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Original Research Article

Sugar-sweetened beverage consumption and weight gain in children and adults: a systematic review and meta-analysis of prospective cohort studies and randomized controlled trials☆

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ABSTRACT

Background: Sugar-sweetened beverages (SSBs) have been implicated in fueling the obesity epidemic.

Objectives: This study aimed to update a synthesis of the evidence on SSBs and weight gain in children and adults.

Methods: MEDLINE, Embase, and Cochrane databases were searched through September 8, 2022, for prospective cohort studies and randomized controlled trials (RCTs) that evaluated intake of SSBs in relation to BMI and body weight in children and adults, respectively. Eligible interventions were compared against a noncaloric control. Study-level estimates were pooled using random-effects meta-analysis and presented as β-coefficients with 95% CIs for cohorts and weighted mean differences (MDs) with 95% CIs for RCTs.

Results: We identified 85 articles including 48 in children (40 cohorts, n = 91,713; 8 RCTs, $n = 2783$) and 37 in adults (21 cohorts, $n = 448{,}661$; 16 RCTs, $n = 1343$). Among cohort studies, each serving/day increase in SSB intake was associated with a 0.07-kg/m^2 (95% CI: 0.04 kg/m^2, 0.10 kg/m^2) higher BMI in children and a 0.42-kg (95% CI: 0.26 kg, 0.58 kg) higher body weight in adults. RCTs in children indicated less BMI gain with SSB reduction interventions compared with control (MD: −0.21 kg/m^2; 95% CI: −0.40 kg/m^2, −0.01 kg/m^2). In adults, randomization to addition of SSBs to the diet led to greater body weight gain (MD: 0.83 kg; 95% CI: 0.47 kg, 1.19 kg), and subtraction of SSBs led to weight loss (MD: −0.49 kg; 95% CI: −0.66 kg, −0.32 kg) compared with the control groups. A positive linear dose–response association between SSB consumption and weight gain was found in all outcomes assessed.

Conclusions: Our updated systematic review and meta-analysis expands on prior evidence to confirm that SSB consumption promotes higher BMI and body weight in both children and adults, underscoring the importance of dietary guidance and public policy strategies to limit intake. This meta-analysis was registered at the International Prospective Register of Systematic Reviews as CRD42020209915.

Keywords: sugar-sweetened beverage, obesity, weight gain, systematic review, meta-analysis

Abbreviations used: BMI, body mass index; CI, confidence interval; FFQ, food frequency questionnaire; HFCS, high-fructose corn syrup; GRADE, Grading of Recommendations Assessment Development and Evaluation; MD, mean difference; NOS, Newcastle–Ottawa Scale; RCT, randomized controlled trial; SD, standard deviation; SE, standard error; SSB, sugar-sweetened beverage.

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Introduction

Despite current public health efforts, obesity has become a global epidemic. Among health risks, excess body weight continues to have the highest rate of increase worldwide [1]. In 2016, an estimated 1.9 billion adults were classified as being overweight, and 650 million adults were classified as being obese, accounting for ~4 million deaths [2, 3]. Alarmingly, the rate of increase in obesity among children and adolescence has surpassed that of adults [1]. The prevalence of childhood obesity has increased more than 4-fold globally since 1975 [3]. This is of particular concern as children with obesity are more likely to have obesity in adulthood, along with associated cardiometabolic and other health risks. Overweight and obesity are important risk factors for noncommunicable diseases, such as cardiovascular disease, type 2 diabetes, and some types of cancer, which have been found to have similar increases in prevalence [4, 5].

Concurrent with the obesity epidemic has been the overconsumption of sugar-sweetened beverages (SSBs) [6]. SSBs, which include sodas, fruit drinks, sports drinks, and energy drinks, are high in caloric sweeteners, including sucrose and high-fructose corn syrup (HFCS). These beverages contribute to high amounts of empty calories, as they are energy dense yet offer no other nutritional value. SSBs are the largest source of added sugar in the North American diet, and despite reductions in intake over the past decade, consumption remains high [7–10]. For these reasons, intake of SSBs has been implicated as an important contributor to overweight and obesity across the globe.

Our previous systematic review and meta-analysis through March 2013 found consistent evidence from cohort studies and trials linking intake of SSBs to excess adiposity [11]. Over the past decade since, additional large prospective cohort studies and randomized controlled trials (RCTs) have been conducted. This further evidence needs to be incorporated into summaries of literature to inform policies, update guidelines, and support clinicians to make evidence-based recommendations about SSBs. Therefore, we conducted a systematic review and meta-analysis to update the evidence on SSB intake in relation to change in adiposity in children and adults. Additionally, we have expanded the scope of this meta-analysis to include a novel dose–response analysis to further inform SSB guidance.

Methods

Protocol and registration

We followed the Cochrane Handbook for Systematic Reviews of Interventions [12] to conduct this systematic review and meta-analysis. Results are reported in compliance with the Meta-analysis of Observational Studies in Epidemiology guidelines [13] for prospective cohort studies and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines [14] for RCTs. The study protocol was prospectively registered at the International Prospective Register of Systematic Reviews (ID: CRD42020209915).

Search strategy and study selection

We searched MEDLINE, Embase, and Cochrane databases using the search terms presented in Supplemental Table 1. A manual search through databases and reference lists was additionally conducted. Searches were performed through September 8, 2022.

Our review included prospective cohort studies and RCTs reporting associations for SSB consumption on weight gain in children and adolescents (aged <18 y) and adults (aged ≥18 y). We defined SSBs as beverages with added sugars, such as soft drinks and fruit drinks. We did not include 100% fruit juice in these analyses as we are conducting a separate meta-analysis on fruit juice and weight gain, and we considered noncaloric sweetened beverages as a comparator for RCTs. We included studies with a minimum duration of 6 mo for prospective cohort studies and 2 wk for RCTs as this is sufficient time to assess the outcomes of body weight and body mass index (BMI) gain. RCTs were eligible if the intervention added (i.e., addition trials) or subtracted (i.e., subtraction trials) SSB intake in adults or subtracted SSB intake in children. Only subtraction trials were included for children as no study has assessed the addition of SSB intake in children. Studies with a mixed intervention or those that combined SSBs with other foods, supplements, or lifestyle factors were excluded. Trials with isocaloric controls, such as fruit juices or milk, were additionally excluded because a mechanistic hypothesis is that SSBs contribute to weight gain by increasing energy intake. In studies with duplicate populations, the study with the largest sample size or that included a change versus change analysis was selected. No language restrictions were applied; however, only studies written in English or translated to English were considered. Titles and abstracts were screened, and relevant articles were reviewed by 2 independent reviewers (MN and SEJ or MGT). Discrepancies were resolved by consensus or discussion with the senior author (VSM).

Data extraction and quality assessment

At least 2 reviewers (MN and SEJ or MGT) independently extracted data from eligible studies including information on study characteristics (study design, sample size, blinding, duration, study setting, and covariates), participant characteristics (sex, age, disease status, and baseline body weight), dietary and outcome assessment methods, intervention or exposure, comparator, dose of SSB, funding source, and whether weight gain was the primary outcome. Data were extracted for the association of SSBs and body weight for adults or BMI for children. We did not evaluate the relation between waist circumference and SSBs because the outcomes of body weight and BMI can be more directly translated into public health recommendations and are more easily understood by the general public. For prospective cohort studies, multivariable-adjusted β-coefficients and the standard errors (SEs) were extracted (see Supplemental Table 2). Because of the potential mediation of total energy intake on the association between SSBs and weight gain, we extracted data with and without adjustments for total energy when possible. For RCTs, the change from baseline differences for the intervention and control groups were extracted as means ± standard deviations (SDs). If unavailable, they were derived from available data using published formulas [12]. For trials with multiple comparator interventions, we chose the control most relevant to our research question; for example, in studies comparing SSBs with water or a diet beverage, we chose the control of a diet beverage because of public health relevance. The authors were contacted to provide additional outcome or study details. In studies where data were presented graphically and numerical values were not available, Plot Digitizer V.2.6.9 was used.

