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RESEARCH

# Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies


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## Abstract

**Objective** To summarise evidence on the association between intake of dietary sugars and body weight in adults and children.

**Design** Systematic review and meta-analysis of randomised controlled trials and prospective cohort studies.

**Data sources** OVID Medline, Embase, PubMed, Cumulative Index to Nursing and Allied Health Literature, Scopus, and Web of Science (up to December 2011).

**Review methods** Eligible studies reported the intake of total sugars, intake of a component of total sugars, or intake of sugar containing foods or beverages; and at least one measure of body fatness. Minimum duration was two weeks for trials and one year for cohort studies. Trials of weight loss or confounded by additional medical or lifestyle interventions were excluded. Study selection, assessment, validity, data extraction, and analysis were undertaken as specified by the Cochrane Collaboration and the GRADE working group. For trials, we pooled data for weight change using inverse variance models with random effects. We pooled cohort study data where possible to estimate effect sizes, expressed as odds ratios for risk of obesity or β coefficients for change in adiposity per unit of intake.

**Results** 30 of 7895 trials and 38 of 9445 cohort studies were eligible. In trials of adults with ad libitum diets (that is, with no strict control of food intake), reduced intake of dietary sugars was associated with a decrease in body weight (0.80 kg, 95% confidence interval 0.39 to 1.21; P<0.001); increased sugars intake was associated with a comparable weight increase (0.75 kg, 0.30 to 1.19; P=0.001). Isoenergetic exchange of dietary sugars with other carbohydrates showed no change in body weight (0.04 kg, –0.04 to 0.13). Trials in children, which involved recommendations to reduce intake of sugar sweetened foods and beverages, had low participant compliance to dietary advice; these trials showed no overall change in body weight. However, in relation to intakes of sugar sweetened beverages after one year follow-up in prospective studies, the odds ratio for being overweight or obese increased was 1.55 (1.32 to 1.82) among groups with the highest intake compared with those with the lowest intake. Despite significant heterogeneity in one meta-analysis and potential bias in some trials, sensitivity analyses showed that the trends were consistent and associations remained after these studies were excluded.

**Conclusions** Among free living people involving ad libitum diets, intake of free sugars or sugar sweetened beverages is a determinant of body weight. The change in body fatness that occurs with modifying intakes seems to be mediated via changes in energy intakes, since isoenergetic exchange of sugars with other carbohydrates was not associated with weight change.

## Introduction

Sugar has been a component of human diets since ancient times, with earliest reports of consumption coming from China and India, and much later from Europe after the Crusades in the 11th century.[1] The suggestion that sugar might have adverse health effects has been a recurring theme for decades, with claims that high intake may be associated with an increased risk of conditions as diverse as dental caries, obesity, cardiovascular disease, diabetes, gout, fatty liver disease, some cancers, and hyperactivity.[2-6] However, inadequate study design, differences in assessing dietary intake, inconsistent findings, and varying

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**Web appendix 1:** Search terms
**Web appendix 2:** GRADE summaries
**Web appendix 3:** Randomised trials excluded from analysis
**Web appendix 4:** Cohort studies excluded from analysis
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**Web table 2:** Pooled estimates of associations between sugars intakes and measures of adiposity from prospective cohort studies in children

definitions of "sugars" have precluded definitive conclusions regarding these associations.

The most consistent association has been between a high intake of sugar sweetened beverages and the development of obesity,[7-12] but not all published meta-analyses have reported a statistically significant link.[7 11] The expert consultations organised by the World Health Organization and the Food and Agriculture Organization of the United Nations and the scientific updates undertaken by WHO[13-15] have adopted a classification of carbohydrates and clarified definitions of various groups of sugars including the category of "free sugars" (table 1⇓). This classification enables a more standardised approach to examining potential adverse health effects.

To update the recommendations through the guideline's development process that was launched in January 2009, WHO commissioned a systematic literature review to answer a series of questions[16] relating to the effects of sugars on excess adiposity. These questions asked whether reducing or increasing intake of dietary sugars influences measures of body fatness in adults and children, and whether the existing evidence provides support for the recommendation to reduce intake of free sugars to less than 10% total energy (box).[15] Body fatness was selected as an outcome in view of the extent to which comorbidities of obesity contribute to the global burden of non-communicable disease.

Since the answers to the questions posed (box) were designed to inform population based dietary guidelines rather than advise individual patients, it was deemed appropriate to include cohort studies and randomised controlled trials of free living people consuming ad libitum diets (that is, with no strict control of food intake). The interventions mainly involved advice to increase or decrease intake of sugars, or of sugar containing foods or beverages, without emphasising the need to achieve weight loss.

We also examined randomised controlled trials comparing higher and lower intakes of sugars, but where energy intake was strictly controlled. Trials specifically designed to achieve weight loss were excluded. We acknowledged that the studies identified by this approach would inevitably be heterogeneous, that it would be difficult to disentangle the effects of a range of dietary changes that might occur after altering the intake of sugars, and that it might be difficult to identify a dose response. However, the findings from such an approach were expected to provide an indication of what might be achieved by population changes in intake of dietary sugars.

## Methods

In accordance with the WHO guideline's development process,[17] systematic reviews and meta-analyses were conducted according to the methods of the Cochrane Collaboration.[18] We prepared tables summarising quality assessment, effect size, and importance of findings, from which recommendations may be derived, in the format required by the Grading of Recommendations Assessment, Development and Evaluation (GRADE) working group. Ethical approval was not required for this research.

### Search strategy

Two separate electronic searches were conducted to identify randomised trials and prospective cohort studies relating intake of dietary sugars to measures or changes of body fatness (web appendix 1). OVID Medline, Embase, PubMed, Cumulative Index to Nursing and Allied Health Literature, Scopus, and Web of Science electronic databases were searched for clinical trials and cohort studies, published up to December 2011, which met the inclusion criteria. In OVID Medline, we used the highly sensitive Cochrane search strategy to limit the first search to clinical trials, meta-analyses, and randomised controlled trials. We hand searched meta-analyses and reviews to identify studies that might have been missed.

