of SSB by one serving per day was associated with 18% greater incidence of T2D (95% CI 8.8 to 28%; I^2=89%) before adjustment for adiposity (Figure 1; Table 2). When adjusted potential mediation and confounding by adiposity, the association was attenuated, with higher incidence by 13% per serving/day (5.8 to 21%; 79%). In the analysis of ASB, in which

adiposity is unlikely to be a mediator, higher consumption of ASB by one serving/day was associated with 25% greater incidence of T2D (95% CI 18 to 33%; I^2=70%) before adjustment for adiposity measures. After the adjustment, the estimate of 25% greater incidence was attenuated to 8% (2.1 to 15%).

In the analysis of fruit juice, the influence of adjustment for adiposity was in the direction opposite to analysis of SSB and ASB. The association of fruit juice consumption with incident T2D was strengthened after adjustment for adiposity measures. Higher consumption of fruit juice by one serving/day was associated with 7% greater incidence of T2D (95% CI 0.8 to 14%).

Each of the beverages showed significant non-linear associations (*P*>0.05) (Figure S2). Calibration for within-person variation strengthened the association between each type of beverages and incident T2D (Figure 1, Table 2). For example, RR (95% CI) per one serving/day of SSB was strengthened from 1.13 (1.06 to 1.21) to 1.28 (1.12 to 1.46). Estimates for ASB were strengthened similarly. The influence was small for fruit juice, where RR was shifted only slightly from 1.07 (1.01 to 1.14) to 1.10 (1.01 to 1.20).

Sensitivity analysis and quality of evidence

None of the study-specific factors evaluated could explain heterogeneity of results for SSB and ASB (*P*>0.1) (Table S5). The results of fruit juice varied by study design. While studies assessing self-reported T2D only showed the positive association, the significant association disappeared in studies ascertaining T2D incidence by medical records or blood glucose or glycated haemoglobin (RR 1.08, 95% CI 0.97 to 1.20) (*P* heterogeneity=0.008). Additionally, studies with repeated measures of diets supported a null (0.98, 0.86 to 1.11; *P* heterogeneity=0.068). These factors of study design explained heterogeneity of the association, reducing I^2 from 29% to 0%. Demographic variables and BMI were not significant sources of heterogeneity (*P*>0.14 each), whereas each of SSB, ASB, and fruit juice was not significantly associated with T2D in studies recruiting more men than women or in Asia, with a fewer number of studies than the main analysis (Table S5).

Publication bias was not evident by Egger's test (*P*>0.05), except for fruit juice (*P*=0.03), where estimates with the greater precision showed stronger associations (Figure 2). Trim-and-fill indicated publication bias for SSB and ASB (Table 2). In particular, publication bias could influence inference for ASB. With adjustment for adiposity, RR (95% CI) per one serving/day of ASB was 1.29 (1.08-1.54) before calibration for publication bias and 1.22 (0.98 to 1.52, 64%) after the calibration.

Contour-enhanced funnel plots indicated findings for ASB and fruit juice were not stable (Figure 2). For example, if a study with RR 0.5 contributed, associations of ASB and fruit juice with T2D incidence would not be significant. In influence analysis, positive associations persisted for SSB and ASB (Figure S3), whereas a significant result of fruit juice was not seen after excluding any single studies supporting the positive association.

The results varied little by methodological assumption (Table S6), using estimates per 250 ml/day (median of 17 studies), not serving/day; excluding studies with a high risk of potential bias or with relatively large within-person dietary variability; and incorporating uncertainty of within-person variability and precision of T2D diagnosis. When we examined potential influence of residual confounding by measured adiposity, bias toward the null would appear substantial for ASB (Figure S4). Under realistic assumption of correlation=0.80 between measured and true adiposity[36], the association of SSB was attenuated by 26% to be RR 1.20 (1.04 to 1.38); of ASB, by 96%, 1.01 (0.81 to 1.25); and fruit juice, strengthened by 19%, 1.12 (1.03 to 1.22).

We rated quality of evidence for each of SSB, ASB, and fruit juice. We rated 'moderate' quality for SSB, because the main findings were likely to be robust against different sources of bias, despite observational design. 'Low' quality was assigned each for ASB and fruit juice. Findings for ASB were subject to publication bias and residual confounding; and for fruit juice, concern of stability of the positive association was present.

Type 2 diabetes risk attributable to SSB consumption

Proportions of consumers were 54.4% in US and 49.4% in UK. Of a total population, (mean±SD) of SSB consumption were 284±412 g/day in US and 114±157 g/day in UK (Figure 3, Table S7). Absolute event rates over 10 years from 2010 were estimated to be 11.0% in US (20.9 million events) and 5.8% in UK (2.6 million events). Assuming a causal effect of SSB consumption partly mediated by obesity status (adiposity unadjusted), SSB consumption in US would cause 2.6 million excess events of T2D over 10 years (PAF=11.9%; 95% CI=11.3 to 12.6%); and in UK, 126 thousand excess events of T2D (PAF=4.9%; 4.3 to 5.4%). Assuming a causal effect of SSB consumption independent of obesity status (adiposity adjusted), SSB consumption would cause 1.8 million excess events in US (PAF=8.7%; 8.3 to 9.2%) and 79 thousands excess events in UK (PAF=3.6%; 3.3 to 4.0%). Younger adults and men would have greater numbers of T2D events related to SSB consumption than older adults and women, respectively (Figure 3, Table S9).

DISCUSSION

We produced summary evidence that habitual consumption of each of SSB, ASB, and fruit juice was prospectively associated with incident T2D, independently of adiposity. Sensitivity analyses consistently supported the positive association of SSB with incident T2D. In contrast, the association of each of ASB and fruit juice with incident T2D was less evident. For ASB, potential publication bias and residual confounding were likely to exist. For fruit juice, the finding appeared to be not stable and was sensitive to study design. Under assumption of causality for the association of SSB with T2D incidence, we estimated that two millions of T2D events in US and 80 thousands of T2D cases in UK over 10 years would be related to SSB consumption.

Strengths and limitations

Limitations typical of observational studies and meta-analysis are present. Although this study has strength of assessing influence of confounding and providing results adjusted for potential confounders, residual confounding by many other factors could exist.[37] Confounding by socioeconomic and dietary factors were not detected to be strong in published studies. However, these variables are likely to have been measured with errors and have caused residual confounding in individual studies and our meta-analysis. Additionally, lifestyle factors and adiposity could change over time. The time-varying characteristics might not be random and could cause bias in an unknown direction and cause insufficient adjustment for adiposity during the follow-up. Reverse causality could also exist, because co-morbid conditions and health consciousness might alter consumption of beverages, particularly ASB, and risks of T2D. Weakness of meta-analysis includes exclusion of eligible cohorts by lack of information. Our meta-analysis included statistical approximation that might involve errors. For example, we derived dose-response estimates partly from categorical estimates and odds ratios. Without such approximations, analysis standardised across different cohorts is of future interest to characterise associations of different beverages with risks of T2D.

This study has a strength of estimating PAF for T2D risks due to SSB consumption in US and UK, using individuals' data on beverage consumption. As population-based measures, effects of taxation of SSB on obesity and T2D incidence have been modelled previously.[12–14] No study combined SSB consumption observed in multiple populations, T2D risk predicted by a validated algorithm, and quantitative evidence on association of SSB consumption with T2D incidence. Future comparison between available estimates is worthwhile to characterise efficacy and effectiveness of policy interventions in different settings. Nonetheless, limitations of PAF should be

appraised. First, causality was assumed, although it has not been established. Second, estimates were under assumptions of no change in lifestyle potentially associated with reducing SSB consumption. Thus, our estimates should be considered as efficacy of reducing SSB, rather than effectiveness. Third, generalisability remains unknown, for example, to Central and South America where the highest per-capita sales of SSB in the world have been recorded; and China and India where the highest prevalence of T2D is expected.[1,66] To address limitations typical of observational research and also needs for a policy intervention in different populations, future research should include a randomised trial examining people's health and behaviours and also a trial examining population impact.

Interpretation in relation to other studies

Other quantitative reviews have been published recently.[4,5,9] None of them quantified PAF. One meta-analysis evaluated influence of adiposity on the association of SSB based on three studies, but not ASB or fruit juice.[5] For SSB, although the main conclusion was similar, we evaluated greater numbers of T2D cases (38,285 vs 19,054) and studies (17 vs 3), including bias assessments and sensitivity analyses. For fruit juice and ASB, we provided summary estimates based on a greater number of studies than previous work (9 vs 4 and 12 vs 4, respectively), by which we could assess influences of adiposity and potential bias. The assessment appeared to be important to indicate potential false-positive findings for ASB and fruit juice.

Plausibility of our findings deserves discussion. Detrimental effects of SSB independent of obesity may exist. Sugars in SSB acutely elevate blood glucose, with a high glycemic index (GI) (80 to 110/100 of white bread) and can elevate T2D risk.[67,68] Independent of the glycaemic and caloric effects, fructose promotes hepatic lipogenesis and further insulin resistance.[2] Effects of caramels for browning[69], caffeine[48,70], phosphoric acid[71], and other constituents have also been suggested. These non-glycaemic effects may be present in ASB, if ASB truly elevates a risk of T2D. ASB might have effects on hormones, microbiota, and taste preference, but these have not been established yet.[6,72,73] Adverse effects of fruit juice would be present, because of its moderately high GI (50-80).[67] Healthful constituents may exist, but decrease during processing.[7,74] This loss explains the discordance between our finding and the reported inverse association of fruit consumption with T2D.[75]

Our analyses indicated a false-positive association of ASB with T2D because of possible publication bias. The bias would be expected by existing public interest over their health effects.[6,76] Residual confounding in the

finding for ASB is also plausible because adults at high risk of T2D preferentially consumed more ASB.[5,6,8,10,11,36] By contrast, confounding in the opposite direction was found in our analysis of fruit juice. This is consistent with observations that leaner, lower-risk adults consumed more fruit juice.[10,11] These observations provide research and clinical implications for better understanding of health-seeking behaviours related to beverage consumption.[6,77]

Clinical and public health implications

Although causality has not been established, our findings and available evidence indicate a benefit of lowering SSB consumption for the primary prevention of T2D. In the same context, our findings also imply that consumption of ASB or fruit juice is unlikely to reduce the T2D risk, and these should not be considered as a healthy option of beverages. However, consuming ASB to lower caloric intake and body weight may have clinical benefit for obese or overweight adults.[6,77] This effect on weight should be considered separately from our study that could not rule out the effect of weight on beverage consumption.[8] In addition, clinical applications of our finding deserve further appraisal about the effects of altering beverage consumption on changes in lifestyle behaviours and on risks of other clinical outcomes.[3,6,8]

Our findings have strong public health implications. Despite the aforementioned limitations, current SSB consumption was estimated to cause approximately two million excess events of T2D in US over 10 years; and 80 thousand excess events, in UK. This could cost nearly £12.0 billion in US and £206 million in UK ($9,800 in US and $3,994 in UK per patient[66] and $1=£0.66 as of 24 January 2015). For future implementation of a policy-based intervention to reduce SSB consumption,[12,13] our estimate of efficacy should be extended to estimates of effectiveness of interventions of reducing SSB, accounting for practical issues in interventions and effects on obesity, T2D risk, and lifestyle change associated with reduction of SSB consumption.[8,77] Despite PAF of no more than 15%, estimates of efficacy and effectiveness are crucial, as 535 million adults are estimated to have T2D in 2035.[1,66] Additionally, the PAF informs that an intervention reducing SSB only would not reduce a large amount of events, and thus confirms importance of multiple modifiable risk factors, rather than a single dietary component, for the prevention of T2D. For ASB and fruit juice, our findings inform little benefit of using them as an alternative to SSB. In addition, fruit juice consumption should not be a part of dietary recommendations for greater consumption of fruits and vegetables, as guided to limit fruit juice consumption among children.[7,78]

Conclusions

In conclusion, observational cohort studies support that consumption of SSB is associated with incident T2D independently of adiposity. This finding was robust against many epidemiological concerns. By contrast, although ASB and fruit juice showed a positive association with incident T2D, potential bias and heterogeneity by study design limit quality of evidence. Although causality has not been established, the available evidence justifies an intervention to reduce SSB consumption in a population level. Moreover, findings support neither ASB nor fruit juice to be alternatives to SSB for the prevention of T2D.

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FI and NGF conceived the study. FI, LO'C, YZ, and NGF designed the study. FI, LO'C, and YZ undertook literature search and data extraction. FI, JM, YH, and SNB did data analysis. FI, LO'C, YZ, and NGF interpreted data. FI developed the first draft. All authors provided critical comments and approved the final version. We affirm that the manuscript is an honest, accurate, and transparent account of the study being reported; that no important aspects of the study have been omitted; and that any discrepancies from the study as planned have been explained as post hoc analysis. Data sharing is available from the corresponding author. We acknowledge the following contributors for provision of additional information

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Figure Legend:

Figure 1. Prospective associations of beverage consumption with incident type 2 diabetes: random-effects meta-analysis. A) sugar-sweetened beverages, B) artificially sweetened beverages, and C) fruit juice were evaluated to estimate relative risks (RR) and 95% confidence intervals (CI) unadjusted for adiposity (Left), adjusted for adiposity (Middle), and adjusted for adiposity and within-person variation (Right). Cohorts were ordered by weights in the most adjusted model. Estimates with 95% CI>10 are not presented.

Figure 2. Funnel plot for associations of sugar-sweetened beverages, artificially sweetened beverages, and fruit juice with incident type 2 diabetes. Dots represent point estimates plotted over precision measures (1/standard error). Estimates outside each panel are not presented. Horizontal lines represent summary estimates and 95% CI across precision. Gray areas represent any of a single estimate that, if included, would make the summary estimate insignificant (P>0.05). P-values by Egger's test are presented: for fruit juice, estimates with greater precision indicated stronger positive association.

Figure 3. Consumption of sugar-sweetened beverages (SSB) and population attributable fraction (PAF) for type 2 diabetes (T2D) in the United States and the United Kingdom. Each diamond represents mean of SSB consumption (left axis) and each bar represents PAF (%) for T2D due to SSB consumption (right axis). Absolute event rates over 10 years were 11.0% in the United States (20.9 million events) (left) and 5.8% in the United Kingdom (2.6 million events) (right).

Table 1. Characteristics of prospective cohort studies included in meta-analysis on associations of consuming sugar-sweetened beverages, artificially-sweetened beverages and fruit juice with incidence of type 2 diabetes (T2D).