The risk of bias for each study was assessed by 2 independent reviewers. For prospective cohort studies, we used the Newcastle–Ottawa Scale (NOS) [15] to assign points according to 3 domains: *1*) selection, *2*) comparability, and *3*) exposure. A maximum of 9 points could be awarded, and studies with a score of ≥6 points were considered to be of high quality. For RCTs, the Cochrane risk-of-bias tool [12] was used to assign a "high risk," "unclear risk," or "low risk" according to 5 domains: *1*) sequence generation, *2*)

allocation concealment, *3*) blinding, *4*) incomplete outcome data, and *5*) selective outcome reporting.

Data synthesis and analysis

Because of the variation in presented data for a number of studies, it was necessary to perform various transformations in the outcome data to obtain uniform units. The details of the transformations are shown in Supplemental Table 2.

For prospective cohort studies in children, our primary analysis assessed the change in BMI (kg/m^2) per every 12-oz serving/d increment of SSB during the duration specified in each study. Studies that reported SSBs in servings other than 12 oz [16–23], in grams [24–35], or in energy units [36, 37] were scaled accordingly (see Supplemental Table 2). In studies where the serving size was not specified, it was assumed to be 12 oz [38–52], which is equivalent to ~355 mL and typical for most glasses and cans. Studies that reported servings per week were converted to servings per day [20, 38, 43, 46, 49, 53]. Studies with effect estimates presented as BMI z-score [16, 18, 20, 22, 24, 25, 28, 31, 33, 35–37, 40, 47–50, 54,55] were converted to BMI using the lambda–mu–sigma method [56], and studies that reported changes in fat mass were converted to BMI [19], making the assumption that differences in fat mass were equal to differences in body weight. Studies that reported effect estimates in at least 3 categories [20, 21, 34, 37, 38, 46–51] were converted to continuous variables by plotting the median intake of each group against weight change and using least squares linear regression to obtain the β and SE, with the assumption of linearity. Several studies reported the change in SSB intake in relation to change in BMI [18, 24, 27, 36–39, 41–44, 46, 54, 55]. A change versus change meta-analysis was conducted separately and scaled for the 1-y change in BMI per serving/day increment of SSB. Nonenergy intake-adjusted models were included in the primary meta-analysis when possible, and a separate meta-analysis was conducted that included energy-adjusted models. Additionally, because of the number of estimates presented as BMI z-score, we conducted a separate meta-analysis assessing the association of SSBs and BMI z-score.

For prospective cohort studies in adults, our primary analysis assessed the change in body weight (kg) per every 12-oz serving/d increment of SSB during the duration specified in each study. Similar transformations were performed to scale reported units to 12-oz/d serving sizes [57–72] and from categorical to continuous data [62, 65, 71–74] (see Supplemental Table 2). Several studies reported the change in SSB intake in relation to change in body weight [57–61, 63–67, 69–71, 75, 76]; thus, a separate meta-analysis was conducted using these studies to show the 1-y change in weight per serving/day increment of SSB. An additional meta-analysis was conducted that included effect estimates that were adjusted for total energy.

For RCTs in children, estimates were transformed to express the mean difference (MD) in BMI (kg/m^2) change between the intervention and control groups. Two trials reported estimates as BMI z-score and were, thus, converted to BMI [77, 78]. For RCTs in adults, estimates were transformed to express the MD in body weight (kg) change between the intervention and control groups. When not provided directly, the differences between the end and baseline values of the intervention and control groups were calculated, and a standard formula was used to calculate the SD [12]. When calculating within-group differences and for crossover trials in pairwise analyses, a correlation coefficient of 0.95 was used to calculate the SD of the MD [79]. The correlation coefficient of 0.95 was used as the correlation between body weight at 2 time points was assumed to be high [11].

Data analyses were conducted in Review Manager version 5.4 (The Nordic Cochrane Centre, The Cochrane Collaboration) and STATA version 16.1 (StataCorp). The $\beta \pm$ SE for the cohort studies and MD $\pm$ SE for the RCTs were pooled for each outcome using the generic inverse-variance method with the DerSimonian and Laird random-effects model. The fixed-effects models were also conducted and evaluated. Pooled results are expressed as β-coefficients with 95% confidence intervals (CIs) for prospective cohort studies and MD with 95% CIs for RCTs.

Dose–response analysis was conducted for cohort studies and RCTs when $\geq$6 comparisons were available [80, 81]. Dose–response analysis was conducted for prospective cohort studies in children and adults to assess the change in BMI or body weight per 12-oz serving of SSB. The dose–response and primary analyses differed in the included studies and number of estimates based on how the models were calculated. We defined the dose of each category as the mean intake for the given group, and if not reported, we used the midpoint of the upper and lower bounds. For open-ended lower categories, we defined the dose as zero [82]. For open-ended upper categories, we added half of the category value to estimate the dose [82]. Dose–response estimates are reported as MDs against the reference category reported in each study. In studies where the categorical estimates were presented as means rather than MDs, we calculated the MD by subtracting each category against a reference group and used a standard equation to calculate the SE [12]. For cohort studies in adults, 2 studies reported negative doses as some groups indicated reduction in intakes [65, 67]. We excluded studies from the dose–response analysis for cohort studies that reported continuous outcomes, as nonlinear dose–response analyses could not be conducted. Dose–response analysis was additionally conducted for addition trials in adults using linear and nonlinear meta-regression. Dose–response analysis was not conducted for RCTs in children and subtraction trials in adults as these studies assessed the subtraction of SSBs from the diet and <6 comparisons with dose were available.

We calculated the I^2 statistic and *P* value for heterogeneity from the Cochrane Q statistic. An $I^2 \geq 50\%$ and $P < 0.10$ were considered to be indicative of substantial heterogeneity [12]. Potential sources of heterogeneity were explored through sensitivity and a priori selected subgroup analyses. A sensitivity analysis was conducted to assess the influence of individual studies by systematically removing each study from the meta-analysis and recalculating the pooled effect estimate and heterogeneity. The removal of any study that changed the direction or significance of the pooled effect or explained heterogeneity was considered an influential study. For outcomes with $\geq$10 observations, subgroup analyses were conducted to explore sources of heterogeneity using meta-regression (significance at $P < 0.05$). Subgroup analysis for prospective cohort studies included age (< median vs. $\geq$ median), baseline BMI for adults (<25 vs. $\geq$25 kg/m^2), study duration (< median vs. $\geq$ median months), source of funding (agency, industry, agency–industry, none, and not reported), dietary assessment method [food frequency questionnaires (FFQs), 24-h dietary recall, and diet records], adjustment for diet quality, adjustment for energy intake, outcome assessment method (measured by investigators vs. self-report), study location (North America, Europe, Asia, South America, and Australia), and NOS (<6 vs. $\geq$6). Subgroup analysis for RCTs included dose (< median vs. $\geq$ median), baseline BMI (<25 vs. $\geq$25 kg/m^2), age (< median vs. $\geq$ median), study duration (< median vs. $\geq$ median weeks), study design (crossover vs. parallel), source of funding (agency, industry, agency–industry, none, and not reported), intervention type (educational vs. direct), whether body weight was the

primary outcome (yes or no), study location (North America, Europe, Asia, South America, and Australia), and risk of bias (low, high, and unclear for the 5 domains of the Cochrane risk-of-bias tool). Stratified analysis by sex was not conducted because of the insufficient number of observations (<10 observations).