### Study selection

Two reviewers assessed titles and abstracts of all identified English language studies. Discrepancies in opinion as to whether studies should be selected for full review were resolved by discussion. A similar approach was used to determine which of these studies should be included in the formal analysis. Animal studies, cross sectional studies, and case-control studies were excluded. Studies were required to report intake of total sugars, intake of a component of total sugars (expressed in absolute amounts or as a percentage of total energy), or intake of sugar containing foods or beverages, assessed by continuous or categorical variables; and at least one measure of body fatness.

Participants were adults and children free from acute illness, but those with diabetes or other non-communicable diseases in whom conditions were regarded as stable could be included. Randomised trials were required to be of at least two weeks' duration, and prospective cohort studies were required to be of at least one year's duration. We included trials comparing diets differing in sugars intakes and in which the effect of sugars could be separated from the effects of other lifestyle or medical interventions.

Two groups of trials were identified. One group included studies in which participants in the intervention arm were advised to decrease or increase sugars, or foods and drinks containing sugars. Although such advice was generally accompanied by the recommendation to increase or decrease other forms of carbohydrate, there was no strict attempt at weight control. These trials are referred to as ad libitum studies. The other group of trials attempted to achieve isoenergetic replacement of sugars with other forms of carbohydrate. Interventions designed to achieve weight loss were excluded because the ultimate aim of the review was to facilitate the development of population based recommendations rather than nutritional recommendations for the management of obesity.

### Data extraction and quality assessment

Data extraction and validity assessment were carried out independently by two reviewers, and any discrepancies resolved by discussion. For both randomised trials and cohort studies, outcomes, data relating to participants, exposure or interventions, potential effect modifiers, and study quality were extracted by use of piloted data extraction forms. In the cohort studies, we aimed to extract the least and most adjusted relative risk, odds ratio, or mean difference when comparing the most exposed group of participants with the least exposed group, or a $\beta$ coefficient for the continuous effect of a one unit change in sugars intake. We extracted these statistics separately for sugars exposures reported as baseline values or as values for change over time.

Cochrane criteria[18] were used to examine validity of each randomised trial, including sequence generation, allocation concealment, blinding of participants, personnel and outcome assessors, incomplete outcome data, and selective outcome reporting. Additional review specific criteria included similarity, or not, of type and intensity of intervention in both arms, and whether the studies were funded by industries with potentially

**Questions posed by the WHO Nutrition Guidance Expert Advisory Group-Subgroup on Diet and Health, to develop recommendations regarding sugars intakes**

What is the effect of a reduction in free sugars intakes in adults?

What is the effect of an increase in free sugars intakes in adults?

What is the effect of a reduction in free sugars intakes in children?

What is the effect of an increase in free sugars intakes in children?

(Where "free sugars" are defined as all monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer; plus sugars naturally present in honey, syrups, and fruit juices.)

vested interests. We examined the effect of bias on the pooled effect estimates by excluding studies that had a high risk of bias for two or more validity criteria in sensitivity analyses.

## Statistical analysis

Studies were grouped to answer the major questions that had been posed (box). We considered data for adults and children separately. Studies of isoenergetic exchange of sugars with other carbohydrates were examined to help explain possible mechanisms through which sugars might exert their effects.

### *Randomised trials*

The effects of decreasing or increasing dietary sugars in adults were examined principally by meta-analysing the randomised trials in which participants were required to consume different amounts of sugar (sucrose) or other sugars (which would now be classified as "free sugars"). Terminology varied among trials. The term "free sugars" refers to all monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer, plus sugars naturally present in honey, syrups, and fruit juices (table 1).[14] The term "added sugar" is sometimes used interchangeably with "free sugar" but is considered to include sugars and syrups added to foods during processing, food preparation, or at the table—but does not include honey, syrups, or fruit juice.[19] "Sugar" is generally assumed to be purified sucrose.[14]

Data for each group of studies were pooled using Review Manager 5.1 software.[20] In trials involving adult participants, we used generic inverse variance of analysis for mean differences in body weight between intervention and control groups to compare the parallel and crossover experimental designs reporting change in body weight. In the studies involving children and adolescents, we used standardised mean differences because studies reported differences in either body mass index (BMI) or standardised BMI units.

Heterogeneity was assessed with the $I^2$ test and Q statistics. We considered an $I^2$ value greater than 50% and $P<0.05$ as indicative of heterogeneity.[18] We used random effects models because significant heterogeneity was associated with trial design and duration in some analyses.

Estimates for the standard error of the difference in means for treatment groups in crossover studies were derived from reported P values when the standard error of the mean difference was not reported.[18] If P values for the differences were reported simply as non-significant, then P=0.2 was assumed.[18]

We did sensitivity analyses to explore the differences between studies in the short term (<eight weeks) and longer term (>eight weeks). We also tested the effects of removing those studies that achieved a difference in sugars intakes of less than 5% of total energy intake between intervention and control groups. Metaregression (using Stata/IC 11.2 software for Mac (StataCorp)) was used to test for a dose-response effect of sugars on weight change, and for associations between weight change and study duration, study design (that is, crossover or parallel), and whether sugars intake changed in the intervention arm.

Publication bias among the randomised controlled trials of adults was examined by visual inspection of a funnel plot and Egger's test for bias.[21] Publication bias is suspected when the funnel plot is asymmetrical. We combined the 15 ad libitum studies for this analysis because it is generally accepted that asymmetry cannot readily be assessed with 10 or fewer studies.[18] Sensitivity analyses examined the influence of small study effects, by comparing the estimates derived from random and fixed effects models[22] and by using the Duval and Tweedie[23] "trim and fill" method in Stata 12 (Metatrim). There were insufficient studies in children to conduct a meaningful examination of publication bias.

### *Prospective cohort studies*

Cohort studies in adults provided limited additional information. Data from cohort studies in children were necessary to determine the effect of increasing sugars intake on adiposity, owing to a lack of suitable randomised trials. We grouped individual studies for meta-analysis on the basis of the methods used for reporting adiposity outcomes and sugars exposure variables.