Cohort, country*	Baseline years	Median follow-up, years	N	Age, range/ mean, year	Men, %	BMI, mean, kg/m^2	Beverage consumption†		Incident T2D§	
							Method of assessment	Types	Method of ascertainment	N cases (rate/1,000)§
FMCHES, Finland[52]	1966-1972	13.7	4,304	40-69	53.1	26.5	Diet history	SSB	Records	175 (3.0)
NHS I, US[41,50]	1984	21.1	74,513	40-69	0	23.6	FFQ†	SSB, ASB, FJ	Self-report	7,300 (4.6)§
KIHD, Finland[53]	1984-1989	18.9	2,481	42-60	100	26.8	4-d diet record	SSB‡	Records, biomarkers	506 (10.8)
CARDIA, US[54,55]‖	1985-1986	18.8	2,160	18-30	46.5	24.5	Diet history†	SSB, ASB, FJ	Self-report, records, biomarkers	174 (4.3)§
HPFS, US[41,48,49]	1986	19.3	40,290	40-75	100	25.5	FFQ†	SSB, ASB, FJ	Self-report	3,229 (4.2) §
Iowa WHS, US[61] ‖	1986	10.7	31,489	55-69	0	27.0	FFQ	SSB, FJ	Self-report	999 (3.0)§
ARIC men, US[63]	1987-1989	7.5	5,414	45-64	100	27.2	FFQ	SSB‡	Self-report, biomarkers	718 (17.7)
women, US[63]	1987-1989	7.7	6,790	45-64	0	27.2	FFQ	SSB‡	Self-report, biomarkers	719 (13.8)
JPHC men, Japan[51] ‖	1990	9.8	12,137	40-59	100	23.5	FFQ†	SSB, FJ	Self-report	397 (3.3)§
women, Japan[51] ‖	1990	9.9	15,448	40-59	0	23.5	FFQ†	SSB, FJ	Self-report	279 (1.8)§
FOS, US[64]	1991	12.1	2,736	54.2	45.5	26.7	FFQ†	SSB, ASB, FJ	Records, self-report, biomarkers	303 (9.1)
NHS II, US[41,59,60]	1991	18.4	90,423	24-44	0	24.4	FFQ†	SSB, ASB, FJ	Self-report	5,121 (3.1)§
EPIC-InterAct, eight European countries[11]	1991-1998	11.7	27,058	52.4	37.8	26.0	FFQ	SSB, ASB, FJ	Records, biomarkers§	11,684 (2.9)
E3N, France[40] ‖	1993	12.4	48,985	52.8	0	22.8	Diet history	SSB, ASB, FJ	Records	1,054 (1.7)
SCHS, Singapore[56]‖	1993-1998	5.7	43,580	45-74	42.9	23.0	FFQ	SSB, FJ	Self-report, records, biomarkers	2,250 (9.0)§
Black WHS, US[57]	1995	7.7	43,960	21-69	0	27.6	FFQ	SSB, ASB, FJ	Self-report	2,550 (7.5)§
HIPOP-OHP, Japan[62]	1999	3.4	6,121	19-69	78.9	22.6	FFQ	SSB, FJ	Self-report, records, biomarkers	212 (10.2)
MESA, US[39]	2000-2002	5.8	5,011	45-84	47.4	27.9	FFQ	SSB, ASB	Self-report, records, biomarkers	413 (14.3)
Occupational cohort, Japan[58]	2003	5.5	2,037	35-55	100	23.3	FFQ	SSB, ASB, FJ	Records, biomarkers	170 (15.1)

Abbreviations: ASB, artificially sweetened beverages; ARIC, Atherosclerosis Risk in Communities Study; CARDIA, Coronary Artery Risk Development in Young Adults Study; EPIC, European Prospective Investigation into Cancer and Nutrition Study; FFQ, food frequency questionnaires; FJ, fruit juice; FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study; HIPOP-OHP, High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHD, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; SCHS, Singapore Chinese Health Study; SSB, sugar-sweetened beverages; WHS, Women's Health Study.

* Ordered in the years of baseline assessments. Numbers represent citations. ARIC and JPHS reported results stratified by sex. In meta-analysis, the sex-stratified estimates were aggregated in advance.
† Diets were assessed repeatedly during the follow-up and incorporated in longitudinal analysis. JPHC measured repeatedly but used the baseline FFQ only.
‡ In ARIC and FOS, SSB and ASB were combined together in their analyses. In KIHD, the article described fruit juices, but treated as SSB, because more than 90% of fruit juice consumed in Finland was sweetened with sugars in 1980s and 1990s (confirmed by the authors).
§ Biomarkers included any of fasting glucose, 2-hour glucose by oral-glucose tolerance test, and glycosylated haemoglobin. Records included medical records or other records from registry, not including self-reported information. Studies ascertaining T2D cases by self-report involved uncertainty in the ascertainment, and thus the numbers of cases were revised by a positive predictive value (a proportion of verified cases among self-reported cases) (Supplementary Text, Table S3). The EPIC-InterAct study adopted different methods across participating cohorts, in which no cohort used self-reported diagnosis only.
‖ Considered as having potential bias (Supplementary Text, Table S2). In each of CARDIA and JPHC, distinct analytic approaches were undertaken in different publications by the same authors. Iowa WHS reported results only in a conference abstract. E3N presented prospective associations adjusted for crude categorical variables for BMI, which could cause substantial residual confounding, while the other studies used BMI as a continuous covariate in statistical adjustment. SCHS presented results without classification between 100% fruit juice and sugar-sweetened fruit juice and had likelihood of attrition bias by loss of follow-up by deaths (15%).

Table 2. Associations of consuming sugar-sweetened beverages, artificially-sweetened beverages and fruit juice with incident type 2 diabetes: meta-analysis of prospective cohort studies.

Beverages (n cohorts) and models of meta-analysis*	Not adjusted for adiposity† RR (95% CI)	I^2, %	Adjusted for adiposity† RR (95% CI)	I^2, %
Sugar-sweetened beverages (n=17)				
Meta-analysis, crude	1.25 (1.14 to 1.37)	89		
+ multivariable-adjusted	1.18 (1.09 to 1.28)	89	1.13 (1.06 to 1.21)	79
+ calibration for information bias	1.43 (1.20 to 1.70)	86	1.28 (1.12 to 1.46)	73
+ calibration for publication bias	1.42 (1.19 to 1.69)	85	1.27 (1.10 to 1.46)	73
Artificially sweetened beverages (n=10)				
Meta-analysis, crude	1.48 (1.35 to 1.62)	85		
+ multivariable-adjusted	1.25 (1.18 to 1.33)	70	1.08 (1.02 to 1.15)	64
+ calibration for information bias	2.13 (1.57 to 2.88)	72	1.29 (1.08 to 1.54)	50
+ calibration for publication bias	1.81 (1.33 to 2.47)	76	1.22 (0.98 to 1.52)	64
Fruit juices (n=13)				
Meta-analysis, crude	0.97 (0.90 to 1.06)	79		
+ multivariable-adjusted	1.05 (0.99 to 1.11)	58	1.07 (1.01 to 1.14)	51
+ calibration for information bias	1.06 (0.98 to 1.14)	49	1.10 (1.01 to 1.20)	29
+ calibration for publication bias	not detected		not detected	

* The number of cohorts varied slightly by models (see Figure 1). Crude meta-analysis pooled estimates without any adjustment. Multivariable-adjusted model pooled estimates adjusted for demographic and lifestyle covariates. Calibration for information bias accounted for within-person variation for dietary consumption and imprecise ascertainment of self-reported diabetes. Calibration for publication bias was carried out, if indicated in trim-and-fill analysis.

† Relative risk (RR) and 95% confidence intervals (CI) per serving/day before and after adjustment for adiposity. All RRs were statistically significant (P<0.05), except for ASB after adjustment for publication bias (P=0.07).

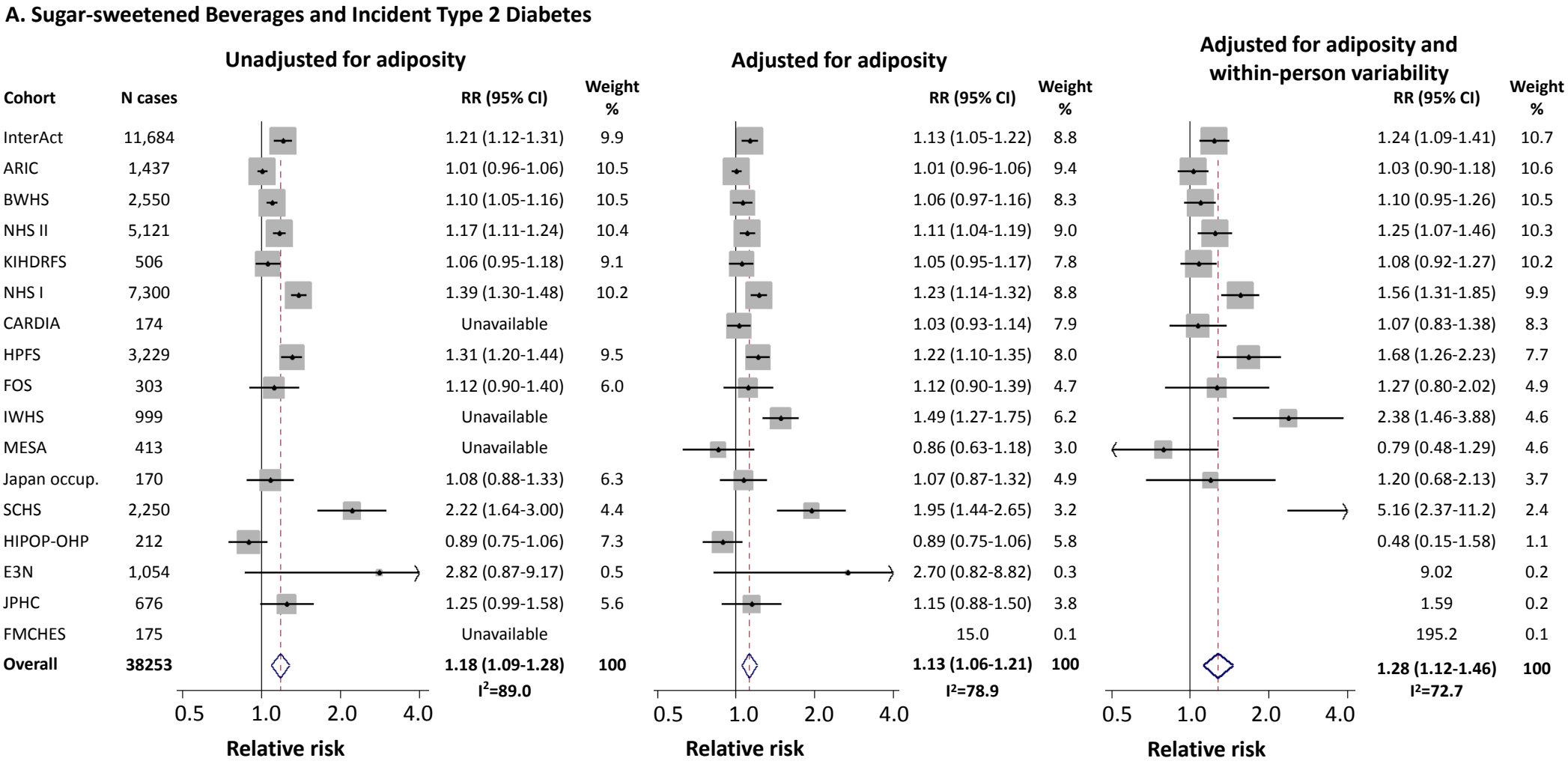
A. Sugar-sweetened Beverages and Incident Type 2 Diabetes
Unadjusted for adiposity
Adjusted for adiposity
Adjusted for adiposity and within-person variability
Cohort
N cases
RR (95% CI)
Weight %
InterAct 11,684 1.21 (1.12-1.31) 9.9 1.13 (1.05-1.22) 8.8 1.24 (1.09-1.41) 10.7
ARIC 1,437 1.01 (0.96-1.06) 10.5 1.01 (0.96-1.06) 9.4 1.03 (0.90-1.18) 10.6
BWHS 2,550 1.10 (1.05-1.16) 10.5 1.06 (0.97-1.16) 8.3 1.10 (0.95-1.26) 10.5
NHS II 5,121 1.17 (1.11-1.24) 10.4 1.11 (1.04-1.19) 9.0 1.25 (1.07-1.46) 10.3
KIHDRFS 506 1.06 (0.95-1.18) 9.1 1.05 (0.95-1.17) 7.8 1.08 (0.92-1.27) 10.2
NHS I 7,300 1.39 (1.30-1.48) 10.2 1.23 (1.14-1.32) 8.8 1.56 (1.31-1.85) 9.9
CARDIA 174 Unavailable 1.03 (0.93-1.14) 7.9 1.07 (0.83-1.38) 8.3
HPFS 3,229 1.31 (1.20-1.44) 9.5 1.22 (1.10-1.35) 8.0 1.68 (1.26-2.23) 7.7
FOS 303 1.12 (0.90-1.40) 6.0 1.12 (0.90-1.39) 4.7 1.27 (0.80-2.02) 4.9
IWHS 999 Unavailable 1.49 (1.27-1.75) 6.2 2.38 (1.46-3.88) 4.6
MESA 413 Unavailable 0.86 (0.63-1.18) 3.0 0.79 (0.48-1.29) 4.6
Japan occup. 170 1.08 (0.88-1.33) 6.3 1.07 (0.87-1.32) 4.9 1.20 (0.68-2.13) 3.7
SCHS 2,250 2.22 (1.64-3.00) 4.4 1.95 (1.44-2.65) 3.2 5.16 (2.37-11.2) 2.4
HIPOP-OHP 212 0.89 (0.75-1.06) 7.3 0.89 (0.75-1.06) 5.8 0.48 (0.15-1.58) 1.1
E3N 1,054 2.82 (0.87-9.17) 0.5 2.70 (0.82-8.82) 0.3 9.02 0.2
JPHC 676 1.25 (0.99-1.58) 5.6 1.15 (0.88-1.50) 3.8 1.59 0.2
FMCHES 175 Unavailable 15.0 0.1 195.2 0.1
Overall 38253 1.18 (1.09-1.28) 100 1.13 (1.06-1.21) 100 1.28 (1.12-1.46) 100
I²=89.0 I²=78.9 I²=72.7
0.5 1.0 2.0 4.0
Relative risk