Publication bias was assessed for outcomes with ≥10 observations through visual inspection of contour-enhanced funnel plots and testing through Egger's and Begg's tests, where $P < 0.05$ was considered evidence for bias [83, 84]. If publication bias was detected, the Duval and Tweedie trim-and-fill method was used to impute missing study data to adjust for funnel plot asymmetry and assess for small-study effects [85].

Grading the certainty of evidence

The certainty of evidence was evaluated using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach [86] by 2 independent reviewers (MN and SBM). Each outcome can be assessed as "very low," "low," "moderate," or "high" certainty of evidence. By default, RCTs are initially rated as "high" certainty of evidence, and prospective cohort studies are initially rated as "low" certainty of evidence because of their inherent limitations. Criteria to downgrade the certainty of evidence include the risk of bias (assessed through Cochrane risk-of-bias tool and NOS), inconsistency (substantial unexplained interstudy heterogeneity, $I^2 \geq 50\%$), indirectness (factors that limit generalizability of results), imprecision (95% CIs for pooled effect estimates are wide or cross the minimally important difference for harm or benefit), and publication bias (assessed through Egger's and Begg's tests). Criteria to upgrade include the presence of a dose–response gradient. To supplement the GRADE approach, we cross-evaluated the evidence using NutriGrade, a scoring system that takes into account nutrition research-specific requirements [87]. The scoring system includes a total of 9 domains—*1*) risk of bias, study quality, and study limitations; *2*) precision; *3*) heterogeneity; *4*) directness; *5*) publication bias; *6*) funding bias; *7*) study design; *8*) effect size; and *9*) dose–response—with a maximum of 10 points to be awarded. Outcomes are assessed as "high-meta evidence" (8–10 points), "moderate-meta evidence" (6–7.99 points), "low-meta evidence" (4–5.99 points), and "very-low-meta evidence" (0–3.99 points).

Results

Search results

Our search identified 14,129 citations, of which 203 articles were reviewed in full, and 85 were included in the final review (see Figure 1) [16–55, 57–78, 88–110]. Of these, 40 articles were prospective cohort studies in children (35 articles included in the primary analysis with 45 comparisons and 5 used for the dose–response analysis only), 21 were

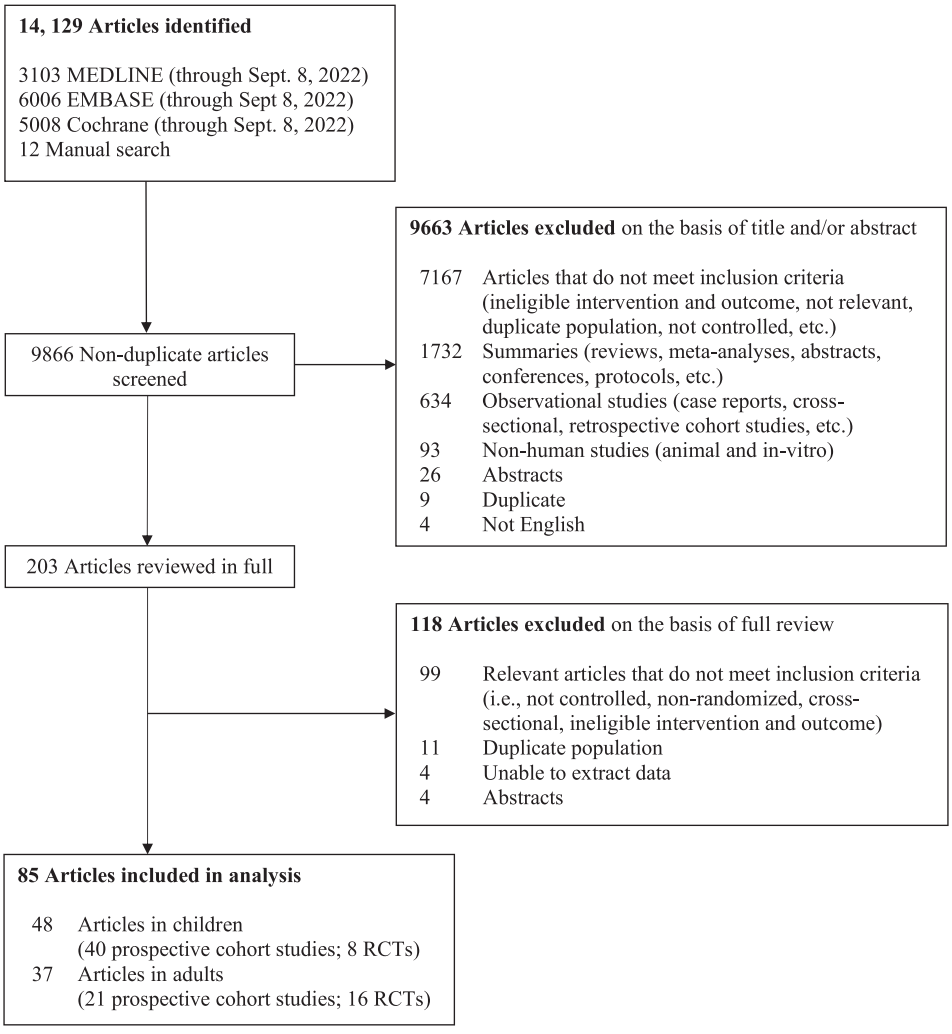

FIGURE 1. Flow of literature. RCT, randomized controlled trial.

TABLE 1
Characteristics of the included prospective cohort studies

Study characteristics[1]	Cohort studies in children	Cohort studies in adults
Number of cohorts	40	21
Study location	North America ($n = 16$), Europe ($n = 12$), Australia ($n = 5$), Asia ($n = 3$), South America ($n = 3$), South Africa ($n = 1$)	North America ($n = 12$), Europe ($n = 5$), Asia ($n = 4$)
Sample size (range)	1048 (98–11,654)	3552 (170–117,992)
Baseline age (range)	10 y (6 mo to 17 y)	46 y (18–75 y)
Baseline BMI[2] (range)	0.30 (−0.08 to 2.96)	26.3 kg/m^2 (21.9–32.0 kg/m^2)
Duration (range)	4 y (6 mo to 30 y)	4 y (1–24 y)
Dietary assessment method	FFQs ($n = 24$), 24-h recalls ($n = 9$), food records ($n = 7$)	FFQs ($n = 18$), 24-h recalls ($n = 2$), food records ($n = 1$)
Outcome assessment method	Conducted by investigators ($n = 36$), self-report ($n = 4$)	Conducted by investigators ($n = 14$), self-report ($n = 7$)
Adjusted for energy intake	Unadjusted ($n = 34$), adjusted ($n = 6$)	Unadjusted ($n = 17$), adjusted ($n = 4$)

[1] Medians.
[2] The baseline BMI z-score is presented for cohort studies in children.

prospective cohort studies in adults (20 included in the primary analysis with 21 comparisons and 1 used for the dose–response analysis only), 8 were RCTs in children, and 16 were RCTs in adults (11 addition trials with 12 comparisons and 5 subtraction trials).

Of the 118 excluded studies, 99 articles did not meet the inclusion criteria, 11 studies were duplicate populations, 4 studies were abstracts, and 4 studies did not provide data in the necessary units. The full list of excluded studies is presented in Supplemental Table 3.