We used four main methods of reporting outcomes:

- β coefficients for the continuous association between sugars exposure at baseline and adiposity outcome.
- Odds ratios for the risk of overweight or obesity comparing participants who had the highest intakes of sugars with those who had the lowest intakes of sugars (groups or frequency of servings).
- Mean differences in change in measures of adiposity over time between participants with the highest intakes of sugars and those with the lowest intakes (groups or frequency of servings).
- β coefficients for the continuous association between increases in sugars exposure over time and adiposity outcome.

Sugars exposures included sugar sweetened beverages, fruit juice, sweets (including jams, syrups, cakes, and desserts), sucrose, or total sugars. Exposures were reported as servings per time period and were converted to servings per day, volume of beverage consumed per day, percentage of energy intake, frequency of consumption, or grams per day. Where possible, we scaled exposures to comparable units to allow data to be pooled. We assumed that one serving of sugar sweetened beverage was equivalent to 240 mL or 8 fluid ounces, and contained 26 g of sucrose.[24] This portion equated to about 5% of daily total energy intake in adults.

Measures of body fatness included weight change, change in BMI or BMI z score, waist circumference, body fat (%), fat mass, and trunk fat (%). If studies reported more than one measure of sugars intake, we derived an average effect size. We ranked adiposity outcomes in terms of importance for pooling, from highest to lowest: BMI z score, BMI, body weight, waist

circumference, percentage body fat, fat mass, and percentage trunk fat. If studies reported outcomes for more than one measure of adiposity, we used the highest ranked adiposity outcome. We generated pooled estimates for the various subgroups using metan commands with random effects in Stata. Two sided P<0.05 was considered significant for all analyses.

### GRADE assessment

GRADE assessment[25] was carried out to assess the totality of the evidence by the authors and then refined by the WHO Nutrition Guidance Expert Advisory Group (NUGAG) Subgroup on Diet and Health (www.who.int/nutrition/topics/advisory_group/en/index.html) to fulfil the required process for developing WHO guidelines.[17] GRADE assessment took into account study design limitations, consistency of results across the available studies, precision of the results, directness, and likelihood of publication bias when assessing the quality of the evidence from the randomised trials.[17 25] Further criteria were considered for the cohort studies. These criteria included magnitude of the effect, evidence of a dose-response gradient, and the direction of plausible biases. The quality of the evidence was categorised as high, moderate, low, or very low. Web appendix 2 shows the relevant GRADE tables.

## Results

Figures 1⇓ and 2⇓ show the process by which the included studies were identified. We identified 7895 potential randomised trials from the electronic search and a further 10 studies through hand searches of relevant review articles and on recommendation from NUGAG panel members. Removing duplicates left 6634 articles, of which 6557 were assessed to be irrelevant. Abstracts and full text articles for the remaining 77 studies were judged as requiring full review and were reviewed by three independent reviewers. Of these remaining studies, 19 met the inclusion criteria for ad libitum studies [26-47] and 11 were identified for the comparative analysis of isoenergetic studies.[48-58] For cohort studies, we identified 9445 potential studies from the electronic search and an additional 10 studies through hand searches of relevant review articles. Of 69 studies selected for full review, 38 were considered to meet the inclusion criteria.[59-74] The 47 excluded randomised trials and 31 excluded cohort studies are described in web appendices 3 and 4.

### Assessment of study quality

Risk of bias varied among the randomised trials (web figs 1 and 2, web appendix 5). Failure to conceal treatment allocation (almost impossible to achieve in dietary trials involving free living participants) was the major potential source of bias (performance bias). In many trials, it was unclear as to whether outcome measures had been assessed by observers unaware of treatment allocation (detection bias) and whether there had been selection bias. Three trials, in which there was evidence of differences between dropouts and completers, reported data only for those who completed the intervention.[28 34 39]

Our analysis included 38 prospective studies lasting at least 12 months, and in which data relating to an association between sugars and a measure of adiposity could be extracted; none was excluded on the basis of study quality. Of these 38 studies, 15 used self reported estimates of adiposity outcomes[59 64-68 70 71 73-80]; seven collected exposure data from questionnaires where the validity for assessing sugars intake was not stated or not assessed[60 61 67 79 81 82]; 19 involved convenience sampling[59 61 62 67 71 73 78 83-93]; and 18 provided estimates that were adjusted for total energy intake.[59 60 64 66 69 72 75 76 86 88 90-97] There was a lack of consistency in the covariates used to adjust analyses and a wide range of methods of assessing sugars exposures and adiposity outcomes, which made pooling studies difficult.

### Effect of reducing dietary sugars on measures of body fatness in adults

Table 2⇓ describes the five studies identified for this analysis,[28 30 31 33 41 49] and figure 3⇓ shows the quantitative meta-analysis (forest plot). Reduction in dietary sugars intake was associated with significantly reduced weight (−0.80 kg (95% confidence interval −1.21 to −0.39); P<0.001) at the end of the intervention period by comparison with no reduction or an increase in sugars intake. The trials all involved a reduction in intake of sugars (classified as free sugars) in the intervention arm compared with the control arm.[28 31 33 39 41] Study durations ranged from 10 weeks to eight months. In four studies, participants were advised to limit sugar containing foods,[31 33 39 41] and in one study, participants were asked to substitute usual sugar rich foods with low sugar alternatives.[28] Three of the five trials reported data for completers only.[28 39 41] However, only two of these studies considered this to be a potential source of bias.[28 41] Exclusion of these two studies from the meta-analysis slightly attenuated the effect, although the effect estimate remained significant (−0.81 kg, −1.41 to −0.21). After excluding three studies[28 39 41] that had a high risk of bias for two or more validity criteria, the effect estimate was no longer significant although the difference in weight was similar (−0.81 kg, −1.69 to 0.07).

Differences in sugar intakes between intervention and control groups ranged from less than 1%[33] to 14% of total energy intake.[39] Two studies achieved a difference in reported sugars intake of less than 5% of total energy intake at the end of the intervention.[28 33] Paineau and colleagues[33] reported a difference in sugars intake between groups of 2.2 g/day, and Gatenby and colleagues[28] reported a difference of about 3% of energy intake (15 g/day). Exclusion of these studies from the meta-analysis strengthened the overall effect of lowered sugar intakes on body weight change (−1.22 kg, 95% confidence interval −1.81 to −0.63). We saw no evidence of heterogeneity ($I^2$=17%, P=0.3), and the test for overall effect showing an association between sugar reduction and increased weight loss was highly significant.