Figure 1A

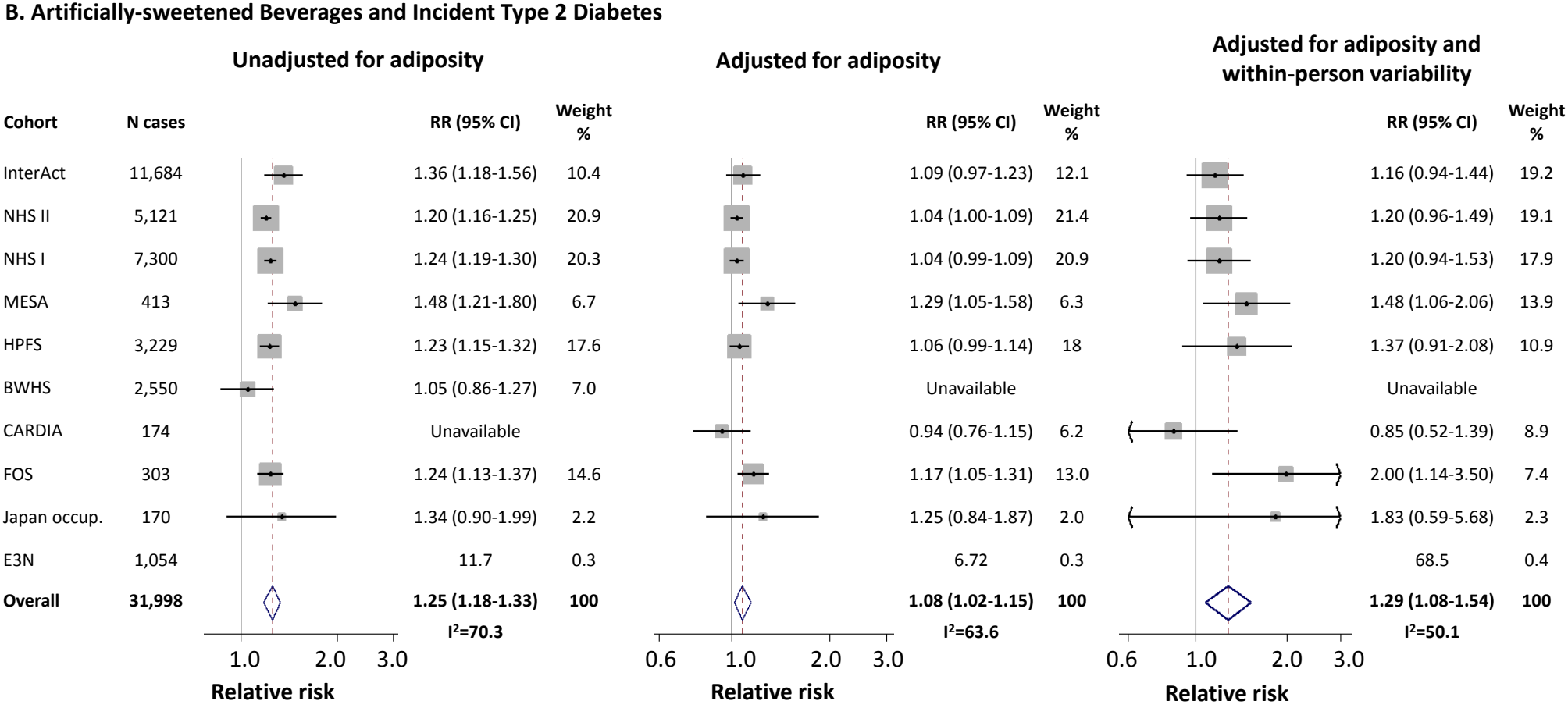
B. Artificially-sweetened Beverages and Incident Type 2 Diabetes
Unadjusted for adiposity
Adjusted for adiposity
Adjusted for adiposity and within-person variability
Cohort
N cases
RR (95% CI)
Weight %
InterAct 11,684 1.36 (1.18-1.56) 10.4
NHS II 5,121 1.20 (1.16-1.25) 20.9
NHS I 7,300 1.24 (1.19-1.30) 20.3
MESA 413 1.48 (1.21-1.80) 6.7
HPFS 3,229 1.23 (1.15-1.32) 17.6
BWHS 2,550 1.05 (0.86-1.27) 7.0
CARDIA 174 Unavailable
FOS 303 1.24 (1.13-1.37) 14.6
Japan occup. 170 1.34 (0.90-1.99) 2.2
E3N 1,054 11.7 0.3
Overall 31,998 1.25 (1.18-1.33) 100
I²=70.3
1.0
2.0
3.0
Relative risk
RR (95% CI)
Weight %
1.09 (0.97-1.23) 12.1
1.04 (1.00-1.09) 21.4
1.04 (0.99-1.09) 20.9
1.29 (1.05-1.58) 6.3
1.06 (0.99-1.14) 18
Unavailable
0.94 (0.76-1.15) 6.2
1.17 (1.05-1.31) 13.0
1.25 (0.84-1.87) 2.0
6.72 0.3
1.08 (1.02-1.15) 100
I²=63.6
0.6
1.0
2.0
3.0
Relative risk
RR (95% CI)
Weight %
1.16 (0.94-1.44) 19.2
1.20 (0.96-1.49) 19.1
1.20 (0.94-1.53) 17.9
1.48 (1.06-2.06) 13.9
1.37 (0.91-2.08) 10.9
Unavailable
0.85 (0.52-1.39) 8.9
2.00 (1.14-3.50) 7.4
1.83 (0.59-5.68) 2.3
68.5 0.4
1.29 (1.08-1.54) 100
I²=50.1
0.6
1.0
2.0
3.0
Relative risk

Figure 1B

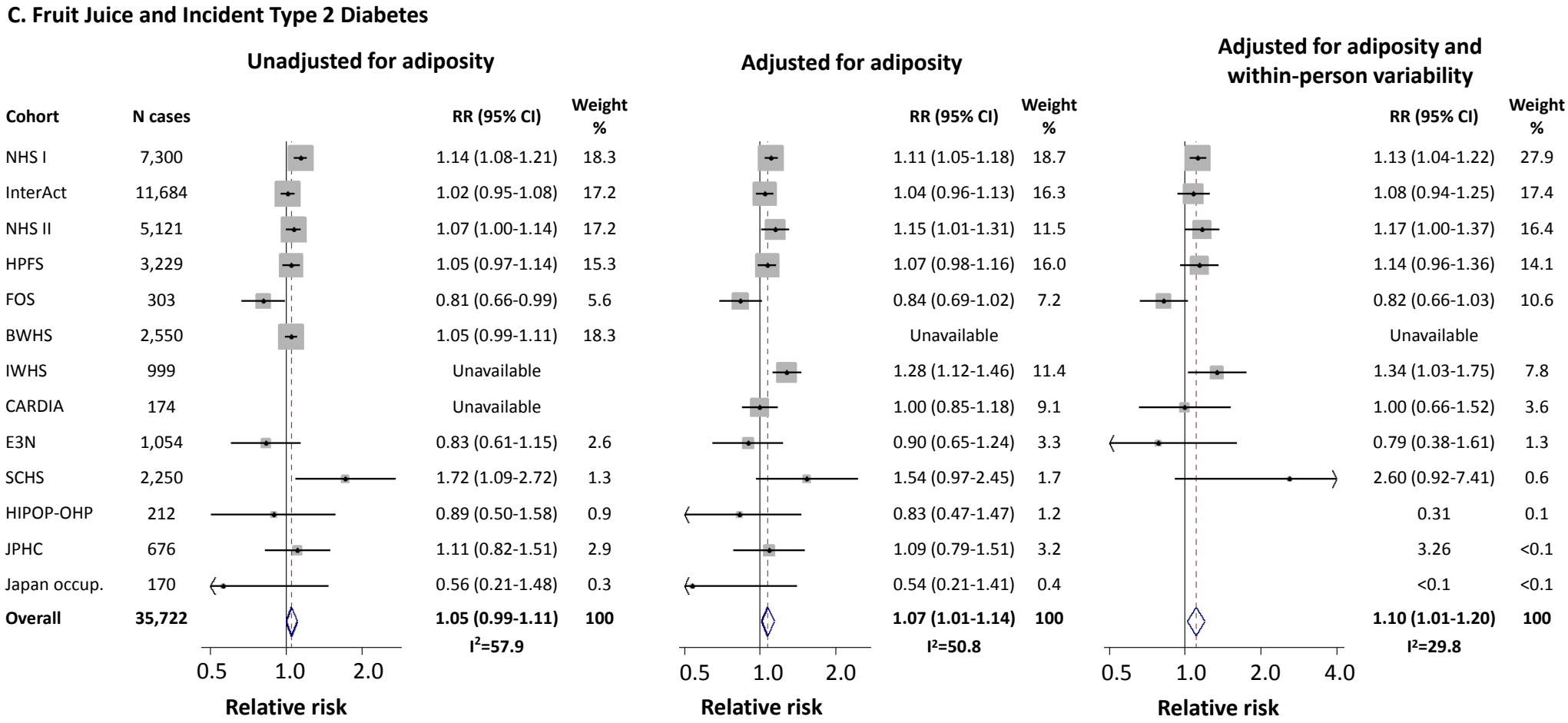
C. Fruit Juice and Incident Type 2 Diabetes
Unadjusted for adiposity
Adjusted for adiposity
Adjusted for adiposity and within-person variability
Cohort
N cases
RR (95% CI)
Weight %
NHS I 7,300 1.14 (1.08-1.21) 18.3
InterAct 11,684 1.02 (0.95-1.08) 17.2
NHS II 5,121 1.07 (1.00-1.14) 17.2
HPFS 3,229 1.05 (0.97-1.14) 15.3
FOS 303 0.81 (0.66-0.99) 5.6
BWHS 2,550 1.05 (0.99-1.11) 18.3
IWHS 999 Unavailable
CARDIA 174 Unavailable
E3N 1,054 0.83 (0.61-1.15) 2.6
SCHS 2,250 1.72 (1.09-2.72) 1.3
HIPOP-OHP 212 0.89 (0.50-1.58) 0.9
JPHC 676 1.11 (0.82-1.51) 2.9
Japan occup. 170 0.56 (0.21-1.48) 0.3
Overall 35,722 1.05 (0.99-1.11) 100
I²=57.9
NHS I 1.11 (1.05-1.18) 18.7
InterAct 1.04 (0.96-1.13) 16.3
NHS II 1.15 (1.01-1.31) 11.5
HPFS 1.07 (0.98-1.16) 16.0
FOS 0.84 (0.69-1.02) 7.2
BWHS Unavailable
IWHS 1.28 (1.12-1.46) 11.4
CARDIA 1.00 (0.85-1.18) 9.1
E3N 0.90 (0.65-1.24) 3.3
SCHS 1.54 (0.97-2.45) 1.7
HIPOP-OHP 0.83 (0.47-1.47) 1.2
JPHC 1.09 (0.79-1.51) 3.2
Japan occup. 0.54 (0.21-1.41) 0.4
Overall 1.07 (1.01-1.14) 100
I²=50.8
NHS I 1.13 (1.04-1.22) 27.9
InterAct 1.08 (0.94-1.25) 17.4
NHS II 1.17 (1.00-1.37) 16.4
HPFS 1.14 (0.96-1.36) 14.1
FOS 0.82 (0.66-1.03) 10.6
BWHS Unavailable
IWHS 1.34 (1.03-1.75) 7.8
CARDIA 1.00 (0.66-1.52) 3.6
E3N 0.79 (0.38-1.61) 1.3
SCHS 2.60 (0.92-7.41) 0.6
HIPOP-OHP 0.31 0.1
JPHC 3.26 <0.1
Japan occup. <0.1 <0.1
Overall 1.10 (1.01-1.20) 100
I²=29.8
0.5
1.0
2.0
4.0
Relative risk

Figure 1C

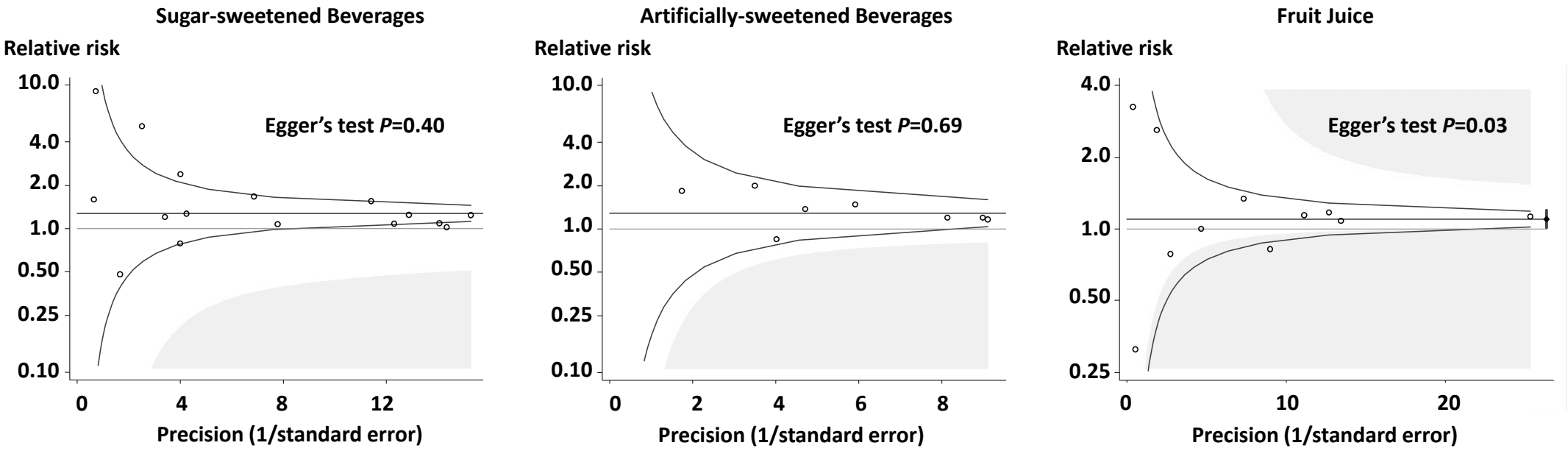
Sugar-sweetened Beverages
Relative risk
10.0
4.0
2.0
1.0
0.50
0.25
0.10
Egger's test P=0.40
0
4
8
12
Precision (1/standard error)
Artificially-sweetened Beverages
Relative risk
10.0
4.0
2.0
1.0
0.50
0.25
0.10
Egger's test P=0.69
0
2
4
6
8
Precision (1/standard error)
Fruit Juice
Relative risk
4.0
2.0
1.0
0.50
0.25
Egger's test P=0.03
0
10
20
Precision (1/standard error)