Study characteristics

The characteristics of the included prospective cohort studies in children and adults are summarized in Table 1. Among the 40 prospective cohort studies in children, 40% ($n = 16$) were set in North America, 30% ($n = 12$) in Europe, 13% ($n = 5$) in Australia, 8% ($n = 3$) in Asia, 8% ($n = 3$) in South America, and 3% ($n = 1$) in South Africa. The median number of participants was 1048 (range, 98–11,654 participants), with a median study duration of 4 y (range, 6 mo to 30 y). Children had a median age of 10 y (range, 6 mo to 17 y) and median baseline BMI z-score of 0.30 (range, −0.08 to 2.96). The studies used a variety of dietary assessment methods, including FFQs (60%, $n = 24$), 24-h recalls (23%, $n = 9$), and food records (18%, $n = 7$), and the majority of outcome assessment measures were conducted by investigators (90%, $n = 36$), followed by self-reported measures (10%, $n = 4$). Among the 21 prospective cohort studies in adults, 57% ($n = 12$) were conducted in North America, 24% ($n = 5$) in Europe, and 19% ($n = 4$) in Asia. The median number of participants was 3552 (range, 170–117,992 participants), with a median study duration of 4 y (range, 1–24 y). Adults had a median age of 46 y (range, 18–75 y) and median baseline BMI of 26.3 kg/m^2 (range, 21.9–32.0 kg/m^2). Diet was assessed through FFQs (86%, $n = 18$), 24-h recalls (10%, $n = 2$), and food records (5%, $n = 1$), with the majority of outcome assessments measured by investigators (67%, $n = 14$), followed by self-reported measures (33%, $n = 7$).

The characteristics of the included RCTs in children and adults are presented in Table 2. Among the 8 RCTs in children, 63% ($n = 5$) were conducted in the United States, and 1 study each in the United Kingdom, the Netherlands, and Brazil. All trials followed a parallel design, with a median sample size of 164 participants (range, 51–927 participants) and median study duration of 6.5 mo (range, 3–18 mo). Children had a median age of 11 y (range, 4–15 y), and the majority were of normal weight status at baseline (75%, $n = 6$). Interventions in children consisted of replacement of SSBs with noncaloric beverages (38%, $n = 3$) or school-based educational programs to reduce SSB consumption (63%, $n = 5$). Among the 11 SSB addition trials in adults, the majority were conducted in Europe (73%, $n = 8$), followed by those in the United States (18%, $n = 2$) and 1 study in Brazil. Of these, 73% ($n = 8$) used a parallel design, and 27% ($n = 3$) used a crossover design, with a median sample size of 47 (range, 20–133 participants) and median study duration of 1 mo (range, 3 wk to 6 mo). The adults in the addition trials had a median age of 29 y (range, 22–42 y), and 55% ($n = 6$) of the studies had participants who were overweight at baseline, and 45% ($n = 5$) of the studies had participants who were normal weight at baseline. The inventions were compared with noncaloric beverages (73%, $n = 8$) or dietary advice to reduce SSB intake (27%, $n = 3$). Among the 5 SSB subtraction trials in adults, 40% ($n = 2$) were conducted in the United States, 40% ($n = 2$) in Mexico, and 1 study in Switzerland. All subtraction trials were parallel in design, with a median sample size of 120 participants (range, 27–240 participants) and median study duration of 6 mo (range, 3–12 mo). The adults in the subtraction trials had a median age of 30 y (range, 18–41 y), and all studies had participants who were overweight at baseline. All interventions were noncaloric beverages in replacement of SSBs. Detailed characteristic lists of all included studies are presented in Supplemental Tables 4–7.

Risk of bias

The risk of bias assessed through the NOS for prospective cohort studies is presented in Supplemental Table 8. Among the prospective cohort studies in children, 38 (95%) received a score of ≥6, and for adults, all studies received a score ≥6, denoting high-quality studies. Overall, there was no serious risk of bias in the included prospective cohort studies. The risk of bias assessed through the Cochrane risk-of-bias tool for RCTs is presented in Supplemental Figures 1 and 2. The overall risk of bias tended to be low or unclear in all domains assessed, indicating no serious risk of bias in the included RCTs.

SSBs and body weight in children

Prospective cohort studies.

Figure 2 presents the association between 1 serving/d increase in SSB and change in BMI based on 45 comparisons from 35 studies ($n = 71,681$). In our primary analysis, pooled multivariable-adjusted estimates using the random-effects model showed a positive association between SSB consumption and BMI. Each 12-oz serving/d greater consumption of SSBs was associated with a 0.07-kg/m^2 (95% CI: 0.04 kg/m^2, 0.10 kg/m^2; $P < 0.01$) higher BMI. Substantial between-study heterogeneity was observed in the analysis ($I^2 = 82\%$, P-heterogeneity < 0.01). In our secondary analyses, the results from the fixed-effects

TABLE 2
Characteristics of the included randomized controlled trials

Study characteristics[1]	Trials in children	Addition trials in adults	Subtraction trials in adults
Number of RCTs	8	11	5
Study location	North America ($n = 5$), Europe ($n = 2$), South America (n = 1)	North America ($n = 2$), Europe ($n = 8$), South America (n = 1)	North America ($n = 4$), Europe ($n = 1$)
Study design	Parallel ($n = 8$)	Parallel ($n = 8$), crossover ($n = 3$)	Parallel ($n = 5$)
Sample size (range)	164 (51–927)	47 (20–133)	120 (27–240)
Baseline age (range)	11 y (4–15 y)	29 y (22–42 y)	30 y (18–41 y)
Baseline weight status	Normal ($n = 6$), overweight ($n = 2$)	Normal ($n = 5$), overweight ($n = 6$)	Overweight ($n = 5$)
Baseline BMI[2] (range)	0.44 (0.03–1.00)	25.2 kg/m^2 (21.8–2.8 kg/m^2)	30.6 kg/m^2 (25.7–36.5 kg/m^2)
Duration (range)	6.5 mo (3–18 mo)	1 mo (3 wk to 6 mo)	6 mo (3–12 mo)
Intervention	Noncaloric beverage (n = 3), school-based educational programs to reduce SSB consumption (n = 5)	SSBs ($n = 11$)	Noncaloric beverage ($n = 5$)
Control	Continue SSB consumption (n = 3), general health advice/programing (n = 4), not specified (n = 1)	Noncaloric beverages (n = 8), dietary advice to reduce SSB consumption (n = 3)	Continue SSB consumption (n = 2), ad libitum diet (n = 2), nutrition counseling (n = 1)

RCT, randomized controlled trial; SSB, sugar-sweetened beverage.
[1] Medians.
[2] The baseline BMI z-score is presented for trials in children.

model were similar (0.07 kg/m^2; 95% CI: 0.07 kg/m^2, 0.08 kg/m^2; $P <$ 0.01; $I^2 = 82\%$; P-heterogeneity < 0.01). The results were similar when energy-adjusted estimates were included in the analysis (0.07 kg/m^2; 95% CI: 0.04 kg/m^2, 0.10 kg/m^2; $P < 0.01$; $I^2 = 85\%$; P-heterogeneity < 0.01) (see Supplemental Figure 3). In our analysis of change in BMI z-score, each serving/day greater consumption of SSB was associated with a 0.03 (95% CI: 0.02, 0.05; $P < 0.01$; $I^2 = 84\%$; P-heterogeneity < 0.01) higher BMI z-score (see Supplemental Figure 4).

The analysis evaluating the 1-y change in SSB intake with concomitant 1-y change in BMI is presented in Supplemental Figure 5, including 19 comparisons from 14 studies ($n = 36{,}933$). The pooled estimate showed a 0.03-kg/m^2 (95% CI: 0.01 kg/m^2, 0.04 kg/m^2; $P <$ 0.01) higher BMI for each additional daily serving of SSB over a 1-y period. Substantial between-study heterogeneity was observed ($I^2 =$ 76%, P-heterogeneity < 0.01).

Randomized controlled trials.