### Effects of increasing dietary sugars on measures of body fatness in adults

Table 3⇓ describes the 10 studies identified for this analysis, and figure 4⇓ shows the quantitative meta-analysis (forest plot).[26 32 34 36-38 43-45 47] Because there was statistical evidence for significant heterogeneity among the studies ($I^2$=82%, P<0.001), we used a random effects model to derive the pooled estimates. Increased intake in dietary sugars was associated with significantly greater weight (0.75 kg (95% confidence interval 0.30 to 1.19); P=0.001) at the end of the intervention period by comparison with no increase in sugars intake. The studies involved an increase in dietary sugars; mostly sugar sweetened beverages, in the intervention arm of the randomised trial. Only two studies lasted longer than eight weeks.[34 36] Subgroup analysis for these two longer term studies resulted in a significantly greater effect size (2.73 kg, 1.68 to 3.78) than the pooled effect for the shorter term studies (0.52 kg, 0.14 to 0.89). The difference between these subgroups was highly significant (P<0.001).

One trial reported a higher rate of participant dropout in the high sugars group than in the low sugars group and presented

results for only participants who completed the whole study.[37] Exclusion of this study from the meta-analysis increased the overall effect size slightly (0.83 kg, 95% confidence interval 0.31 to 1.35). The association also remained significant after excluding from the meta-analysis five studies[26 32 34 37 43] that had a high risk of bias for two or more validity criteria (0.96 kg, 0.06 to 1.85).

## Isoenergetic exchanges of dietary sugars with other carbohydrates or other macronutrient sources

We identified 12 studies that involved isoenergetic exchange of dietary sugars with other macronutrients (table 4⇓).[48-58] Interventions ranged from two weeks to six months, and sugars were in the form of either sucrose or fructose used to sweeten foods or liquids. We saw no evidence of difference in weight change as a result of differences in sugars intakes when energy intakes were equivalent (0.04 kg (95% confidence interval −0.04 to 0.13); fig 5⇓).

## Findings of cohort studies

Table 5⇓ describes 16 cohort studies in adults that provided analyses of the relation between sugars exposures and measures of adiposity.[59-62 64-74 76] With a vote counting approach, 11 studies reported one or more significantly positive associations between a sugars exposure and a measure of adiposity,[59-62 64 65 68-71 73 74] and one study reported a significantly negative association.[73] Two studies reporting changes in intake of sugar sweetened beverages during follow-up showed a significantly greater increase in weight change among participants with the highest intake than in those with the lowest intake.[71 74] Web table 1 summarises pooled estimates for the relation between sugars intakes and various measures of adiposity from all other prospective studies in adults that met the inclusion criteria. Forest plots for these comparisons are provided in web figures 3-5 (web appendix 5).

## Effects of reducing dietary sugars on measures of body fatness in children

Table 6⇓ describes the five intervention trials identified for this analysis, and figure 6⇓ shows the forest plot.[27 29 33 40 46] Interventions generally included advice to reduce sugar sweetened beverages and other foods containing (free) sugars. We saw no association between such advice to reduce intake of dietary sugars and change in standardised BMI or BMI z score in children (0.09, 95% confidence interval −0.14 to 0.32). The studies included in this meta-analysis involved advice to reduce the intake of sugar sweetened beverages alone,[27 29 40] or together with a further reduction in other sugar rich foods and an increase in dietary fibre.[33 46] Poor compliance with the intervention advice was reported in three of the five studies,[29 33 46] and the effect of the intervention was a reduction of 51 mL/day in another study.[40] Significant heterogeneity was observed and a random effects model was used for the meta-analysis. Excluding the study by Davis and colleagues,[46] which had a high risk of bias for two or more validity criteria, did not alter the effect estimate.

## Effects of increasing dietary sugars on measures of body fatness in children

There were no randomised trials available in children, thus we used data from 21 cohort studies in children (reported in 22 articles) to assess the effect of increasing sugars intakes on body fatness (table 7⇓). Most studies related to intake of sugar sweetened beverages. A quantitative meta-analysis (fig 7⇓) was based on five cohort studies, with seven comparisons. These studies reported data for the odds of being overweight at follow-up in children consuming about one daily serving of sugar sweetened beverages at baseline compared with children consuming none or very little.[80 94-97] Comparison of the higher intakes with lower intakes suggested a significantly increased risk of being overweight associated with higher intakes (odds ratio 1.55, 95% confidence interval 1.32 to 1.82). We saw no evidence of heterogeneity, and all the studies reported a positive association. When assessing the 23 cohort studies in children using a "vote counting" approach, 15 reported a positive association between increased sugars intake and a measure of adiposity.[75 79-82 86 88 89 91 92 94-98] Fourteen of these 15 studies reported the sugars exposure as a sugar sweetened beverage. By contrast, only four studies reported a negative association,[87 90 93 98] of which two reported fruit juice as the sugars exposure.[90 98]

Web table 2 summarises pooled and unpooled estimates for the association between sugars intakes and measures of adiposity from all other prospective studies in children that met the inclusion criteria. Because of the wide variation in how the study effects were reported, it was not always possible to pool studies reporting similar outcomes, and there was no evidence of association between increased sugars and adiposity. Web figures 6 and 7 (web appendix 5) show forest plots.

## Sensitivity analyses

The overall meta-regression of randomised trials examining the effect of sugars on adiposity in adults showed no evidence of a dose-response association between sugar as a percentage of total energy intake and body weight (0.02 kg (95% confidence interval −0.03 to 0.08); P=0.392). The difference in weight changes associated with differing intakes of sugars was unrelated to study design (crossover or parallel design trials; 0.30 kg (−0.44 to 1.05); P=0.393), study duration (0.01 kg per week (−0.02 to 0.05); P=0.460), or whether sugars intakes were reduced or increased in the intervention arm relative to the control arm (0.12 kg (−0.73 to 0.96); P=0.817).