Figure 2

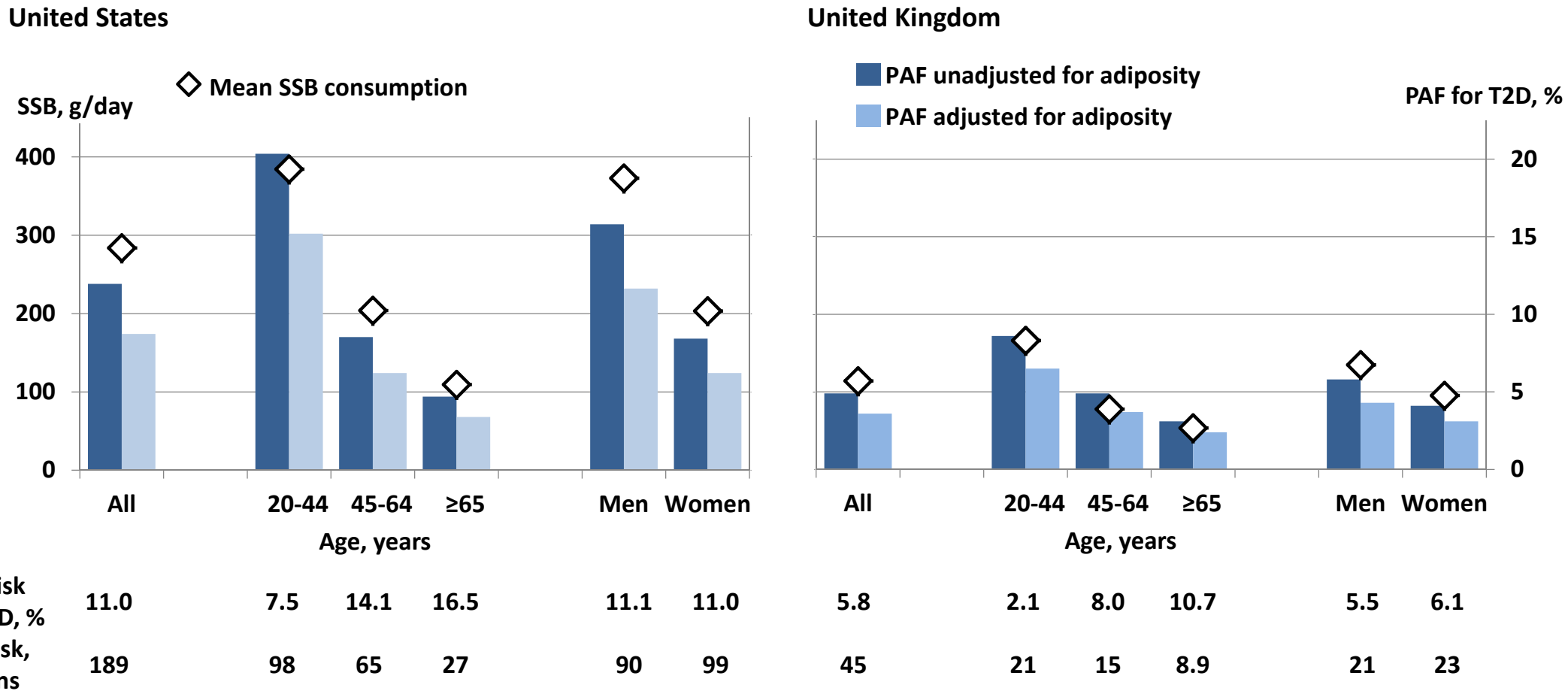
United States
Mean SSB consumption
SSB, g/day
400
300
200
100
0
All
20-44
45-64
≥65
Men
Women
Age, years
10-y risk for T2D, %
11.0
7.5
14.1
16.5
11.1
11.0
N at risk, millions
189
98
65
27
90
99
United Kingdom
PAF unadjusted for adiposity
PAF adjusted for adiposity
PAF for T2D, %
20
15
10
5
0
All
20-44
45-64
≥65
Men
Women
Age, years
5.8
2.1
8.0
10.7
5.5
6.1
45
21
15
8.9
21
23

Figure 3

Supplementary materials for "Consumption of sugar-sweetened beverages, artificially sweetened beverages, and fruit juice and incidence of type 2 diabetes: a systematic review, meta-analysis, and estimation of population attributable fraction"

Fumiaki Imamura, Laura O'Connor, Zheng Ye, Jaakko Mursu, Yasuaki Hayashino, Shilpa N Bhupathiraju, Nita G. Forouhi

Contents

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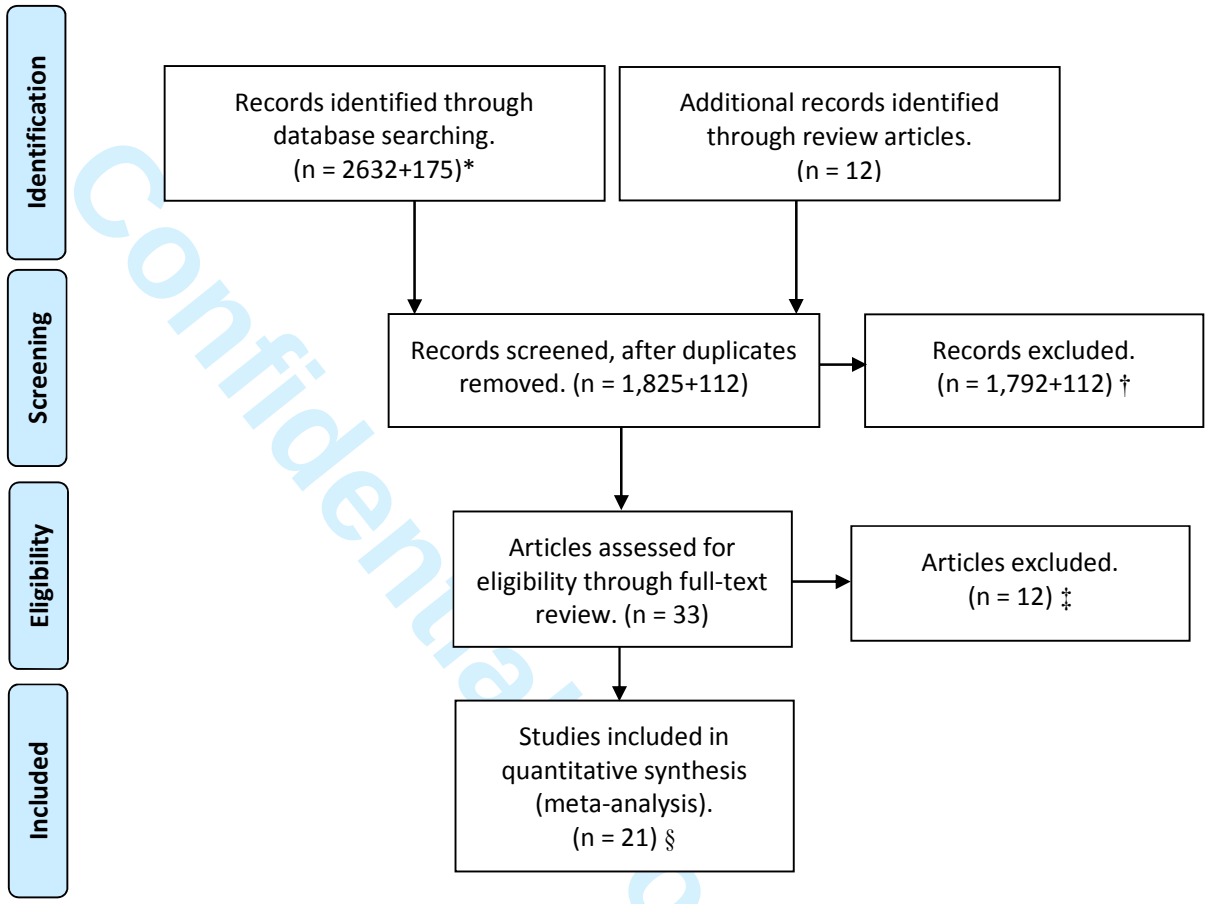

Figure S1. Systematic identification of published literature on beverage consumption and type 2 diabetes. Search terms are described in Supplementary Text. * Two values indicate search on 31 May 2013 and search on 10 February 2014. † Major reasons for exclusions for initial screening: main exposures were alcohol, coffee, or other dietary factors, rather than sweetened beverages or fruit juice; outcomes were not diabetes, either recruiting diabetes patients or assessing diabetes as a covariate; studies were cross-sectional; studies recruited children; and publications are reviews, editorials, commentaries or other formats. ‡ See Table S1 for reasons for exclusion. § Seventeen cohorts, as a few cohorts published more than one article examining different beverages. One cohort met eligibility criteria after we obtained additional information.

Table S1. Studies reviewed in full text for eligibility and included or excluded for meta-analysis.*

Cohort, country*	Results from full text review and author contact *	Decision for the present meta-analysis
Identified as potentially eligible and reviewed †		
BRHS, UK[1,2] Cohort in Australia[3] EBSHP, US.[4]	Ineligible, no information on any assessments of diets. We did not contact the authors.	Excluded.
EPIC-NL, Netherlands.[5]	Eligible, but the study was included in EPIC-InterAct.	Excluded.
Hisayama, Japan[6]	Ineligible. A diet and incident diabetes were assessed, but the authors confirmed no information on consumption of sweetened beverages and fruit juice.	Excluded.
SUN Study, Spain[7] WHS, US[8,9] PHS, US[9] NIH-AARP, US[10] D.E.S.I.R, France[11]	Eligible, but not included. Each cohort had information on dietary consumption and incident T2D. The authors could not respond to our request, because a resource was limited to conduct analyses we requested.	Excluded.
HIPOP-OHP, Japan[12]	Eligible, reported information of SSB consumption and incident T2D. After we contacted the authors, the authors provided sufficient information.	New unpublished estimates were used.
Identified as eligible		
FMCHES, Finland[13]	SSB and sugar-sweetened berry juices were reported separately. The authors did not respond to our request to combine the two.	Reported statistics on SSB were used.
NHS I, US[14,15] HPFS, US[14,16,17] NHS II, US[14,18,19]	Each cohort was censored at different time-point depending on types of beverages. Also, other publications from the cohorts indicate availability of data based on longer follow-up.	Analyses were updated using the censoring date in each dataset up to date (NHS, up to 2008; NHS II, 2011; and HPFS, 2010).
KIHDS, Finland[20]	Information was available in analyses excluding hyperglycaemic adults.	Updated analyses additionally including hyperglycaemic adults at risk of developing diabetes (little change in results).
CARDIA, US[21,22]	Eligible, evaluating prospective associations between beverage consumption and hyperglycaemia, and having information on incident T2D. We requested analysis on T2D. After positive responses, new estimates were eventually not available.	We used reported estimates for hyperglycaemia, accounting for the proportion of T2D cases (see Table S3).
Iowa WHS, US[23]	Only published as an abstract presented at a conference, thus not fully peer-reviewed and missing information needed.	Generic information on the cohort was obtained by another publication from Iowa WHS.[24] Exposure distributions of the adults were

		approximated by consumption observed in women in ARIC[25], considering similarity in chronological and demographic characteristics of women of the two cohorts.
FOS, US[26]	The original paper examined SSB and hyperglycaemia only. One author provided estimates needed for meta-analysis, evaluating each of SSB, ASB, and fruit juice, by methods as previously reported.[27]	New estimates were used as provided.
ARIC, US[25]	Fruit juice, SSB and ASB were combined. We requested to separate them and update estimates matched with our objectives. No additional data became available.	Only reported statistics were used. We recorded potential bias due to misclassification of types of beverages. The authors stated no change in results after adjustment for measures of adiposity (body-mass index and waist-to-hip ratio). ‡
JPHC, Japan[28]	Repeated measures were available in the cohort, but not used.[29] We requested analyses using them, but the authors decided not to do, being concerned of lack of peer-review of the specific analysis.	Reported statistics based on 10-year follow-up were used. Repeated measures of diets were not used, although they could be used.[29,30]‡
EPIC-InterAct, eight European countries[31]	Measures of associations of each type of beverages with incident T2D before and after adjustment for measures of adiposity were available, based on analyses treating each type of beverage consumption as a continuous variable. The most recent data were analysed. Thus, no contact was attempted	Reported statistics were used.
E3N, France[32]	This cohort participates in EPIC-InterAct[31,33]. Two articles partly included the same adults. To avoid double-counts of overlapping adults, we requested analyses of 48,985 women after excluding 20,851 adults eligible for EPIC-InterAct. The authors responded to our request.	Estimates without overlap with InterAct were used.
SCHS, Singapore[34]	Availability of ASB was not clear. Fruit juice was evaluated with vegetable juice. We requested information for the clarity and additional analysis, but did not receive any.	Reported statistics were used.
Black WHS, US[35]	SSB and sugar-sweetened berry juices were reported separately. Results after adjustment for body-mass index were presented partially (estimates for the extreme categories) and presented by stratification. We requested the authors to do analysis combining the two beverage types, but could not obtain any information.	Reported statistics for SSB were used.
MESA, US[36]	Eligible. ASB was assessed as a main variable. SSB was assessed, but presented only in the text. Analyses using ASB, SSB, and fruit juice separately were requested, but the author confirmed no availability of data at the time of request. ‡	Reported statistics were used. The article reported null associations between SSB consumption and incidence of T2D, but available in a review article.[37]
Occupational cohort,	We identified availability of fruit juice based on a publication on dietary assessment	Estimates for SSB, ASB, and fruit juice

Japan[38]	they used. Thus, we requested estimates for fruit juice consumption and incident T2D, as well as for SSB and ASB, with and without adjustment for adiposity measures. The author responded to our request.	consumption were used, as provided. The update was unlikely to involve any bias.

Abbreviations: ARIC, Atherosclerosis Risk in Communities Study; ASB, Artificially-sweetened beverages; BRHS, British Regional Heart Study; CARDIA, Coronary Artery Risk Development in Young Adults Study; D.E.S.I.R., Data from an Epidemiological Study on the Insulin Resistance Syndrome; EBSHP, The East Boston Senior Health Project; EPIC, European Prospective Investigation into Cancer and Nutrition Study; EPIC-NL, EPIC-Netherlands Study; FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study (Framingham Heart Study, the second generation); HIPOP-OHP, the High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHDS, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; NIH-AARP, National Institute of Health American Association of Retired Persons Diet and Health Study; OGTT, oral-glucose tolerance test; PHS, Physicians Health Study; SCHS, Singapore Chinese Health Study; SSB, sugar-sweetened beverages; SUN, Sequimiento University of Navarra; T2D, type 2 diabetes; WHS, Women's Health Study.

* When we contacted the authors (October 2013 to December, 2013), we specified our requests to obtain categorical and continuous estimates before and after adjustment for obesity status for prospective associations between each type of SSB, ASB, and fruit juice and incident T2D. We specified statistical methods, categorization, and covariates adjusted for, based on prior publications. After we obtained information usable in this meta-analysis, we did not request further information. We did not request any additional information when a publication reported estimates adjusted for potential confounders and before and after adjustment for adiposity measures (eg EPIC-InterAct).

† Some exclusion might be related to publication bias, because these cohorts could technically provide information useful for this meta-analysis. According to the publications not included in this meta-analysis, 283,058 of whom 23,270 cases arose were not included in this meta-analysis in total.

‡ The authors reported estimates after stratification by demographics or by body-mass index, we merged the estimates by fixed-effect meta-analysis in main analysis. In analyses to test heterogeneity by demographics or body-mass index, stratified results were used.

Table S2. Quality assessment of cohort studies included in meta-analysis of sweet beverages and incidence of type 2 diabetes.

Cohort	Domains of bias*, + Low risk of bias, − High risk of bias, ? unknown. Con-founding	Selection	Dietary measures	Follow-up	Missing data	Diagnosis of T2D	Selective report	Overall †	Additional consideration on potential sources of bias
NHS I[14,15]	?	+	?	+	+	?	+	?	Analyses were updated. A risk of bias was unlikely to be high.
NHS II[14,18,19]	?	+	?	+	+	?	+	?	Analyses were updated. A risk of bias was unlikely to be high.
ARIC[25]	?	+	−	−	+	+	−	−	SSB and ASB were not separated.
Iowa WHS[23]	?	?	−	−	?	?	−	−	Only the conference abstract was published.
FOS[26]	?	+	−	+	+	+	?	?	Modified substantially for updating the original analysis.
HPFS[39,40]	?	+	?	+	+	?	+	?	Analyses were updated. A risk of bias was unlikely to be high.
Black WHS[41]	?	+	?	+	+	?	−	?	Results were reported selectively.
MESA[42]	?	+	−	−	+	+	−	?	Results were reported selectively.
EPIC-InterAct[31]	?	+	?	−	+	?	+	?	A risk of bias was unlikely to be high.
E3N[43]	−	+	?	−	+	?	+	−	Adjustment for adiposity was likely to be biased.[44–47]
SCHS[48]	?	+	−	−	−	?	?	−	Fruit juice and fruit drinks (SSB) were not separated. Exclusion might have caused bias.
JPHC[49]	?	+	?	−	+	?	−	?	Main and subgroup analyses were internally and externally inconsistent.[28,30]
Occup. cohort[50]	?	+	−	−	+	+	?	?	Modified substantially for updating the original analysis.
HIPOP-OHP[51]	?	+	−	−	−	+	?	−	Exclusion might have caused bias, losing 31% of participants during the follow-up.
CARDIA[52]	?	+	−	+	?	−	−	−	Main and subgroup analyses were internally and externally inconsistent.[21,22]
KIHDS[53]	?	+	−	−	+	+	+	?	Habitual consumption not measured well.
FMCHES[54,55]	?	+	?	−	+	+	+	?	Generalizability to the modern population is concerning.