Figure 3 presents the effect of subtracting SSB intake on BMI change in 8 trials of children and adolescents ($n = 2783$). In our primary analysis, pooled effects using the random-effects model indicated a significant effect of reducing SSB consumption on BMI change (MD: −0.21 kg/m^2; 95% CI: −0.40 kg/m^2, −0.01 kg/m^2; $P = 0.04$). Substantial between-study heterogeneity was observed in the analysis ($I^2 =$ 99%, $P < 0.01$). In our secondary analyses, the results from the fixed-effects model were similar (MD: −0.34 kg/m^2; 95% CI: −0.36 kg/m^2, −0.33 kg/m^2; $P = 0.02$; $I^2 = 99\%$; P-heterogeneity < 0.01). Our analysis evaluating the effect of SSB reduction in the subset of studies with BMI z-scores showed no statistically significant effect (MD: −0.11; 95% CI: −0.24, 0.03; $P = 0.11$; $I^2 = 99\%$; P-heterogeneity $<$ 0.01) (see Supplemental Figure 6).

SSBs and body weight in adults

Prospective cohort studies.

Figure 4 presents the association between 1 serving/d increase in SSBs and change in body weight based on 21 comparisons from 20 studies ($n = 343{,}651$). In our primary analysis, pooled multivariable-adjusted estimates using the random-effects model showed a positive association between SSB consumption and body weight. Each 12-oz serving/d greater consumption in SSBs was associated with a 0.42-kg (95% CI: 0.26 kg, 0.58 kg; $P < 0.01$) higher body weight. Substantial between-study heterogeneity was observed ($I^2 = 90\%$, P-heterogeneity < 0.01). In our secondary analyses, the results from the fixed-effects model showed a weaker association (0.23 kg; 95% CI: 0.20 kg, 0.27 kg; $P < 0.01$; $I^2 = 88\%$; P-heterogeneity < 0.01). When the model was additionally adjusted for total energy intake, the association was modestly attenuated (0.39 kg; 95% CI: 0.24 kg, 0.54 kg; $P < 0.01$; $I^2 =$ 89%; P-heterogeneity < 0.01) (see Supplemental Figure 7).

The analysis evaluating the 1-y change in SSB intake with concomitant 1-y change in weight is presented in Supplemental Figure 8, including 16 comparisons from 15 studies ($n = 247{,}772$). The pooled estimate showed a 0.20-kg (95% CI: 0.13 kg, 0.28 kg; $P <$ 0.01) higher body weight for each additional daily serving of SSB over a 1-y period. The presence of substantial interstudy heterogeneity was observed ($I^2 = 81\%$, P-heterogeneity < 0.01).

Randomized controlled trials.

Figure 5 presents the effect of the addition of SSBs on body weight change in 12 trial comparisons in adults ($n = 558$). In our primary analysis, pooled effects using the random-effects model showed a significant effect of SSB consumption on body weight (MD: 0.83 kg; 95% CI: 0.47 kg, 1.19 kg; $P < 0.01$). There was evidence of substantial between-study heterogeneity ($I^2 = 87\%$, P-heterogeneity < 0.01). In our secondary analyses, the results from the fixed-effects model were similar (MD: 0.93 kg; 95% CI: 0.84 kg, 1.01 kg; $P < 0.01$; $I^2 = 87\%$, P-heterogeneity < 0.01). Figure 6 presents the effect of the subtraction of SSBs on body weight change in 5 trials in adults ($n = 694$). In our primary analysis, pooled effects using the random-effects model showed a significant effect of SSB reduction on body weight (MD: −0.49 kg; 95% CI: −0.66 kg, −0.32 kg; $P < 0.01$). There was no evidence of between-study heterogeneity ($I^2 = 0\%$, P-heterogeneity $<$ 0.58). The results from the fixed-effects model were equivocal.

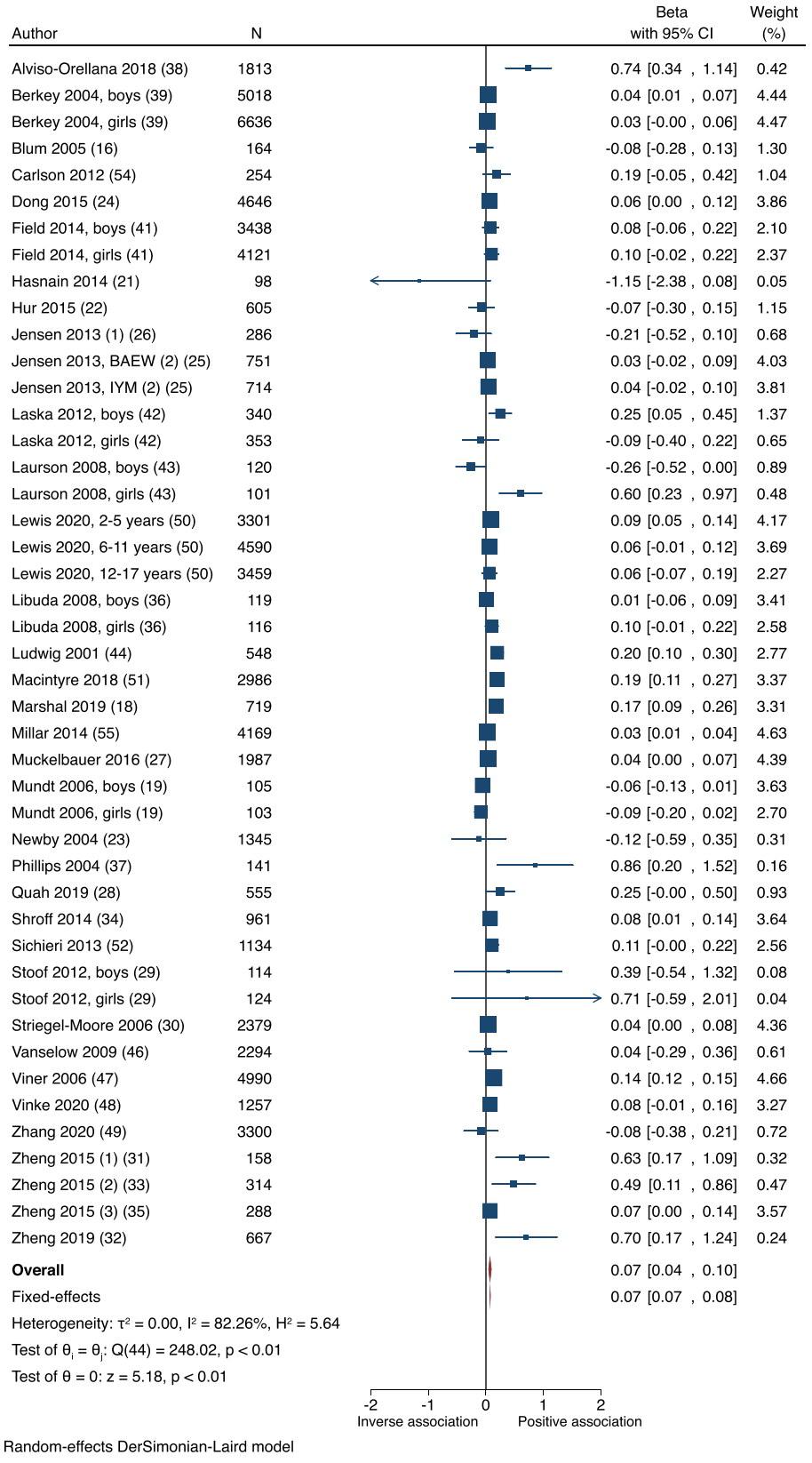

FIGURE 2. Pooled effect estimates for prospective cohort studies in children assessing the change in body mass index per 1 serving/d increase in sugar-sweetened beverage. The overall (random) effect estimate is represented by the diamond. Data are presented as β-coefficients with 95% confidence intervals (CIs), using the random- and fixed-effects models. 1 serving = 12 oz (355 mL).