## Publication bias

The funnel plot of all 15 randomised ad libitum trials conducted in adults was asymmetrical and the Egger's test for bias was significant (P=0.001), which suggested possible publication bias (fig 8⇓). The pooled effect size for all 15 trials was 0.78 kg (95% confidence interval 0.43 to 1.12), based on a random effects model which accounted for significant heterogeneity ($I^2$=77%, P<0.001) seen between the relatively short term crossover trials with small variances and the longer term parallel trials with larger variances. Use of fixed effects models attenuated the overall effect (0.42 kg, 0.28 to 0.56), but it remained significant. Excluding the studies with the largest study variances[34 41] from the analysis had little effect (0.72, 0.37 to 1.06). Trim and fill analysis showed a somewhat attenuated but significant effect size (0.50, 0.18 to 0.21). Visual inspection of the funnel plot and the Egger's test for bias (P=0.248) did not suggest publication bias among the isoenergetic trials.

# Discussion

The meta-analyses based on controlled trials provide consistent evidence that increasing or decreasing intake of dietary sugars from current levels of intake is associated with corresponding changes in body weight in adults. Although some evidence of potential publication bias existed, this did not seem to have an

important effect on the findings. Results from cohort studies were generally comparable with the trial findings. The reviewed studies largely related to the manipulation or observation of intake of sugars which, using current terminology, would be described as "free sugars." Two six month trials, [99 100] published subsequent to the census date for this systematic review, involved different intakes of sugar sweetened beverages in adults. The trials also showed a trend towards increased body weight in participants with raised intake, but the difference between groups was not significant, perhaps owing to small number of participants.

Poor compliance with dietary advice could explain why the data from trials in children were equivocal. This was confirmed by two controlled trials published after our systematic review's census date.[101 102] De Ruyter and colleagues[101] showed a smaller increase in BMI z score after 18 months among trial completers who were provided with sugar free, artificially sweetened beverages, compared with participants who received equal quantities of sugar sweetened beverages. Ebbeling and colleagues[102] showed the potential of an intervention designed to decrease the consumption of sugar sweetened beverages in overweight and obese adolescents. BMI and body weight were significantly reduced after one year in the intervention group compared with the control group. However, after a further year's follow-up with no further intervention, the difference between the groups was no longer significant.

Cohort studies in children confirmed a link between intake of sugar sweetened beverages and the risk of becoming overweight, but showed no consistent associations between other measures of sugars intake and adiposity. Although comparison of groups with the highest versus lowest intakes in cohort studies was compatible with a recommendation to restrict intake to below 10% total energy, currently available data did not allow formal dose-response analysis.

## Strengths and limitation

An important strength of this in depth review of the literature lay in the overall quality and consistency of the data, especially those derived from adult populations. Although the trials were published over a long timeframe and used different experimental approaches, the results were consistent. Evidence was derived principally from randomised trials, but data from cohort studies that compared higher and lower groups of intake were also confirmatory. Criteria from both GRADE[25] and the World Cancer Research Fund[103] for judging strength of evidence of association specify randomised controlled trials as the highest level of evidence, but evidence from another study type is recognised as providing important confirmation.

We found less consistent findings from the trials conducted in children, which can be attributed to several factors. These trials tended to last longer than adult trials, and where compliance was assessed, it was clear that adherence to dietary advice (typically advice to reduce sugar sweetened beverages) was poor. For example, in a trial by Davis and colleagues,[46] children receiving nutrition education to improve carbohydrate quality achieved a reduction in added sugars intake of only 8 g/day, compared with control children. However, in children (as in adults), comparison of the highest intakes with the lowest intakes (usually of sugar sweetened beverages) suggested that those participants consuming the largest quantities had a higher body weight or other measure of adiposity.

The limitations of these findings are those inherent to the primary research on which they are based, notably inadequacy of dietary intake data, and variation in the nature and quality of the dietary intervention. Most cohort studies and some trials reported effects largely or solely related to the consumption of sugar sweetened beverages. Most trials involved different levels of intake of sugar (sucrose) and other monosaccharides and disaccharides in the control and intervention arms. These compounds have been described as "free sugars," as defined by WHO (all monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer, plus sugars naturally present in honey, syrups, and fruit juices).[14] We had originally intended to report separately on the effects of total sugars as well as the various subcategories of sugars, but presentation of data in the studies precluded such analyses.

Assessment of dietary intake of sugars, whether by some method of recall as used in the trials, or by food frequency questionnaires as in cohort studies, was associated with a considerable degree of measurement error even when using validated methods. This is probably one explanation why a dose-response effect could not be shown between change in dietary intake and magnitude of weight change. Nevertheless, even crude estimates of intake provided assistance in interpreting potentially inconsistent findings. The studies of long term intervention in children[27 29 33 40 46] and two studies of interventions reducing dietary sugars in adults[28 33] found little difference in intakes between intervention and control groups, and no meaningful change in weight.

The heterogeneity of the studies, especially in terms of the consequences of altering intake of sugars in ad libitum diets, resulted in difficulties in fully explaining the effects of different dietary changes. Nevertheless, the changes in weight observed in studies of adults provided some indication of what might be achieved by the implementation of a dietary guideline relating to sugar, and conversely what might occur if consumption continued to increase.

The potential problem of residual confounding to explain some or all of an effect is inherent to all cohort studies. However, the overall consistency of our findings, regardless of study type, is reassuring. The only potential major source of bias identified in the trials was that four trials in adults reported data for completers. These data could have overestimated the effect, but we saw no meaningful difference in the magnitude of the effect between these trials and the other studies. Both participants and researchers in many of the trials were not blinded to intervention allocation. Studies providing beverages as a means of manipulating sugars intakes were blinded, but blinding was clearly not possible in studies relying on the provision of dietary advice to manipulate sugars intake. However, we do not believe that a lack of blinding altered our findings substantially. Measurement of body weight did not involve judgment that was subject to bias.