Abbreviations: ARIC, Atherosclerosis Risk in Communities Study; CARDIA, Coronary Artery Risk Development in Young Adults Study; EPIC, European Prospective Investigations into Cancer and Nutrition Study; FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study (Framingham Heart Study, the second generation); HIPOP-OHP, High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHDS, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; SCHS, Singapore Chinese Health Study; SSB, WHS, Women's Health Study.

* See supplementary text for details. For dietary measures, bias was considered as high, if quality of dietary measures was not assessed within a study; as unknown for the other studies, with possible misclassification. Follow-up was rated based on use of repeated dietary measures. Missing data on exposure were considered unlikely to cause bias in any studies, except SCHS and HIPOP-OHP losing 15% and 31% of participants, respectively, in follow-up. Bias for type 2 diagnosis (T2D) diagnosis was rated as a low risk, if a study took approach to detect undiagnosed diabetes. See also Table S3 for validity measures of dietary measures and ascertainment of T2D; and Table S5, for potential confounders.

† Overall possibility of bias reflects possibility of bias specifically on the estimates used in the meta-analysis (see the first right column and the supplementary text on page 16). Sensitivity meta-analysis was performed after excluding these studies (Table S6).

Table S3. Validity measures of beverage consumption and incident type 2 diabetes.

Cohort*	Assessment of within-person variability of dietary estimates by FFQ or DR									Ascertainment of incident type 2 diabetes§		
	Internal substudy †	Reference ×n of assessments	N	SSB		ASB		Fruit juice		Self-report only	N cases identified (Person-years, ×1000)	PPV, n for validation
				r‡	s_Q/s‡	r‡	s_Q/s‡	r‡	s_Q/s‡			
NHS I[56,57]	Yes	7d DR ×4	173	0.84	1.83	0.36	1.83	0.84	1.00	X	7,449 (1,571)	0.98, 62
NHS II	No									X	5,225 (1,660)	0.98, 62
ARIC[25]	No										1,437 (92.5)	
Iowa WHS[24]	No									X	1,561 (330.0)	0.64, 44
FOS[27,58]	No										303 (33.3)	
HPFS[39,40,59]	Yes	7d DR ×2	127	0.84	2.37	0.40	2.24	0.82	1.66	X	3,364 (777.3)	
Black WHS[35,41]	Yes	7d DR ×4	403	0.67	1.17	0.67	1.17	0.64	1.19	X	2,713 (338.9)	0.94,229
MESA[36,42]	No	7d DR ×4	186	0.46	0.71	0.46	0.71	‖	‖		413 (27.6)	
EPIC-InterAct[33] **	Yes	24hR ×12, 24, or 10, 4d DR ×4, or 7d DR ×2	999	0.65	1.13	0.64	1.14	0.73	1.30		11,684 (3,990)	
E3N[32,43]	Yes	24hR × 9-12	119	0.55	1.22	0.55	1.22	0.55	1.22		1,054 (607.0)	
SCHS[48,60]	Yes	24hR ×2	810	0.49	1.20	‖	‖	0.58	1.29		2,273 (249.2)	0.99, 702
JPHC (men) [49,61]	Yes	7d DR ×4 or 2	94	0.27	2.46	‖	‖	0.17	2.46	X	824 (271.7)	0.82, 93
(women)	Yes	7d DR ×4 or 2	107	0.24	2.46	‖	‖	0.18	2.46			
Occup. cohort, Japan[38,50]	No	7d DR ×4	92	0.39	1.06	0.39	1.06	0.24	1.98		170 (11.3)	
HIPOP-OHP[12,51]	Yes	24hR ×4	76	0.32	2.00	‖	‖	0.32	2.00		212 (20.8)	
CARDIA[21,52,62]	Yes	24hR ×7	128	0.68	1.90	0.68	1.90	0.59	1.78		288 (67.2††)	0.62
KIHDS[20,53,63]	No	24hR ×10	96	0.68	1.00	‖	‖	‖	‖		506 (46.8)	
FMCHES[13,54,55]	No	7d DR ×1	79	0.62	1.17	‖	‖	‖	‖		175 (58.8)	

Abbreviations: 24hR, 24-hour recalls; ARIC, Atherosclerosis Risk in Communities Study; ASB, artificially-sweetened beverages; CARDIA, Coronary Artery Risk Development in Young Adults Study; DR, diet records; EPIC, European Prospective Investigations into Cancer and Nutrition Study; FFQ, food-frequency questionnaires, FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study (Framingham Heart Study, the second generation); HIPOP-OHP, High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHDS, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; PPV; predictive positive value; SCHS, Singapore Chinese Health Study; SSB, sugar-sweetened beverages; WHS, Women's Health Study.

* Citations represent articles we cited to derive measures of within-person variability of assessment of consumption of SSB, ASB, and fruit juice; and of validity of case ascertainment.

† NHS II, ARIC and Iowa WHS used the questionnaires developed for the nurses in the NHS I; FOS, FFQs for the NHS I at the analysis baseline and for the HPFS at the follow-up; MESA, FFQ developed for multi-ethnic populations in the Insulin Resistance Atherosclerosis Study; the occupational cohort in Japan, FFQ developed and validated in another setting. ARIC aggregated SSB, ASB, and fruit juice in their analysis, and correlations were averaged after Z-transform. Iowa WHS reported internal validation study was published[64], but we did not use it, because reference methods (24hR × 5) were implemented only in February and March, which was unlikely to be suitable as a reference method of the FFQ that was designed to capture 1-year habitual diet. Finish cohorts did not perform internal validation studies to examine whether each method could capture habitual

diets. Thus, two studies in Canada and in Netherlands validating a diet-history method were reviewed. Because of similarity in geography, the study in Netherlands was focused; yet, validity for assessment of sugar intake was similar (r=0.62 and 0.60) in the two studies.

‡ r represents correlation coefficients between estimates based on FFQ or diet history and estimates based on average of reference methods. We used energy-adjusted estimates corrected for within-person variations, if available.[65,66] s_Q/s represents a ratio of a standard deviation of FFQ or diet history to that of a reference method; for diet records, standard deviations were assumed to be unbiased. If specific measures of r and s_Q/s were not available for SSB, ASB, or fruit juice, variables related to refined sugars (disaccharides, sucrose, or carbohydrates) were used for SSB and ASB, and averages of variables related to sugars and vitamin C intakes were used for fruit juice. Averages of correlations were based on Z-transformed values[67,68]; of ratios, log-transformed values.

§ For studies using objective measures of diagnosis (Table 1), PPV was assumed to be 1.0.Person-year was coded as presented or imputed by using the number of participants, the number of incident cases, and the maximum duration of follow-up. The presented numbers of cases and person-years were not corrected for positive predictive values (PPV). Thus, some values were different from those in Table 1.

|| Beverages were not assessed for associations with incident diabetes and not included in this meta-analysis.

** In EPIC-InterAct, FFQs were developed specifically in each of the eight countries of the consortium. Measures of validity and reliability were calculated by weighted averages of the measures from the eight cohorts[43,53,69–78] (available on request), for which weights were those of country-specific estimates to the overall estimates in EPIC-InterAct. The total number of adults were based on the number of adults contributing to the measures of validity for SSB and ASB. For fruit juice, the size was 1,258.

†† CARDIA reported associations of beverage consumption with hyperglycaemia. We included the study in this meta-analyses, considering the overlapping definitions of hyperglycaemia and incident type 2 diabetes (including, use of antidiabetic medications). PPV represents the proportion of patients with type 2 diabetes to patients with hyperglycaemia based on another publication from CARDIA examining type 2 diabetes as an outcome.

Table S4. Assessment of potential confounders in the studies included in the meta-analysis of beverage consumption and type 2 diabetes.

	Sugar-sweetened beverages			Artificially sweetened beverages			Fruit juice		
Cohort, country	Crude RR	Adjusted RR (95% CI)*	Note on adjustment†	Crude RR	Adjusted RR (95% CI)*	Note on adjustment†	Crude RR	Adjusted RR (95% CI)*	Note on adjustment†
FMCHES[13]	1.94	na*		na	na		na	na	
NHS I[14,15]	1.51	1.39 (1.30-1.48)		1.42	1.24 (1.19-1.30)		1.42	1.24 (1.19-1.30)	
NHS II[14,18,19]	1.31	1.17 (1.11-1.24)		1.36	1.20 (1.16-1.25)		1.36	1.20 (1.16-1.25)	
HPFS[14,16,17]	1.21	1.31 (1.20-1.44)		1.51	1.23 (1.15-1.32)		1.51	1.23 (1.15-1.32)	
KIHDS[20,63]	0.97	1.06 (0.95-1.18)		na	na		na	na	
CARDIA[21,22]	na	na		na	na		na	na	
Iowa WHS[23]	na	na		na	na		na	na	
FOS[26,27]	1.25	1.12 (0.90-1.40)		1.35	1.24 (1.13-1.37)		1.35	1.24 (1.13-1.37)	
ARIC[25]	1.08	1.01 (0.96-1.06)	Not for a diet‡	na	na		na	na	
JPHC[28]	1.21	1.25 (0.99-1.58)		na	na		na	na	
EPIC-InterAct[31]	1.39	1.21 (1.12-1.31)	Not for a diet‡ and clinical factors.§	1.60	1.36 (1.18-1.56)	Not for a diet‡ and clinical factors.§	1.60	1.36 (1.18-1.56)	Not for a diet‡ and clinical factors.§
E3N[32]	2.64	2.82 (0.87-9.17)		12.6	11.7 (4.03-34.3)		12.6	11.7 (4.03-34.3)	
SCHS[34]	2.04	2.22 (1.64-3.00)	Not for clinical factors.	na	na		na	na	Not for clinical factors.
Black WHS	1.16	1.10 (1.05-1.16)		na	1.05 (0.86-1.27)		na	1.05 (0.86-1.27)	
HIPOP-OHP[12]	0.79	0.89 (0.75-1.06)		na	na		na	na	
MESA[36]	na	na		1.35	1.48 (1.21-1.80)	Not for a diet and clinical factors.	1.35	1.48 (1.21-1.80)	Not for a diet and clinical factors.
Occup. Japan[38]	1.12	1.08 (0.88-1.33)		3.17	1.34 (0.90-1.99)		3.17	1.34 (0.90-1.99)	
Pooled (Table 2)	1.25	1.18 (1.09-1.28)		1.48	1.25 (1.18-1.33)		0.97	1.05 (0.99-1.11)	

Abbreviations: ARIC, Atherosclerosis Risk in Communities Study; CARDIA, Coronary Artery Risk Development in Young Adults Study; EPIC, European Prospective Investigation into Cancer and Nutrition Study; FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study (Framingham Heart Study, the second generation); HIPOP-OHP, High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHDS, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; PA, physical activity; SCHS, Singapore Chinese Health Study; WHS, Women's Health Study.

* Relative risk (RR) and 95% confidence interval (CI) adjusted for potential confounders except adiposity measures. Models adjusted for adiposity measures are presented in Table 2 and Figure 1. 'na' indicates that the authors did not report statistics for the specific estimate (eg some authors reported adiposity-adjusted estimates only).

† highlighting factors not adjusted in main estimates of a study. Unless noted, studies adjusted for socio-demographic and lifestyle covariates, including age, sex, race/ethnic groups, socioeconomic (SES) variables (education history, income, or occupation), physical activity, and smoking status. Race and SES were considered as adjusted for in some cohorts recruiting participants in a population homogenous in race/ethnic status and in occupation (NHS I, NHS II, HPFS, and occupational cohort in Japan). Age was considered adjusted for in a cohort using it as a time-scale in longitudinal analysis (NHS I, NHS II, HPFS, EPIC-InterAct, and E3N).

‡ ARIC, EPIC-InterAct and MESA did not adjust for dietary factors in main analyses. In secondary analysis, EPIC-InterAct and MESA tested influence of potential dietary confounders and reported little influence of them. ARIC applied dietary adjustment only for fibre intake.

§ Clinical factors mean either family history of diabetes, use of anti-hypertensive or lipids-lowering drugs, or history of cardiovascular diseases, hypercholesterolemia, or hypertension. EPIC-InterAct had not collected family history of diabetes among 51.7% of the random sub-cohort, and thus the variable was not used in the analysis, but sensitivity analysis excluding adults with known family history of diabetes confirmed little influence of the variable.[31]

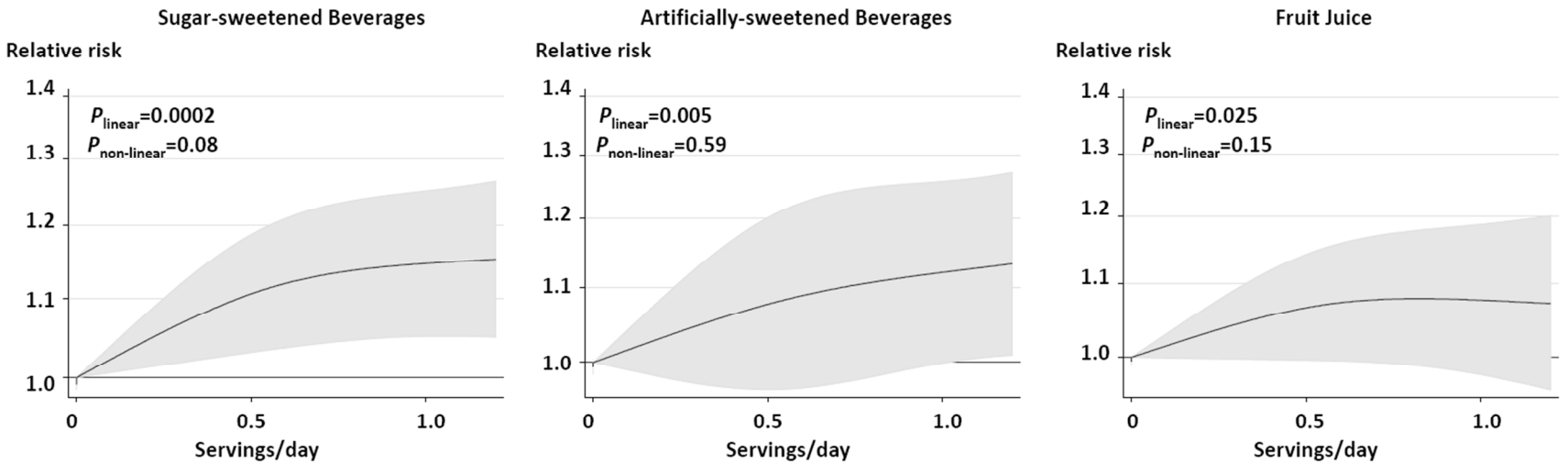

Figure S2. Non-linear associations of consuming sugar-sweetened beverages, artificially-sweetened beverages, and fruit juice with incident type 2 diabetes. Estimates were obtained by random-effects meta-analysis adjusted for adiposity. The curves and *P* for a non-linear associations ($P_{non\text{-}linear}$) were obtained by cubic spline meta-analysis.[79] Solid lines are the central estimates of relative risks (RR) and shaded areas are the corresponding 95% confidence interval (CI). The analysis needed categorical estimates, rather than continuous estimates per one serving/day, thus we needed to drop the studies reporting only continuous estimates: for sugar-sweetened beverages, 13 estimates were used, not including European Prospective Investigations into Cancer and Nutrition Study (EPIC)-InterAct and Coronary Artery Risk Development in Young Adults Study (CARDIA), Iowa Women's Health Study and Multiethnic Study of Atherosclerosis; for artificially-sweetened beverages, 7 estimates were used, not including EPIC-InterAct, CARDIA; for fruit juice, 10 estimates were used, not including EPIC-InterAct and CARDIA. *P* for a linear association (P_{linear}) was obtained by meta-analysis using all estimates available. Using the limited categorical data, calibration for within-individual variability applied to categorical estimates[80,81] provided steeper effects with similar curves and wide CI (data not shown).