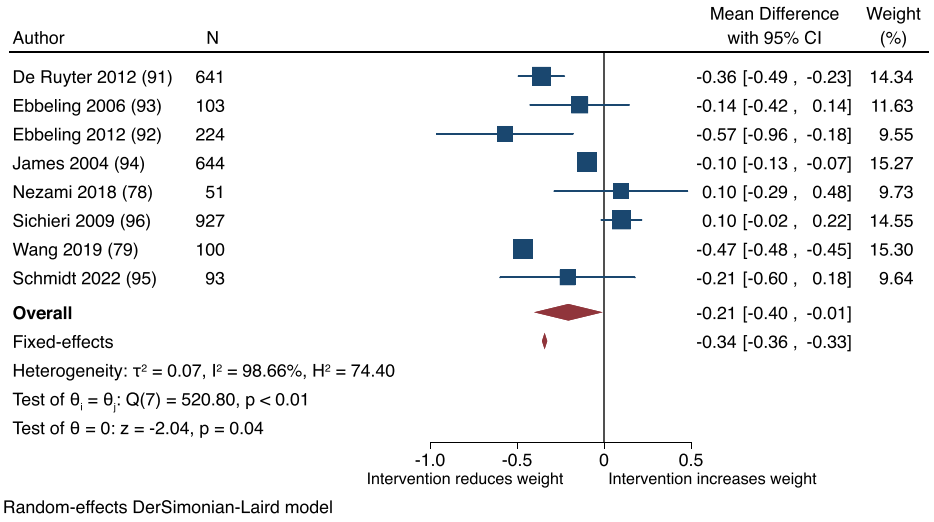

FIGURE 3. Pooled effect estimates for randomized controlled trials in children assessing the effect of the subtraction of sugar-sweetened beverage intake on body mass index change between the intervention and control groups. The overall (random) effect estimate is represented by the diamond. Data are presented as mean differences with 95% confidence intervals (CIs), using the random-and fixed-effects models.

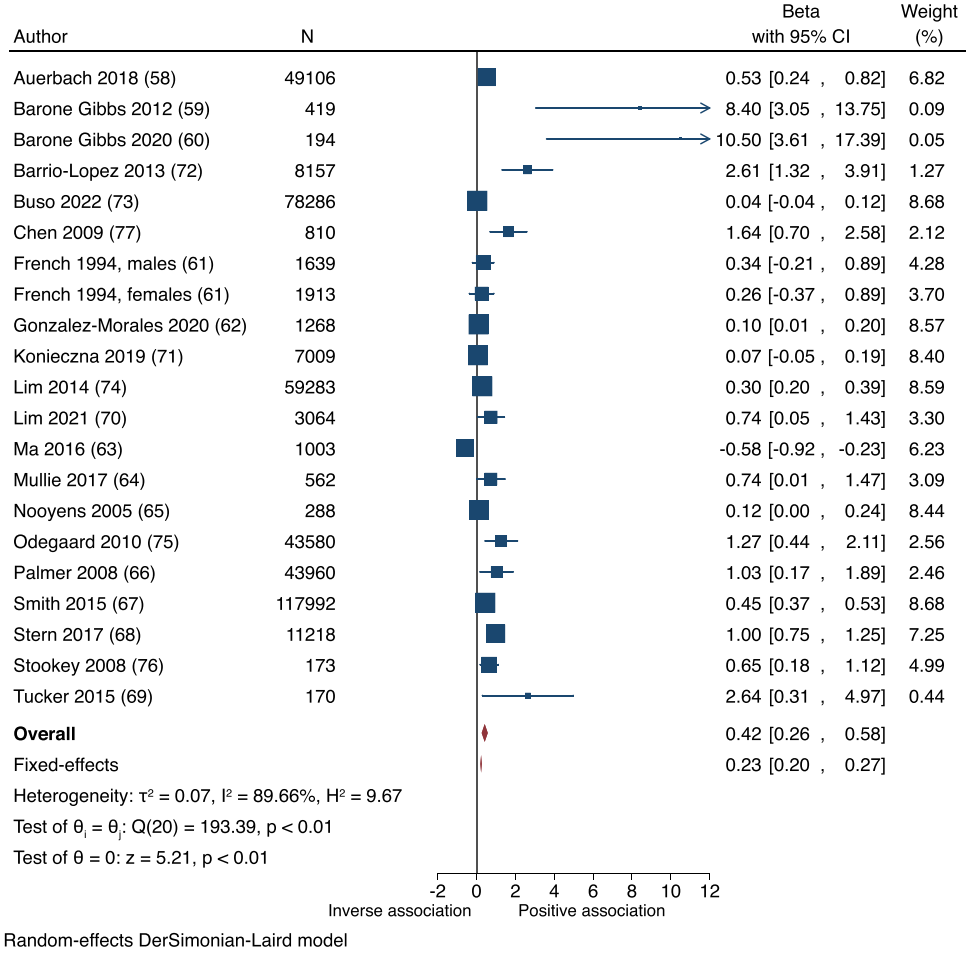

FIGURE 4. Pooled effect estimates for prospective cohort studies in adults assessing the change in body weight per 1 serving/d increase in sugar-sweetened beverage. The overall (random) effect estimate is represented by the diamond. Data are presented as β-coefficients with 95% confidence intervals (CIs), using the random-and fixed-effects models. 1 serving = 12 oz (355 mL).

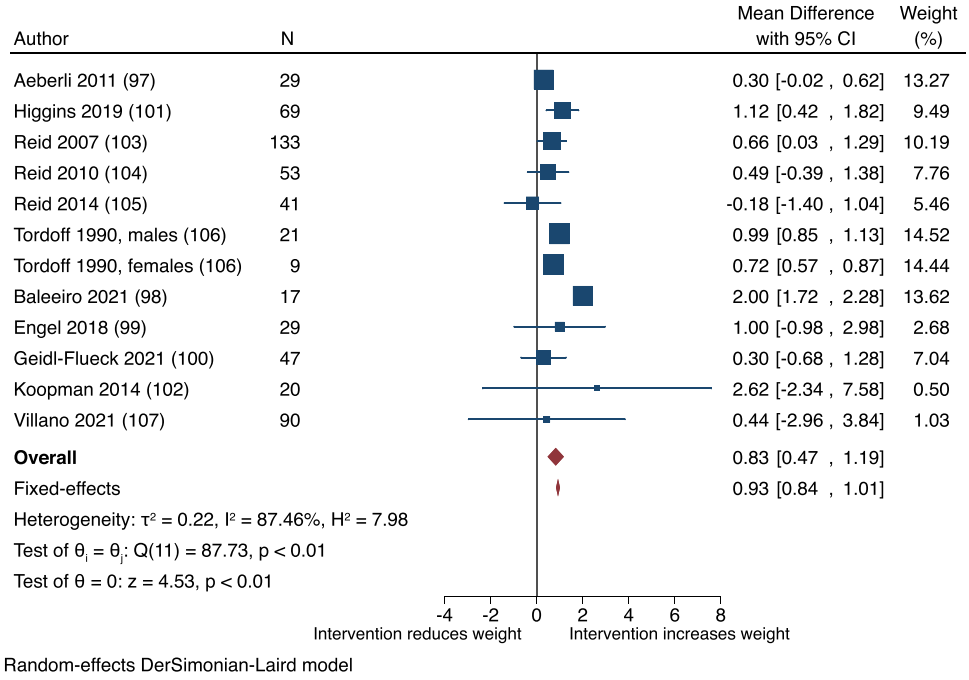

FIGURE 5. Pooled effect estimates for randomized controlled trials in adults assessing the effect of the addition of sugar-sweetened beverage intake on body weight change between the intervention and control groups. The overall (random) effect estimate is represented by the diamond. Data are presented as mean differences with 95% confidence intervals (CIs), using the random- and fixed-effects models.