## Potential mechanisms

The most obvious mechanism by which increasing sugars might promote weight gain is by increasing energy consumption to an extent that exceeds energy output and distorts energy balance. For sugar sweetened beverages, it has been suggested that energy in liquid form could be less satiating than when derived from solid foods, resulting in increased consumption.[104] Solid foods containing sugars are typically (although not invariably) energy dense, and frequent and substantial consumption of energy dense foods is associated with excessive weight gain and other measures of excess adiposity. We observed that isoenergetic replacement of dietary sugars with other macronutrients resulted in no change in weight (fig 5). This finding strongly suggested that energy imbalance is a major determinant of the potential

for dietary sugars to influence measures of body fatness. However, other less direct mechanisms independent of energy balance have been proposed.

Sugars (particularly table sugar, sucrose, and high fructose corn syrup) contribute to the intake of fructose, which in turn can, at least in some people, increase levels of uric acid and hyperinsulinaemia.[105] Hyperuricaemia has been identified as a potentially important and independent predictor of obesity and the metabolic syndrome.[2] Sugar sweetened beverages and other sources of dietary fructose have been suggested to promote the deposition of liver, skeletal, and visceral fat and an increase in serum lipids independently of an effect on body weight.[106] Although this issue is relevant to any overarching discussion regarding the health consequences of dietary sugars and the extent to which they should be restricted, it is beyond the scope of this review.

## Results in the context of existing knowledge

Most of the relevant published studies, reviews, and meta-analyses related to the association between intake of sugar sweetened beverages and body weight, weight gain, or other measures of adiposity. Widely discrepant conclusions have emerged, ranging from strong or convincing evidence for an association[8 107] to evidence described as inconclusive or equivocal.[3 7 11 108-110] This variance is hardly surprising, owing to the poor compliance in most intervention trials, the insensitive instruments used for assessing dietary intakes in cohort studies, and that in such studies, intakes might have changed between initial dietary assessment and measurement of outcome. One meta-analysis combined data for adults and children.[11] We found no evidence for an association between intake and weight in children when considering the intervention trials, nor were the data sufficient to examine for a dose-response effect when considering β coefficients for the continuous association between baseline sugars exposure and adiposity outcome. Nevertheless, we were able to show a consistent effect when comparing groups with the highest intakes of sugars with those with the lowest intakes.

There have been fewer reviews and meta-analyses relating to sugars or sugar rather than sugar sweetened beverages. In a systematic review and meta-analysis, Sievenpiper and colleagues concluded that isoenergetic substitution of fructose for other carbohydrates was not associated with weight gain.[110] However, free fructose at high doses that provided excess calories modestly increased body weight to an extent probably due to the extra calories rather than any particular metabolic attributes of fructose. Dolan and colleagues[111] drew similar conclusions when reviewing studies in which fructose was fed at "normal levels of intake." Van Baak and Astrup[3] and Ruxton[104] recently concluded that there was insufficient evidence to indicate that replacing sugars with other carbohydrates resulted in a reduction in body weight. However, by limiting analyses to ad libitum trials, and considering studies in adults and children separately, our systematic review showed a clear positive association between higher intake of sugars and body fatness in adults, and provided an explanation as to why the findings in children were less conclusive.

## Conclusions

This series of meta-analyses provides evidence that intake of sugars is a determinant of body weight in free living people consuming ad libitum diets. The data suggest that the change in body fatness that occurs with modifying intake of sugars results from an alteration in energy balance rather than a physiological or metabolic consequence of monosaccharides or disaccharides. Owing to the multifactorial causes of obesity, it is unsurprising that the effect of reducing intake is relatively small. The extent to which population based advice to reduce sugars might reduce risk of obesity cannot be extrapolated from the present findings, because few data from the studies lasted longer than ten weeks. However, when considering the rapid weight gain that occurs after an increased intake of sugars, it seems reasonable to conclude that advice relating to sugars intake is a relevant component of a strategy to reduce the high risk of overweight and obesity in most countries.

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WHO agreed to the publication of this systematic review in a scientific journal, because it serves as the background evidence review for updating WHO guidelines on total sugars intake and should therefore, be available widely.

Contributors: The questions for the review were discussed and developed by the WHO NUGAG Subgroup on Diet and Health in February 2010, and the protocol was approved by the NUGAG Subgroup on Diet and Health. LT and SM supervised study searches. LT, SM, and JIM assessed inclusion, extracted data, and assessed validity. LT did the meta-analyses. LT and JM wrote the manuscript. The NUGAG Subgroup on Diet and Health reviewed the first draft of the report and contributed to the GRADE assessment. All authors read and approved the final draft of the report.

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**What is already known on this topic**

Excessive intakes of dietary sugars have been linked to obesity, and a higher risk of chronic diseases, but the link with obesity is tenuous

The most consistent association has been between a high intake of sugar sweetened beverages and the development of obesity

No upper safe limit of intake has been agreed universally, but WHO has suggested that intakes of free sugars should be less than 10% of the total energy intake

**What this study adds**

Among free living people, advice to reduce free sugars was associated with an average 0.80 kg reduction in weight; advice to increase intake was associated with a corresponding 0.75 kg increase

This parallel effect seems to be due to an altered energy intake; isoenergetic replacement of sugars with other carbohydrates did not result in any change in body weight

Evidence was less consistent in children than in adults


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# Tables

**Table 1| Classification of dietary carbohydrates**

| Class* | Subgroup | Principal components |
|---|---|---|
| Sugars (1-2) | Monosaccharides | Glucose, fructose, galactose |
| | Disaccharides | Sucrose, lactose, maltose, trehalose |
| | Polyols (sugar alcohols) | Sorbitol, mannitol, lactitol, xylitol, erythritol, isomalt, maltitol |
| | Free sugars | All monosaccharides and disaccharides added to foods by the manufacturer, cook, or consumer; sugars naturally present in honey, syrups, and fruit juices |
| Oligosaccharides (3-9) | Malto-oligosaccharides (α glucans) | Maltodextrins |
| | Non-α glucan oligosaccharides | Raffinose, stachyose, fructo and galacto oligosaccharides, polydextrose, inulin |
| Polysaccharides (≥10) | Starch (α glucans) | Amylose, amylopectin, modified starches |
| | Non-starch polysaccharides | Cellulose, hemicellulose, pectin, arabinoxylans, β glucan, glucomannans, plant gums and mucilages, hydrocolloids |

*Degree of polymerisation or number of monomeric (single sugar) units in brackets.