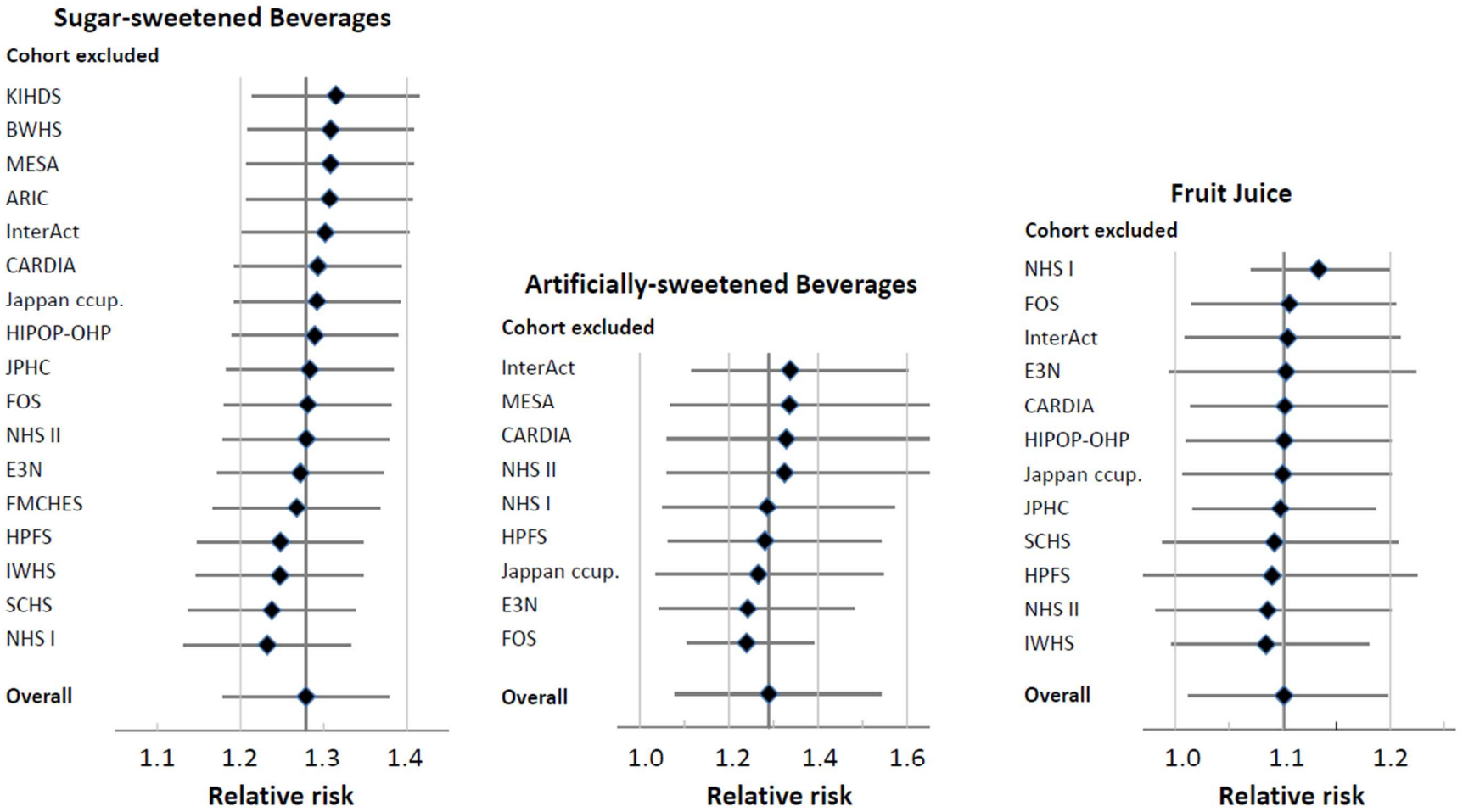

Figure S3. Influence analysis for the prospective associations of consuming sugar-sweetened beverages, artificially-sweetened beverages, and fruit juice with incident type 2 diabetes.

All estimates were obtained by random-effects meta-analysis adjusted for adiposity, for within-person variability of beverage consumption, and for imprecision of self-reported diabetes. Overall estimates were based on analysis using all estimates from the studies presented. The estimate accompanied to each cohort was based on meta-analysis excluding the study. Variations in relative risks ranged from -19% to +16% for SSB, -20% to +23% for ASB, and -7% to +16% for fruit juice. Abbreviations: ARIC, Atherosclerosis Risk in Communities Study; CARDIA, Coronary Artery Risk Development in Young Adults Study; FMCHES, Finnish Mobile Clinic Health Examination Survey; FOS, Framingham Offspring Study (Framingham Heart Study, the second generation); HIPOP-OHP, High-risk and Population Strategy for Occupational Health Promotion Study; HPFS, Health Professional Follow-up Study; JPHC, Japan Public Health Center-based Prospective Study; KIHDS, Kuopio Ischaemic Heart Disease Risk Factor Study; MESA, Multi-Ethnic Study of Atherosclerosis; NHS, Nurses' Health Study; SCHS, Singapore Chinese Health Study; SSB, sugar-sweetened beverages; WHS, Women's Health Study.

Table S5. Assessment of potential sources of heterogeneity for the prospective associations of consuming sweet beverages with type 2 diabetes.

Potential sources of heterogeneity (n cohorts)*	Sugar-sweetened beverages RR (95% CI) †	$P_{heterogeneity}$‡	Artificially-sweetened beverages RR (95% CI) †	$P_{heterogeneity}$‡	Fruit juice RR (95% CI) †	$P_{heterogeneity}$‡
Geographic location						
United States (n=9)	1.22 (1.07-1.38)		1.33 (1.06-1.67)		1.10 (0.99-1.22)	
Europe (n=4)	1.53 (1.12-2.09)		1.50 (0.98-2.30)		1.05 (0.90-1.21)	
Singapore or Japan (n=4)	0.94 (0.43-2.08)	0.39	1.83 (0.59-5.68)	0.92	0.42 (0.02-7.19)	0.51
Age on average						
<53 years (n=9)	1.20 (1.07-1.33)		1.13 (0.89-1.44)		1.15 (0.99-1.33)	
≥53 years (n=8)	1.36 (1.10-1.68)	0.16	1.49 (1.19-1.87)	0.12	1.08 (0.97-1.19)	0.48
Sex, proportion>50%						
Women (n=10)	1.28 (1.13-1.46)		1.42 (1.14-1.77)		1.08 (0.98-1.18)	
Men (n=7)	1.17 (0.90-1.53)	0.42	1.28 (0.92-1.78)	0.59	1.14 (0.95-1.36)	0.88
Body-mass index on average†						
<26.0 kg/m^2 (n=8)	1.45 (1.16-1.81)		1.41 (1.03-1.92)		1.11 (1.04-1.19)	
≥26.0 kg/m^2 (n=9)	1.16 (1.03-1.30)	0.49	1.38 (1.07-1.79)	0.41	1.08 (0.93-1.26)	0.57
Incidence of type 2 diabetes						
<6.0 / 1,000 person-years (n=8)	1.53 (1.27-1.85)		1.41 (1.09-1.81)		1.13 (1.07-1.20)	
≥6.0 / 1,000 person-years (n=9)	1.12 (1.00-1.25)	0.12	1.37 (1.00-1.87)	0.34	1.01 (0.85-1.20)	0.48
Duration of follow-up						
<10 years (n=6)	1.11 (0.93-1.32)		1.70 (1.23-2.36)		1.15 (0.14-9.35)	
≥10 years (n=11)	1.36 (1.19-1.56)	0.68	1.33 (1.07-1.64)	0.13	1.09 (1.02-1.17)	0.96
N of dietary measurements						
Once, only at baseline (n=11)	1.28 (1.06-1.56)		1.55 (1.19-2.01)		1.10 (0.97-1.24)	
Repeated (n=6)	1.26 (1.11-1.43)	0.25	1.24 (0.96-1.61)	0.90	1.08 (0.97-1.20)	0.068
Ascertainment of type 2 diabetes						
Self-reported only (n=6)	1.36 (1.15-1.60)		1.22 (1.05-1.42)		1.15 (1.08-1.22)	
Objective measures (n=11)	1.19 (1.02-1.39)	0.20	1.52 (1.14-2.03)	0.93	0.98 (0.86-1.11)	0.008

* Stratified analysis was prespecified for demographics and factors significantly predicting heterogeneity of associations for any type of beverages (p<0.1). For each type of beverages, a fewer cohorts contributed to the estimates: sugar-sweetened beverages, n=17 in total; artificially sweetened beverages, n=9; and fruit juice, n=12.

† Random-effects meta-analysis was performed in each stratum to estimate relative risks (RR) and 95% confidence intervals (CI). All estimates were adjusted for within-person variations and precision of type 2 diabetes diagnosis. If cohorts reported estimates after stratification by demographics and after adjustment for adiposity measures, the stratified estimates were used: for example, estimates stratified by sex in the Atherosclerosis Risk in Communities Study and in Japan Public Health Center-based Prospective Study. Use of stratified estimates had more precise estimates. For example, when restricting populations to those with BMI<26.0 kg/m^2, RRs for sugar-sweetened beverages were 1.45 (1.16-1.81) with stratified estimates and 1.52 (1.11-2.06) without stratified estimates; and BMI≥26.0 kg/m^2, 1.16 (1.03-1.30) with stratified estimates and 1.17 (1.03-1.33) without stratified estimates.

‡ *P* for heterogeneity. Significant (*P*<0.1) for repeated measures of dietary assessments and ascertainment of type 2 diabetes (the last two sets of rows) in the analysis of fruit juice. Variables with *P*<0.2 were mutually adjusted. Variables with *P*>0.2 were obtained in the model including the variables meeting the criterion of P<0.2 for entry. Heterogeneity was not significant (*P*>0.1) for the other factors for any types of beverages: duration of follow-up, use of FFQ or other methods, and publication status (published or not).

Table S6. Associations of consuming sweet beverages with incident type 2 diabetes by sensitivity meta-analysis.

Consideration	Sugar-sweetened beverages		Artificially-sweetened beverages		Fruit juice	
	N studies	RR (95% CI)*	N studies	RR (95% CI)*	N studies	RR (95% CI)*
Random-effects or fixed-effects modelling.						
Random-effects.	17	1.28 (1.12-1.46)	9	1.29 (1.08-1.54)	12	1.10 (1.01-1.20)
Fixed-effects.	17	1.21 (1.14-1.28)	9	1.23 (1.06-1.44)	12	1.11 (1.05-1.17)
Unit of beverage consumption.						
per 1 serving/day (original estimates).	17	1.28 (1.12-1.46)	9	1.29 (1.08-1.54)	12	1.10 (1.01-1.20)
per 250 ml/day. †	17	1.28 (1.11-1.47)	9	1.25 (1.06-1.48)	12	1.13 (1.01-1.25)
Selected on the basis of quality of study.						
Studies without possibility of crucial bias by design. ‡	11	1.25 (1.10-1.41)	7	1.26 (1.13-1.41)	8	1.09 (1.00-1.19)
Studies without less valid dietary assessment. §	16	1.30 (1.13-1.49)	6	1.28 (0.92-1.80)	9	1.10 (1.01-1.20)
Aggregation of cohorts in the consortium analysis.‖						
Cohorts within EPIC-InterAct, aggregated.	17	1.43 (1.20-1.70)	9	2.13 (1.57-2.88)	12	1.06 (0.98-1.14)
Cohorts within EPIC-InterAct, separated.	25	1.40 (1.22-1.61)	17	2.00 (1.57-2.54)	19	1.06 (1.00-1.14)
Analysis accounting for errors of measures of validity						
Estimates of ln(RR), measures of validity of exposure (γ), and PPV , randomly drawn from SE variation. **	17 ×10,000	1.29 (1.10-1.53)	9 ×10,000	1.33 (1.06-1.11)	11 ×10,000	1.11 (1.00-1.25)
Calibrated for potential misclassification for adiposity measurements. ††						
r_a = 0.9 between observed and true adiposity measures	17	1.22 (1.07-1.41)	9	1.08 (0.87-1.34)	11	1.12 (1.02-1.21)
r_a = 0.8	17	1.20 (1.04-1.38)	9	1.01 (0.81-1.25)	11	1.12 (1.03-1.22)
r_a = 0.7	17	1.17 (1.02-1.35)	9	0.93 (0.75-1.15)	11	1.13 (1.04-1.23)

Abbreviations: CI, confidence interval; EPIC, European Prospective Investigation into Cancer and Nutrition Study; PPV, positive predictive value; RR, relative risk; SE, standard error.

* Random effects meta-analysis was performed in each stratum, except for the estimates derived from fixed-effects modelling. All were adjusted for adiposity measures and calibrated for misclassification of exposure and outcome.

† Median serving size of beverage consumption in the cohorts included in this present meta-analysis. Different studies defined one serving differently.

‡ Bias was determined by qualitative assessment (Table S2).

§ defined as r<0.4 compared to reference methods). Relatively low validity for dietary assessment (Table S3) was also used as a source of bias. As the validity measures were not all specific to each beverage, the results were interpreted cautiously as supplements.