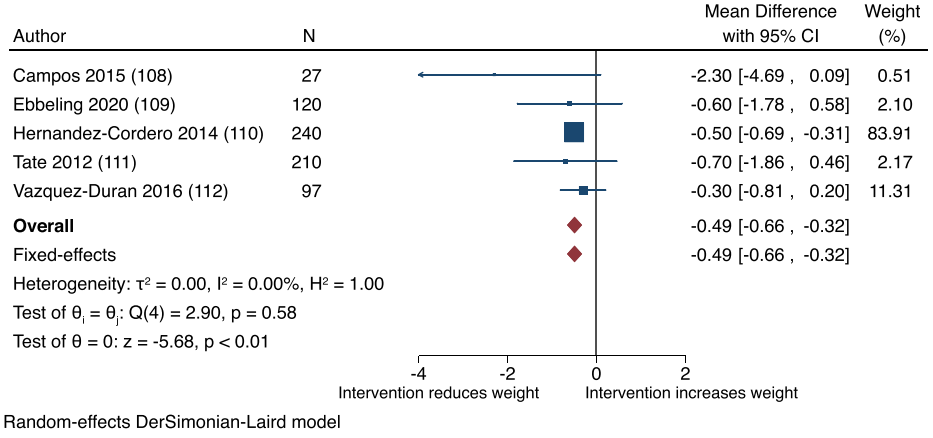

FIGURE 6. Pooled effect estimates for randomized controlled trials in adults assessing the effect of the subtraction of sugar-sweetened beverage intake on body weight change between the intervention and control groups. The overall (random) effect estimate is represented by the diamond. Data are presented as mean differences with 95% confidence intervals (CIs), using the random- and fixed-effects models.

Sensitivity and subgroup analyses

Sensitivity analysis by systematically removing individual studies and recalculating the overall effect is presented in Supplemental Figures 9–13. Removal of the studies by de Ruyter et al. [89], Ebbeling et al. [90], James et al. [92], and Schmidt et al. [93] changed the significance of the effect for RCTs in children (see Supplemental Figure 11). Sensitivity analysis did not change the heterogeneity, significance, or direction of the pooled effects in any other of our meta-analyses.

Subgroup analyses in prospective cohort studies in children indicated significant subgroup differences for adjusting for diet quality ($P < 0.05$), where adjusted estimates showed a stronger association (0.30 kg/m^2; 95% CI: −0.01 kg/m^2, 0.61 kg/m^2) than unadjusted estimates (0.07 kg/m^2; 95% CI: 0.04 kg/m^2, 0.09 kg/m^2) (see Supplemental Figure 14). Subgroup analyses in prospective cohort studies in adults revealed significant subgroup differences for age ($P < 0.01$), duration ($P = 0.01$), funding ($P = 0.01$), and outcome assessment method ($P < 0.04$) (see Supplemental Figure 15). Individuals aged <45 y showed a stronger association (0.62 kg; 95% CI: 0.41 kg, 0.84 kg) than those individuals aged ≥45 y (0.20 kg; 95% CI: 0.02 kg, 0.37 kg). Studies with a shorter duration (<48 mo) showed a stronger association (0.72 kg; 95% CI: 0.35 kg, 1.08 kg) than studies with a longer duration (≥48 mo) (0.27 kg; 95% CI: 0.11 kg, 0.44 kg). Studies where funding was not reported showed a stronger association (5.00 kg; 95% CI: −0.55

kg, 10.55 kg) than studies funded by agency (0.37 kg; 95% CI: 0.21 kg, 0.53 kg) or agency–industry (0.74 kg; 95% CI: 0.33 kg, 1.15 kg). Studies where body weight was self-reported showed a stronger association (0.72 kg; 95% CI: 0.45 kg, 0.99 kg) than studies where body weight was measured by investigators (0.24 kg; 95% CI: 0.06 kg, 0.40 kg). Subgroup analysis of addition trials in adults found a significant subgroup difference by location ($P < 0.01$) (see Supplemental Figure 16). Studies conducted in South America showed greatest effects (2.00 kg; 95% CI: 1.72 kg, 2.28 kg), followed by those in North America (0.88 kg; 95% CI: 0.65 kg, 1.12 kg) and Europe (0.37 kg; 95% CI: 0.12 kg, 0.62 kg). Subgroup analyses by risk of bias in addition trials in adults found no significant effects (see Supplemental Figure 17). Subgroup analyses in RCTs in children and subtraction trials in adults were not shown as there were <10 trial comparisons available for analysis.

Dose–response

Linear and nonlinear dose–response analyses for cohort studies in children and adults are presented in Supplemental Figures 18–21. In children, a significant linear dose–response association was found for BMI gain (MD for serving/d: 0.18 kg/m^2; 95% CI: 0.08 kg/m^2, 0.29 kg/m^2; $P < 0.01$), with no evidence of departure from linearity ($P = 0.96$). A significant linear dose–response association was found in adults for body weight gain (MD for serving/d: 0.68 kg; 95% CI: 0.14 kg, 1.22 kg; $P = 0.01$), with no evidence of departure from linearity ($P = 0.30$). Dose–response analysis of addition trials in adults is presented in Supplemental Figure 22. There was evidence of a dose–response relationship per serving/day increase in SSB intake and body weight (MD: 0.55 kg; 95% CI: 0.31 kg, 0.80 kg; $P < 0.01$), with no evidence of departure from linearity ($P = 0.12$). Dose–response analysis was not conducted for subtraction trials in children and adults because of the insufficient number of studies (<6 trial comparisons).

Publication bias

Visual inspection of contour-enhanced funnel plots for prospective cohort studies in children and adults showed evidence of asymmetry (see Supplemental Figures 23 and 24). This is supported by Egger's and Begg's tests for cohort studies in children ($P = 0.02$ and $P = 0.01$, respectively) and adults ($P < 0.01$ and $P = 0.02$, respectively). The trim-and-fill method imputed 3 studies for prospective cohort studies in children and 7 studies for adults; however, it did not alter the magnitude or significance of the effect, suggesting that there was no meaningful influence of publication bias on the results (see Supplemental Figures 25 and 26). Visual inspection of contour-enhanced funnel plots for addition trials in adults showed no evidence of asymmetry, and this was supported by Egger's and Begg's tests ($P = 0.63$ and $P = 0.95$, respectively) (see Supplemental Figure 27). Publication bias assessment for trials in children and subtraction trials in adults were not conducted because of the insufficient number of studies (<10 trial comparisons).

GRADE assessment

The GRADE assessment for prospective cohort studies is presented in Supplemental Table 9. The association between SSB consumption and BMI and body weight was graded as "low" for children and adults. Downgrades were made for serious inconsistency, and upgrades were made because of positive linear dose–response associations between SSB consumption and BMI and body weight. The GRADE assessment for RCTs is presented in Supplemental Table 10. The effect of SSB subtraction on BMI in children was graded as "low" because of downgrades for serious inconsistency and serious imprecision. The effect of SSBs on body weight in addition and subtraction trials in adults was graded as "moderate" for both. Downgrades were made for serious inconsistency and serious imprecision for addition trials in adults and for serious imprecision for subtraction trials. An upgrade was made because of a positive linear dose–response association between SSB consumption and body weight in adults. NutriGrade assessments for cohort studies and RCTs are presented in Supplemental Tables 11 and 12. All outcomes were graded as "moderate" quality.