Adapted from references 13 and 14.

**Table 2| Characteristics of trials examining the effect of reducing intake of free sugars on measures of body fatness in adults**

| Study | Methods | Participants | Diet | | Outcomes | Study duration | Dose |
|---|---|---|---|---|---|---|---|
| | | | Intervention | Control | | | |
| Gatenby 1997[28] | Randomised intervention study | 31 healthy women of normal weight, aged 18-50 years | Ad libitum diet using reduced sugar foods | Usual diet | Dietary intake, change in weight | 10 weeks | Data not clearly reported; intervention reduced sugar intake by about 4% of total energy *v* about 1% of total energy in control group |
| Mann 1972[30,31] | Randomised controlled trial (parallel design) | 51 Apparently healthy male office workers from one company, aged 36-55 years | Ad libitum diet with low sugar (sucrose) | Usual diet | Serum lipids, body weight change | 22 weeks | Mean reduction of 71 g/day in the intervention group *v* increase of 3 g/day in controls. Difference of 11% TE |
| Paineau 2008[33] | Randomised controlled trial | 1013 children from 54 elementary schools in Paris and 1013 parents | Nutrition education to reduce fat and sugar intake and increase intake of complex carbohydrates | Nutrition education to reduce fat intake and increase intake of complex carbohydrates | Changes in anthropometry including BMI, BMI z score, and changes in dietary intakes in children and parents | 8 months | Parents had reduction of 9.6 g/day *v* 7.3 g/day. Difference of <1% TE |
| Saris 2000[39] | Randomised controlled trial | 159 healthy, overweight, and obese (BMI 25-35) adults, aged 20-55 years | Ad libitum diet high in SCHO (that is, sugars) | Ad libitum diet high in complex carbohydrates | Weight change, fat mass, dietary intakes, lipids, fasting glucose, fasting insulin, leptin | 6 months | Increase of 33 g/day SCHO versus a reduction of 44.5 g/day SCHO. Difference of 11% TE* |
| Smith 1996[41] | Randomised controlled trial | 32 middle aged and overweight men with hypertriglyceridaemia | Ad libitum, sugar free diet | Usual diet | Lipids, weight, dietary variables | 6 months | Reduction of 48 g/day sucrose *v* no change. Difference of about 8% TE |

SCHO=simple carbohydrates; TE=total energy intake.

*About 60% of all food was supplied via a controlled study shop.

**Table 3| Characteristics of randomised trials examining the effect of increasing intake of free sugars on measures of body fatness in adults**

| Study | Methods | Participants | Diet | | Outcomes | Study duration | Dose | Notes |
|---|---|---|---|---|---|---|---|---|
| | | | Intervention | Control | | | | |
| Aeberli 2011[47] | Randomised crossover trial | 29 healthy normal weight men aged 20-50 years, living in and around Zurich, Switzerland | High sugars (fructose, glucose, or sucrose), providing 80 g/day | Moderate sugars (fructose or glucose), providing 40 g/day | Lipid and glucose metabolism (focusing on LDL particle size in particular), inflammatory markers, weight change | 3 weeks per treatment, 4 week washout between treatments | 80 g/day added sugars *v* 40 g/day. Difference of 6.6% TE | Study involved 6 treatments. Data were aggregated for the intake comparisons between moderate and high sugars groups |
| Brynes 2003[26] | Randomised crossover trial | 17 healthy middle aged men with one or more cardiac risk factors | High carbohydrate, high sucrose diet, providing additional 90 g/day of sucrose | High carbohydrate, high glycaemic index diet | Weight change, fasting lipids, postprandial glucose, insulin and lipids, HOMA insulin sensitivity | 24 days per treatment | 132 g/day *v* 46 g/day of sucrose. Difference of 14% TE | Energy intake was intended to be consistent between treatments |
| Marckmann 2000[32] | Randomised crossover trial | 20 post-obese adults; controls matched by age, height, and weight | Ad libitum, high sucrose diet | Ad libitum high starch diet | Weight change, change in fat mass | 2 weeks per treatment | 23% *v* 2.5% total energy sucrose. Difference of 20.5% TE | — |
| Poppitt 2002[34] | Randomised controlled trial | 28 overweight adults with three or more metabolic risk factors | Ad libitum, low fat, high simple carbohydrate diet | Ad libitum, low fat, high complex carbohydrate diet | Weight change, change in energy intake, waist circumference, BMI, lipids | 6 months | Increase in simple carbohydrate of 87 g/day *v* 2 g/day. Difference of 14% TE | 13 participants also included in the CARMEN study[39] |
| Raben 2002[35] | Randomised controlled trial | 41 healthy overweight adults (BMI 25-30) aged 20-50 years | Ad libitum diet supplemented with sucrose containing foods and beverages providing 28% of total energy | Ad libitum diet supplemented with artificially sweetened foods and beverages | Dietary intake, weight, fat mass, fat free mass | 10 weeks | Increase of 105 g/day sucrose *v* decrease of 15 g/day. Difference of 23% TE | — |
| Reid 2007[38] | Randomised controlled dietary intervention | 133 normal weight women aged 20-55 years | Ad libitum diet with 1 L/day of sugar sweetened beverages | Ad libitum diet with 1 L/day of artificially sweetened beverages | Mood, weight change, dietary intake | 4 weeks | Sugar sweetened drinks provided 105 g/day of sucrose. Difference of about 20% TE | Weight change data extracted from figures in article |
| Reid 2010[37] | Randomised controlled dietary intervention | 53 overweight women (BMI 25-30) aged 20-55 years | Ad libitum diet with 1 L/day of sugar sweetened beverages | Ad libitum diet with 1 L/day of artificially sweetened beverages | Mood, weight change, dietary intake | 4 weeks | Sugar sweetened drinks provided 105 g/day of sucrose. Difference of about 20% TE | Weight change data extracted from figures in article |
| Szanto 1969[43] | Crossover trial | 19 apparently healthy men | High sucrose diet (substituting sucrose for starch) | Low sucrose diet (10 g/day) | Glycaemic responses, insulin responses, lipids, weight | 2 weeks per treatment, 2 week washouts | 438 g/day *v* 10 g/day of sucrose. Difference of >20% TE | 2 week washout sufficient to restore weight to baseline values |
| Tordoff 1990[44] | Randomised crossover trial | 30 healthy, normal weight adults | Ad libitum diet supplemented with 1135 g/day of HFCS-sweetened soda | Ad libitum diet supplemented with 1135 g/day of aspartame sweetened soda | Dietary intake, weight, height, dietary restraint | 3 weeks per treatment | Drinks provided 133 g/day *v* 1 g/day high fructose corn syrup. Difference of 18% TE | — |
| Werner 1984[45] | Randomised crossover trial | 12 adults with radiolucent gallstones and bile supersaturated with cholesterol, but with normal liver function, insulin and glucose status | Ad libitum, high sucrose (>100g/day), fibre depleted diet | Ad libitum, low sucrose, fibre depleted diet | Weight, dietary intakes, bile, cholesterol saturation index, bilary secretion rates, bile acid pool, bilary lipids, blood lipids, fasting plasma glucose | 6 weeks per treatment | 112 g/day *v* 16 g/day of refined sugar. Difference of 18% TE | — |