‖ Using all cohorts available, but EPIC-InterAct was considered as a single cohort or separated.[31] The publication did not report the cohort-specific estimates adjusted for measures of adiposity. Thus, in the main analyses, we used the estimates combined within EPIC-InterAct. Additionally, the publication reported the cohort-specific estimates (11 cohorts in total from 8 countries) without adjustment for measures of adiposity.[31] Thus, the sensitivity to the aggregation was assessed here. Cohort-specific calibration for dietary measurement errors was applied.

** Iterative sensitivity analysis (10,000 times) was performed after incorporating quantitative bias and uncertainty[82] in different measures: dose-response estimates, within-person variability of beverage consumption, and precision of incident diabetes. Uncertainty of each was randomly drawn from each standard error. Out of 10,000 repeats, 2.5th, 50th (median), and 97.5th percentiles were obtained for 95% confidence limits and point estimate of RR.

†† Estimates were obtained after adopting specific unobserved, but realistic assumptions: 1) adiposity was measured with misclassification (r_a); 2) observed estimates adjusted for measured adiposity were biased to the extent related to r_a; and 3) estimates calibrated for r_a were obtained by a formula following simulation extrapolation (see text and Figure S4). A recent article[83] indicated r_a was greater than 0.73, thus assumed to be 0.7 or higher.

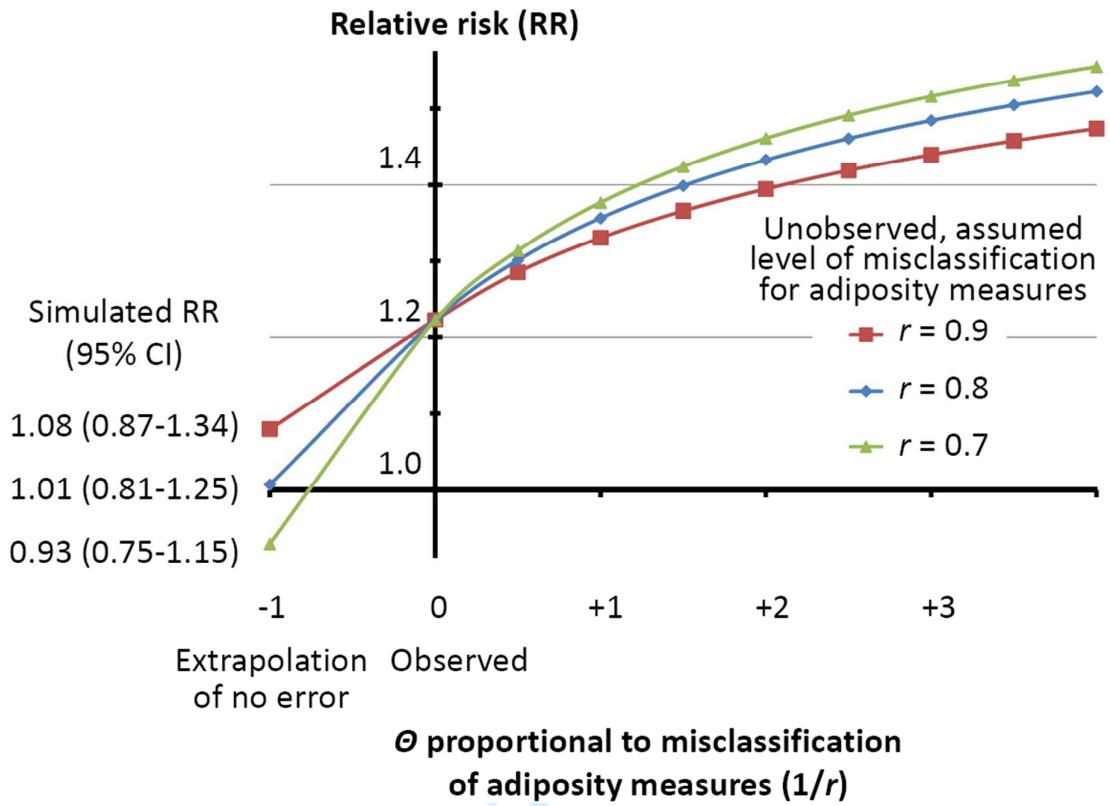

Figure S4. Assessment of a prospective association of consuming artificially sweetened beverages with incident type 2 diabetes after adjustment for assumed misclassification of adiposity measurements.

Estimates were obtained after adopting specific unobserved, but realistic assumptions: 1) adiposity was measured with misclassification (r_a); 2) observed estimates adjusted for measured adiposity were biased to the extent related to r_a; and 3) estimates calibrated for r_a were obtained by a formula following simulation extrapolation, $\frac{\sigma_{between}}{\sigma_{between}+\sigma_{within}(1+\theta)}$; θ=0 would produce observed RR=1.22 (0.98-1.52); θ=∞ would produce ln(RR) unadjusted for adiposity measure, RR=1.80 (1.13-2.46); θ=-1 would produce ln(RR) adjustged for potential misclassification of measured adiposity. The extrapolation for θ=-1 from the observable range, θ>0, was performed by non-linear association derived from θ ={0, 0.5, 1.0, 1.5, 2.0}.[84] A recent article[83] indicated r_a was greater than 0.73, thus we assumed r_a to be 0.7 or higher.

Table S7. The number of type 2 diabetes events over 10 years from 2010 related to sugar-sweetened beverage consumption among adults in the United States and in the United Kingdom.*

Population	Adults free of diabetes, N / 1,000	Consumption of SSB, mean±SD, g/day (% consumers)†	T2D in 10 years, N / 1,000 (10-year risk) ‡	T2D events prevented by eliminating SSB consumption §			
				Unadjusted for adiposity		Adjusted for adiposity	
				N / 1,000	PAF (95% CI)	N / 1,000	PAF (95% CI)
United States							
All	189,076	284±412 (54.4)	20,878 (11.0)	2,564	11.9 (11.3-12.6)	1,824	8.7 (8.3-9.2)
Age, years							
20-44	97,586	384±435 (65.3)	7,317 (7.5)	1,556	20.2 (18.4-22.0)	1,102	15.1 (13.6-16.5)
45-64	64,940	204±294 (44.8)	9,179 (14.1)	800	8.5 (7.3-9.7)	572	6.2 (5.4-7.1)
≥65	26,550	109±228 (37.7)	4,381 (16.5)	208	4.7 (4.1-5.3)	150	3.4 (3.0-3.9)
Sex							
Men	89,692	373±457 (61.7)	9,948 (11.1)	1,626	15.7 (14.6-16.8)	1,152	11.6 (10.7-12.5)
Women	99,383	203±295 (47.8)	10,930 (11.0)	937	8.4 (7.4-9.4)	673	6.2 (5.4-6.9)
United Kingdom							
All	44,719	114±157 (49.4)	2,593 (5.8)	126	4.9 (4.3-5.4)	79	3.6 (3.3-4.0)
Age, years							
20-44	20,865	166±181 (63.0)	441 (2.1)	38	8.6 (7.6-9.6)	21	6.5 (5.7-7.2)
45-64	14,937	78±126 (39.9)	1,195 (8.0)	59	4.9 (4.1-5.7)	37	3.7 (3.1-4.3)
≥65	8,920	53±95 (33.6)	954 (10.7)	29	3.1 (2.4-3.7)	21	2.4 (1.9-2.9)
Sex							
Men	21,243	135±173 (51.6)	1,170 (5.5)	67	5.8 (4.8-6.7)	43	4.3 (3.6-4.9)
Women	23,474	95±140 (47.4)	1,423 (6.1)	59	4.1 (3.6-4.7)	36	3.1 (2.7-3.4)

* ×1, 000 for counts (N) derived from the United States National Health and Nutrition Examination Survey, 2009-2010 (n=4,729 adults free of diabetes) and from the United Kingdom National Dietary Nutrition Survey, 2008/2009-2011/2012 (n=1,932 adults free of diabetes) (Supplementary Text for details). All statistics accounted for sampling weights. PAF, population attributable fraction; SD, standard deviation, SSB, sugar-sweetened beverages; T2D, type 2 diabetes.

† The distribution of SSB consumption was positively skewed in every population group. In data from the United States, consumers were defined by consumption of sugar-sweetened beverage at least once in a 24-hour recall of dietary consumption or by daily consumption reported in a dietary screener questionnaire. In those from the United Kingdom, consumers were defined as adults who recorded consumption of any of SSB during four days of dietary recording.

‡ 10-year risk of T2D was predicted using measured risk factors for T2D and a published risk-prediction algorithm in each of the United States and the United Kingdom.

§ Calculated based on the predicted T2D risk varying according to observed SSB consumption vs. the counterfactual T2D risk if no one in each population consumes SSB. The risks associated with SSB were estimated under different assumptions: Left. the effect of SSB consumption was partly mediated by obesity, modelled with relative risk unadjusted for adiposity measures; and Right. the effect of SSB was independent of obesity, modelled with relative risk adjusted for adiposity measures.

Supplementary Text

Search Strategy

We undertook electronic searches, using the Internet browser (Firefox 27.0.1). We initially searched existing reviews available at Cochrane Library, Centre for Reviews and Dissemination, Systematic Review Data Repository, PubMed, and OVID, on sugar-sweetened beverages (SSB), artificially-sweetened beverages (ASB), and fruit juice. We identified 15 reviews directly related to the topic, and then hand-searched potentially eligible publications for this meta-analysis. To identify additional publications, we systematically searched electronic databases, using specific search terms as described below. No restriction of time or language was applied. This search was performed on May 31, 2013. We identified all articles included in prior meta-analyses[85–87] and additional studies. Addition of "fizzy", "artificially sweetened beverages" did not change the search results. We repeated electronic searches on February 10, 2014, restricting period of publication from June 1 to 'present'.

OVID and Embase: (("soda" OR "pop" OR "juices" OR "juice" OR "drink" OR "drinks" OR "beverage" OR "beverages") and ("diabetes") and ("prospective" OR "longitudinal" OR "cohort" OR "cohorts" OR "follow-up" OR "case-cohort" OR "nested case-control")) in abstract, title, and sub-headings; 599 hits on May 31, 2013, 52 hits on Feb 10, 2014

PubMed: ("soda"[tiab] OR "pop"[tiab] OR "juices"[tiab] OR "juice"[tiab] OR "drink"[tiab] OR "drinks"[tiab] OR "beverage"[tiab] OR "beverages"[tiab] OR "beverage[MeSH]" OR "beverages" [MeSH]) and ("diabetes"[tiab] or "diabetes" [MeSH]) and ("prospective"[tiab] OR "longitudinal"[tiab] OR "cohort"[tiab] OR "cohorts"[tiab] OR "follow-up"[tiab] OR "nested case-control"[tiab] OR "case-cohort"[tiab] OR "Prospective Studies"[Mesh] OR "Cohort Studies"[Mesh] OR "Longitudinal Studies"[Mesh]); 477 hits on May 31, 2013, 30 hits on Feb 10, 2014

Web of Knowledge: Topic=(juice* OR beverage* OR drink OR drinks OR soda OR pop) AND Topic=(diabetes) AND Topic=(prospective OR longitudinal OR cohort* OR follow-up OR case-cohort OR nested case-control); 1556 hits on May 31, 2013, 94 hits on Feb 10, 2014

Open Grey: (juices OR juice OR drink OR drinks OR beverage OR beverages OR soda OR pop) AND (diabetes) AND (prospective OR longitudinal OR cohort OR cohorts OR follow-up OR case-cohort OR nested case-control); 0 hit on May 31, 2013, 0 hits on Feb 10, 2014

Identification of studies and contact to authors

The articles reviewed in full-text are presented in **Table S1**. From each cohort, we hand-searched multiple publications and examined availability of information on dietary consumption and incident T2D.

We contacted authors of the identified articles between October and December in 2013 and requested information needed for this meta-analysis to minimize publication bias. If a publication reported estimates based on either continuous or categorical variables of beverage consumption for both adiposity-adjusted and unadjusted associations, we did not request additional data. In the absence of these estimates, we requested estimates based on continuous and categorical variables. We sent a reminder two weeks after an initial contact, in case we received no reply.

Quality assessment

We collected information to identify potential bias, in concordance with A Cochrane Risk of Bias Assessment Tool[88] and for Non-Randomized Studies of Interventions (ACROBAT-NRSI)[89]. As instructed by the Bias Assessment Tool[88], a 'high', 'low', or 'unknown' risk of bias in each study was assigned to seven different bias domains[89] and overall risk of bias (**Table S2**). Considerations for bias corresponding to seven domains and overall bias are described here:

- Confounding: Residual confounding is likely in any of observational research. Thus, a 'low' risk of bias was not assigned to any studies, as anticipated.[89] A 'high' risk was assigned to the E3N cohort, which might fail to adjust for adiposity in analysis of ASB.[32,44–46] Potential confounders adjusted for in each study are summarized in **Table S4**. With exception of adiposity measures, there was little indication of bias due to confounding by each of socio-demographic variables, lifestyle factors, and other covariates, in a multivariable model specified in each study. However, comparison between crude and adjusted analyses indicates confounding in analysis of each beverage, particularly of ASB (**Table S4; Table 2**).
- Selection: Selection of participants into a study would cause bias, if selection were related to both beverage consumption and incidence of T2D. This possibility was not identified in any of the studies. Selection was partly based on completion of data in any studies and considered in the assessment of the domain of missing data (see below).

• Measurement errors: Because any dietary assessments involve measurement errors, any studies had risks of this bias. However, inclusion in this meta-analysis incorporated the quantity of the bias partly (see next subsection). Accounting for it, a 'high' risk of bias was assigned to studies that did not verify quality of a diet-assessment method within a study population[23,25,26,42,50,53–55]; or studies that assessed diets during a month or less and did not confirm long-term reproducibility of dietary measures.[12,52,53]
• Misclassification of exposure: We focused on exposure assessment during follow-up. A 'low' risk of bias was assigned to studies that assessed dietary exposure repeatedly and incorporated them in analysis.[14–17,26,35]
• Missing data: All studies excluded participants with missing information. The number of participants excluded was not large in each study, the exclusion was considered to be unlikely to cause bias. A 'high' risk was assigned to two studies[12,34] because participants were excluded based on missing outcomes, which might cause attrition bias, during the follow-up: deaths in the Singapore Chinese Health Study (15% of adults) and unknown loss to follow-up in the High-risk and Population Strategy for Occupational Health Promotion Study (31% of adults).
• Outcome assessment: A 'low' risk was assigned to studies that attempted to minimize both false-positive and false-negative cases in a whole cohort by using objective information on incidence of T2D. A 'high' risk would have been assigned if a differential misclassification had occurred. This bias was not indicated in any cohorts.
• Selective reporting: A 'high' risk was assigned to four studies that reported estimates of associations selectively on the basis of whether or not findings were significant or not[23,25,35,36]; 'unknown', to two studies, on the basis of multiple analyses in different sub-groups, that presented inconsistent methods across articles from each cohort for similar research questions.[21,22,28,30]
• Overall bias: We considered multiple sources of bias in each study and ACRBAT-NRSI's anticipation that an observational study is unlikely to be at low risk of bias[89]. Here we describe a primary concern of bias for studies we rated as 'high' risk of bias. We rated two studies were at high risk of bias due to misclassification of types of beverage[25,48]; one study, due to lack of clarity based on documentation of a conference abstract only and potential publication bias[23]; one study, due to substantial bias due to confounding[44–47]; one study with substantial inconsistency in analytic methods across publications, indicating selective reporting[21]; and one study, due to loss of adults during follow-up[51]. These studies had <20% of weights in the main meta-analyses, and exclusion of these studies did not change results (see sensitivity analysis, below).