Discussion

This systematic review and meta-analysis of 48 studies in children (40 prospective cohort studies and 8 RCTs) and 37 studies in adults (21 prospective cohort studies and 6 RCTs) demonstrates a positive relation between SSB consumption and body weight outcomes in children and adults. Pooled analysis of prospective cohort studies indicates that 1-serving/d greater intake of SSB is associated with a 0.07-kg/m^2 (95% CI: 0.04 kg/m^2, 0.10 kg/m^2; $P < 0.01$) higher BMI in children and adolescents and 0.42-kg (95% CI: 0.26 kg, 0.58 kg; $P < 0.01$) higher body weight in adults. This is supported by RCTs where a reduction in SSB consumption decreased BMI in children and adolescents (MD: −0.21 kg/m^2; 95% CI: −0.40 kg/m^2, −0.01 kg/m^2; $P = 0.04$) and body weight in adults (MD: −0.49 kg; 95% CI: −0.66 kg, −0.32 kg; $P < 0.01$) compared with noncaloric beverage consumption. Conversely, the addition of SSBs significantly increased body weight in adults (MD: 0.83 kg; 95% CI: 0.47 kg, 1.19 kg; $P < 0.01$).

Findings in the context of existing literature

Our findings are in agreement with previously published systematic reviews and meta-analyses of prospective cohort studies and RCTs, showing an overall positive association between SSBs and higher weight and higher weight gain in children and adults [11, 111, 112]; however, 2 reviews reported no relationship of SSBs with body weight [113, 114]. One of these reviews used estimates that were adjusted for total energy, which may have underestimated the true effect [114], and the other review showed a significant increase in body weight with SSB intake but only a marginally significant benefit on weight loss [113]. Findings from our updated review align with the previous meta-analysis conducted in 2013 [11], with the inclusion of 39 additional cohorts—including a number of large-scale studies from around the world, i.e., from the Women's Health Initiative cohort [57], Mexican Teachers Cohort [67], and UK Millennium Cohort Study [53]—and 16 RCTs. Our findings of statistically significant weight gain in children and adults using large prospective cohorts with long follow-up durations substantiate long-term weight gain with SSB consumption.

Evidence from trials where SSBs are added to the diet as excess calories is limited to adults. Our finding, a 0.83-kg (95% CI: 0.47 kg, 1.19 kg; $P < 0.01$) increase in body weight, is in line with a previous meta-analysis showing a 0.85-kg (95% CI: 0.50 kg, 1.20 kg) increase in body weight with the addition of SSBs to the diet [11]. Similarly, a World Health Organization commissioned meta-analysis found that increasing free sugars, mainly as SSBs, led to a 0.75-kg (95% CI: 0.30 kg, 1.19 kg; $P < 0.01$) increase in body weight [112]. Importantly, our meta-analysis additionally assesses the evidence of hypocaloric trials, or the subtraction of SSBs from the diet. Fewer trials have been conducted evaluating weight loss, likely as achieving and maintaining weight loss can be difficult. In children and adults, we found a

significant decrease in BMI and body weight with SSB reduction. Interestingly, subgroup analysis found no difference between educational messaging and direct interventions, such as replacement with noncaloric beverages (data not shown). This suggests that cost-effective educational programs to reduce SSB consumption can be a viable weight management strategy. In addition, we found no differences in weight change in adults and children between individuals who had normal weight or were overweight at baseline, suggesting that reducing intake of SSBs is a beneficial weight management strategy across different age and weight status groups.

Notably, our updated meta-analysis includes a dose–response analysis, in which we observed a positive linear association between greater SSB consumption and greater body weight and weight gain. This was consistent for cohort studies in both children and adults and RCTs in adults. Collectively, these findings provide strong support for the role of SSBs in higher weight and weight gain and are consistent with other recent meta-analyses on SSBs and the risk of cardiometabolic disease [115–117].

SSBs are thought to contribute to higher weight and weight gain through several mechanisms. First, SSBs can lead to weight gain through low satiety and incomplete compensation of energy at subsequent meals following ingestion of liquid calories [118]. The overconsumption of highly palatable sugary beverages can result in excess energy and, thus, energy imbalance and weight gain if energy intake is not adjusted at the following meals. Studies have also suggested greater weight gain after isocaloric consumption of beverages than after that of solid foods [119]. This suggests that drinking liquid sugars contributes to greater energy intake than solid sugar calories. SSBs may also contribute to weight gain due to the metabolic response from fructose when consumed in caloric surplus. These beverages are often sweetened with HFCS or sucrose, both of which are fructose-containing sugars. A hypothesis linking fructose to weight gain and obesity suggests that fructose does not stimulate leptin, the satiety hormone, nor does it suppress ghrelin, the hunger hormone [120, 121]. The impaired satiety signaling due to fructose may promote positive energy balance. When consumed in excess, fructose has been shown to increase cardiometabolic risk factors, including obesity, hypertension, insulin resistance, and dyslipidemia [122]. However, the harmful metabolic effects of fructose on weight gain seem to be largely driven by conditions of excess energy [123]. Fructose, unlike glucose, has a unique endocrine signature where it is preferentially metabolized in the liver. When consumed in excess, fructose can lead to hepatic de novo lipogenesis, which can lead to the production of very-low-density lipoproteins and postprandial triglycerides [124]. The potential deleterious effects of excess fructose may be further exacerbated with low physical activity, which is often clustered with unhealthy lifestyle habits including consumption of SSBs [125].

Strengths and limitations

Our meta-analysis and systematic review has several strengths. Through a vigorous search and selection method, we were able to provide a comprehensive analysis of both cohort studies and RCTs in children and adults with large sample sizes and study durations. An analysis of both study designs is critical to examine the totality of evidence, as cohort studies examine long-term weight gain and trials can examine causality. For cohorts, we included a separate 1-y change analysis in studies that reported the change in weight in relation to the change in SSB intake. These studies include repeated assessments of both diet and weight and have features of a quasiexperimental design. This strengthens the generalizability of the findings to real-world settings as participants are able to freely modify their diet and lifestyle. We also included a separate analysis for studies that included adjustments for energy intake as well as a sensitivity analysis for cohort studies and trials in children using BMI z-score as the outcome rather than BMI. To our knowledge, this study is also the first to conduct a dose–response analysis of SSBs and body weight using data from prospective cohort studies. Finally, we assessed the certainty of evidence using both GRADE and NutriGrade. As the GRADE system initially regards cohort studies as "low" certainty of evidence, NutriGrade may be more appropriate for nutritional epidemiology studies and GRADE for RCTs.

Our study is not without limitations. Despite the majority of the included prospective cohort studies being of high quality, as indicated by the NOS, the inherent nature of the observational design makes these studies prone to residual confounding. In addition, self-reported dietary assessment tools used in these studies may lead to inaccuracies and misreporting errors. RCTs are not without limitations either, as they are limited by short study durations and incomplete adherence, which may underestimate true effects. Substantial heterogeneity was detected in our meta-analyses and could not be explained by subgroup or sensitivity analysis. Furthermore, we necessarily performed a number of data transformations with inherent assumptions that may limit the validity of our estimates. In studies where the serving size of the beverage was not reported, we made the assumption of 12 oz, which is consistent with typical cans or glasses of SSB; however, this measurement error may have led to an overestimation or underestimation of actual serving sizes. Although our analyses only assess body weight and BMI, these measures can be directly translated to public health recommendations, and BMI has been shown to be well correlated with adiposity as measured by more direct methods [126]. Additionally, reporting effect estimates in children as BMI rather than BMI z-score needs to be interpreted with caution because of the age range in the sample. However, our sensitivity analysis using BMI z-score showed consistent results.

In conclusion, our updated systematic review and meta-analysis provides consistent evidence that SSB consumption promotes weight gain in children and adults. As overweight and obesity are significant predictors of cardiometabolic risk and poor health, our study highlights the importance of greater efforts to reduce consumption of SSBs in children and adults.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ajcnut.2022.11.008.

Data in this manuscript will be made available upon request.

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