Adjustment for within-person variation of beverage consumption
In epidemiologic studies on dietary habits and other exposure related to chronic diseases, random within-person variability is concerning as a source of bias.[90] We applied statistical correction for the potential bias, using measures of the within-person variability, in addition to false-positive ascertainment of self-reported T2D (**Table S3**)[91,92]. Information extracted and assumptions are presented here in compliance with PRISMA.

We extracted correlation coefficients (r) between estimates from the two methods compared; ratios of two standard deviations (SDs) from the two dietary methods (s_{obs}/s_{ref}); and sample sizes (n). For studies without these measures derived within a study population[13,20,23,25,38], we extracted information from external sources, assuming consistency of within-person variations of dietary assessments in different cohorts.[90,93–98] This assumption was supported previously[67,99] and also by Atherosclerosis Risk in Communities Study (ARIC) that confirmed correlations of sugar intakes based on FFQ externally developed with a biomarker of sugar intakes.[100]

Kuopio Ischaemic Heart Disease Risk Factors Study in Finland evaluated beverage consumption by 4-day diet records implemented only at baseline.[63] A single 4-day diet record is unlikely to capture habitual diets.[66] Thus, we assumed similarity between r of single 4-day diet records and r of a seasonal variation of diet within a year; and took the measure from another study assessing diets among men in North Sweden[53] selected by demographic similarity.[63,67] We also assumed no error in a between-individual SD in the cohort (s_{obs}/s_{ref}, =1.0).

European Investigation into Cancer and Nutrition Study (EPIC)-InterAct carried out analyses pooling cohorts across Europe.[31,33] We extracted within-person variations of participating cohorts[43,53,69–78] (available on request) and pooled estimates by weights contributing to EPIC-InterAct's estimates[31].

When there was no information on measures of validity specifically for each type of beverages, we used information on foods or nutrients that were likely to have similar measures of the variations of consumption, as performed on another topic.[101] For example, if only non-alcohol beverages were assessed, we used them. If nutrients, not foods, were assessed, we extracted information on sucrose, disaccharides or total carbohydrates as surrogates for SSB and ASB; these sugars are not in ASB, but we assumed similarity in within-person dietary variations between ASB consumption and sugar intake.

We adopted a model used in prior meta-analyses[90,93–98] to adjust diet-disease association for a within-person dietary variation: $f(risk)=\alpha+\beta_{true}\cdot x_{true}$ and $\beta_{true}=\beta_{obs}/\gamma$, where x_{true} is true dietary factor and β_{true} is unobserved, unbiased log(RR) without a within-person variation (σ^2_{within}). β is attenuated to be β_{obs} by degree of γ, a attenuation factor, representing a variance ratio: $\sigma^2_{within}/(\sigma^2_{within}+\sigma^2_{between})$. In each cohort, γ was calculated by $\gamma=s_{ref}/s_{obs}\cdot r$, given a linear regression of $x_{true}=\alpha+\beta\cdot x_{obs}$. Dietary habits were measured repeatedly in six cohorts to minimize regression dilution or a degree of attenuation (**Table S2**; **Table S3**).[14,16–19,21,22,27,35] To account for this, γ was recalibrated for the number of repeated measures and measures of reproducibility[90]. Measures of reproducibility were obtained from existing literature along with those of validity (data not shown).

Adjustment for precision of incident type 2 diabetes

Some studies used self-reported T2D only[15–19,23,28,35,102] (**Table S3**), raising possibility of false-positive diagnosis expressed as positive predictive value (PPV). Thus, correction for PPV<1.0 was applied.[91,92] We assumed PPV=1 for studies using objective measures of T2D diagnosis. In CARDIA. two studies on beverages[21,26] ascertained cases with hyperglycaemia, not T2D. Thus, calibration in CARDIA was applied throughout in the meta-analysis, assigning PPV as a proportion of T2D cases among those with incident hyperglycemia[58,62]

Assessment of heterogeneity

Meta-regression was used to assess potential sources of heterogeneity (**Table S5**). Estimates used were those adjusted for adiposity, within-person dietary variation, and precision of T2D; results were similar in post hoc meta-regression using estimates without adjustment for within-person dietary variations (data not shown). Variables assessed by meta-regression were pre-specified, including study-specific factors: geographical location (the United States or Europe, or Asia, categorized *post hoc*), average age (years), sex (% men), average BMI (kg/m^2), follow-up duration (years), absolute risk of T2D (cases / person-years), methods of dietary assessments (FFQ, diet history), and methods of T2D diagnosis (self-reported, others). Assessment of publication status (published or not) was additionally evaluated *post hoc* after we collected all the data and recognized the potential importance of the variable. In stratified analysis for a continuous variable, a median across identified cohorts was used.

Independent sources of heterogeneity were selected by meta-regression with forward-variable selection. If variables in meta-regression showed $P<0.20$, the variable with the lowest P-value was retained in the model. Then, adjusting for the variable retained, mete-regression was repeated for remaining variables. If any of additional variables did not produce $P<0.20$, the model was considered best fitted. A variable with $P<0.10$ was considered as a significant source of heterogeneity and meta-analysis stratified by the factor was performed.[103]

Sensitivity analysis

We performed sensitivity analysis to confirm robustness of our findings against decision of modelling, assumptions and different use of available information (**Table S6)**. Sensitivity analysis included influence analysis[47] by excluding a single study and repeating random-effects meta-analysis (**Figure S3**). We also took an iterative stochastic sensitivity analysis, accounting for additional uncertainty of adjustment for within-person variations and precision of T2D diagnosis.[47,79,82] To confirm stability of our main analysis, we repeated the main meta-analysis (10,000 times) after ln(RR), γ and PPV were randomly drawn from each standard errors (SE). SE of ln(RR) was obtained by dose-response estimation; SE of γ, derived from information available in published records assessing within-person variations; SE of PPV, derived from 95% confidence interval (CI) calculated by Wilson score interval[104] or, when PPV=1, the rule of three.[105] In each iteration, trim-and-fill analysis was applied, to control for publication bias.[106] Medians of ln(RR) and 95% confidence limits of ln(RR) were used as the estimate accounting for uncertainty of our approach.[82]

Sensitivity analysis for residual confounding

We included a simulation-based analysis to examine influence of residual confounding. We recognized that the abovementioned correction for within-person variations was not applied to confounders.[6] Residual confounding by within-person variation of adiposity measures would be expected and crucial source of bias.[31,107] Adjustment for the bias could have been done, using measures of the within-person variation of adiposity and associations of adiposity with beverage consumption and incident T2D. Because the information was not available in any studies, we did *post hoc* analysis of simulation extrapolation (SIMEX), an imperfect, but useful, technique when structure of measurement errors is likely to be complex or unknown.[84]

In SIMEX, we used estimates after adjustment for potential publication bias by trim-and-fill method to control for publication bias. Inference became similar without trim-and-fill method (data not shown). In SIMEX, first, we assumed that adiposity was measured with within-person variation (r_a): when $r_a=1.0$, observed ln(RR) adjusted for adiposity would be unbiased; when $r_a=0$, adiposity measures would be a random variable and

ln(RR) would be the same as ln(RR) unadjusted for adiposity. We assumed r_a =0.9, 0.8, and 0.7 based on a study assessing validity of adiposity measures.[83]

We assumed that ln(RR) adjusted for measured adiposity could be expressed as $ln(RR) = \alpha + \beta \cdot tanh^{-1}(r_a)$ (Model A). This model supports that, r_a=0 would make ln(RR) equivalent to ln(RR) unadjusted for adiposity. Then, α and β could be readily solved algebraically after specifying r_a, observed ln(RR) unadjusted for adiposity measures, and observed ln(RR) adjusted for adiposity measures.

Separately, a non-linear SIMEX formula was modelled: $ln(RR) = a + 1/(b + c\theta)$ (Model B)[84], where a, b, and c were constants; and θ was a degree of within-person variations of measured adiposity, following $r_a = \frac{\sigma_{between}}{\sigma_{between}+\sigma_{within}(1+\theta)}$. This denotes that, when θ=0, ln(RR) was ln(RR) adjusted for adiposity, given r_a assumed; and when θ=∞, ln(RR) would be unadjusted for adiposity measures.

We used the Model A and B with r_a={0.9, 0.8, 0.7} and solve relationships between ln(RR) and θ. We then obtained a, b and c based on θ={0, 0.5, 1.0, 2.0}. Finally, using $ln(RR) = a + 1/(b + c\theta)$, we extrapolated ln(RR) of $\theta = -1$, producing $\sigma_{within} = 0$ and ln(RR) corrected for mis-measurements of adiposity.[84]

Estimation of type 2 diabetes events over 10 years from 2010 attributable to consumption of sugar-sweetened beverages in the United States and in the United Kingdom.
We estimated population attributable fraction (PAF) for T2D due to SSB consumption. We evaluated adults aged 20 years or older and free of T2D who participated in each of the national dietary surveys: US National Health and Nutrition Examination Survey (US NHANES), 2009-2010[108]; and the UK National Dietary Nutrition Survey (UK NDNS), 2008/2009-2011/2012[109]. The contemporary national surveys strengthen the implication from the present meta-analysis, beyond estimation solely relying on cohorts limited in generalizability to a general population.[110] Moreover, the use of individuals' data can avoid some assumptions often adopted in estimation of population-level impact: for example implausible assumption of normal distribution of dietary consumption and no correlations between different risk factors.[110–112]

Overall, in each survey, we (1) estimated habitual consumption of SSB among adults; (2) predicted 10-year risk ('assumed control risk', *ACR*[113]) of developing T2D of each adult; (3) estimated separate ideal 10-year risk (R_i) for each adult if SSB consumption was reduced to zero; and (4) estimated ($ACR–R_i$) for each adult and Σ(ACR-R_i) × population size as a number of cases attributable to SSB consumption in a population. PAF was derived as Σ(ACR-R_i)/ Σ(ACR).

As a simple example, if one adult consumed 1 serving/day of SSB and had ACR of 0.10 and if SSB consumption became zero, his or her risk (R_i) would be 0.10/1.13=0.088, where 1.13 is RR adjusted for adiposity. This calculation was applied to all adults, and pooling them as Σ(ACR) and Σ(R_i), the population-based estimates were obtained.[110] This estimation has advantage that there is no need of assumption in exposure distribution.

We used two RR separately: RR unadjusted for adiposity and RR adjusted for adiposity (1.18 and 1.13, respectively). We did not use RR unadjusted for within-person dietary variation, because the 10-year risk prediction was based on T2D risk factors unadjusted for within-person variations; and because reduction of SSB consumption would occur with a random within-person fluctuation of SSB consumption. In addition to uncertainty in this probability-weighting analysis, the uncertainty of RR was incorporated by one thousand iteration varying RR as normally distributed with variance of ln(RR).

The next subsections describe each estimation of PAF in the US and the UK, followed by description about validation analysis implemented by using the US NHANES.

Population attributable fraction for T2D due to SSB in the United States
In the US NHANES, we evaluated 4,729 non-diabetic adults who represented 189,075,538 adults in the US 2009-2010 according to sampling probability, after excluding 5,928 individuals: 4,319 children and adolescents (age<20 years) and 1,033 adults with prevalent diabetes (13.7% in weighted analysis) defined by reported diagnosis or anti-diabetic drug use or by fasting glucose ≥7.0 mmol/L or glycated haemoglobin ≥6.5%.[114]

Habitual consumption of SSB was estimated by using two 24-hour recall and a dietary screener questionnaire simultaneously analysed through a method to minimize within-individual dietary variation.[115] Using a set of risk factors (z) for T2D, 10-year risk of T2D was estimated by using an algorithm developed in ARIC and validated in Multiethnic Study on Atherosclerosis (MESA), a community-based cohort in US.[116,117] The formula was a

logistic function, ACR=1/(1+*exp*(−*X*|*z*)). The original prediction was for a 9-year risk of T2D, and thus converted to a 10-year risk as Pr=1− (1−Pr)$^{10\div9}$. The model was developed among adults younger than 65 years, adopting a rare-disease assumption. Mortality, reducing T2D cases identified over 10 years, was accounted by age-sex-specific mortality due to non-diabetes cause (1-annual mortality)$^{10\ years}$, based on US vital statistics.[118]

Population attributable fraction for T2D due to SSB in the United Kingdom
We used the UK NDNS data collected in 2008/2009-2011/2012.[109] Sampling weights were applied, which appeared, unlike US NHANES, not to estimate an absolute number of adults in the UK. Thus, to estimate absolute numbers of T2D cases attributable to SSB, we used age-sex-specific population sizes in the UK in 2010 as the source population (47,704,520 adults in total, aged 20 or older).[119] Of the UK NDNS, we evaluated 1,932 non-diabetic adults, after excluding 2,096 children and adolescents (age<20 years) and 128 adults with prevalent diabetes (6.2% in weighted analysis, 2.9 million in UK) defined by diagnosis, anti-diabetic drug use, or compliance to an anti-diabetic diet assessed through an interview; or by fasting glucose ≥7.0 mmol/L or glycated haemoglobin ≥6.5%.

Habitual consumption of SSB was estimated by 4-day food records with within-individual dietary variation minimized.[115] Ten-year risk of T2D was estimated by a risk-prediction algorithm, QDScore®-2013[120], developed in the prospective analysis of nation-wide electronic records collected in UK general practice; validated externally[121,122]; and made publically available for research purpose.[120] The formula allowed estimation of ACR over 10 years from basic demographic variables, deprivation index, smoking status, use of an oral corticosteroid, use of an anti-hypertensive drug, prevalent cardiovascular diseases, family history of diabetes.[120–122] Family history of diabetes and Townsend deprivation index were not available in NDNS. These were imputed, respectively, by the population average as found in the nation-wide electronic record[121] and by household income, as recommended previously[121].

Validation of 10-year risk prediction
We assessed validity of 10-year risk prediction in US NHANES, using the 1999-2000 and 2009-2010 cycles. We first estimated prevalent T2D predicted by NHANES 1999-2000. Adults with T2D in 1999-2000 were assumed to have T2D in 2009-2010, with Pr= (1-annual mortality)^{10}. For non-diabetic adults, 10-year risk prediction was applied as described above. Then, the two numbers of T2D cases from T2D cases and non-cases in 1999-2000 were summed as the predicted number of T2D cases in 2009-2010. The sum was compared to observed number of cases in 2009-2010. The two estimates were not statistically different (*P*=0.48). The number of T2D cases in 2009-2010 predicted by NHANES 1999-2000 was 32.1 million [95% CI=27.1-37.1]); and that observed in NHANES 2009-2010 was (30.0 million [95% CI=26.7-33.3]). Based on this result, we considered validity of 10-year risk prediction to be sufficient in this work.

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