HFCS=high fructose corn syrup; LDL=low density lipoprotein; HOMA=homeostasis model assessment for insulin sensitivity; TE=total energy intake.

**Table 4| Characteristics of trials comparing the effect on body weight change in adults of isocaloric diets high in free sugars with diets relatively low in free sugars**

| Study | Methods | Participants | Diet | | Outcomes | Study duration | Dose |
|---|---|---|---|---|---|---|---|
| | | | Intervention | Control | | | |
| Bantle 1992[48] | Randomised crossover trial of isocaloric metabolically controlled dietary interventions | 12 men and women with type 2 diabetes | High fructose, high carbohydrate diet (55% of energy) | High starch, low sugars, high carbohydrate diet (55% of energy) | Plasma glucose, urinary glucose, lipids, postprandial triglycerides, body weight | 4 weeks per treatment | 20% of total energy/day from sucrose *v* <3% of total energy/day from fructose |
| Bantle 1993[49] | Randomised crossover trial of isocaloric metabolically controlled dietary interventions | 12 men and women with type 2 diabetes; 6 with type 1 diabetes | High sucrose, high carbohydrate diet (55% of energy) | High starch, low sugars, high carbohydrate diet (55% of energy) | Plasma glucose, urinary glucose, lipids, postprandial triglycerides, body weight | 4 weeks per treatment | 19% of total energy/day from sucrose *v* <3% of total energy/day from sucrose |
| Grigoresco 1988[50] | Randomised crossover comparing isocaloric, free living diets | 8 well controlled adults with type 2 diabetes | Isoglucidic diet replacing 30g/d starch with 30g/d fructose | Starch diet | Glycaemic control, glycaemic and insulinaemic responses, uric acid, lipids, body weight | 2 months | 30 g/day of fructose *v* 30 g/day of starch |
| Koivisto 1993[51] | Double blind, randomised, isocaloric, hospital inpatient, crossover study | 10 men and women with type 2 diabetes | High carbohydrate diet supplemented with crystalline fructose | High carbohydrate diet | Glycaemic control, lipid and lipoprotein metabolism, insulin sensitivity | 4 weeks | 20% of total energy (45-65 g/day) from fructose replacing complex carbohydrate |
| Malerbi 1996[52] | Crossover trial of isocaloric, weight maintaining diets | 16 free living men and women with type 2 diabetes | (1) High fructose diet; (2) high sucrose diet | High starch diet | Metabolic and B cell secretion effects | 28 days per treatment with 14 day washout periods | (1) 63 g/day of fructose and 5 g/day of sucrose; (2) 3 g/day of fructose and 79 g/day of sucrose; control: 3 g/day of fructose and 14 g/day of sucrose |
| Mann 1972b[53] | Randomised crossover trial of hospital treatments | 9 normolipidemic men with history of non-metabolic health condition in previous year | Typical Western diet with about 140 g/day of sucrose | Typical Western diet with isocaloric replacement of sucrose with complex carbohydrate | Serum lipids | 14 days | 140 g/day in the intervention group, which was replaced with starch in the control group |
| Mann 1973[54] | Clinical metabolic inpatient crossover trial of isocaloric, controlled energy diets | 9 men (7 with chronic neurological disorders, 2 apparently healthy) | High sucrose, Western diet | Normal sucrose Western diet | Serum lipids | 14 days | 160 g/day *v* 80 g/day of sucrose (difference of 17% TE)* |
| Osei 1989[55] | Randomised crossover study | 13 men and women with type 2 diabetes (outpatients) | Weight maintaining, diabetic, high carbohydrate (50% of energy), high fructose diet | Isocaloric, weight maintaining, diabetic diet, high in complex carbohydrates (50% of energy) | Serum glucose, $HbA_{1c}$, lipids, serum uric acid, serum lactic acid, weight | 6 months per treatment | 60 g/day of crystalline fructose replacing complex carbohydrate |
| Peterson 1986[56] | Randomised crossover trial of two 6 week diets | 23 non-obese men and women with type 1 and type 2 diabetes, otherwise healthy | High sucrose diet (same as for control diet but 45 g/day sucrose replacing starch) | High fibre, high CHO diet | Glycaemic control, lipids | 6 weeks per treatment | 45 g/day of sucrose replaced with 45 g/day of complex carbohydrate |
| Santacroce 1990[57] | Randomised crossover study comparing two isocaloric diets | 12 adults with insulin dependent diabetes | Typical Italian, high CHO, low fat, diabetic diet supplemented with sucrose | Typical Italian, high CHO, low fat, low sucrose diabetic diet | Glycaemic control, lipids | 2 months per treatment | 30 g/day of sucrose replaced with 30 g of starch |
| Swanson 1992[58] | Randomised crossover design feeding trial comparing isoenergetic diets | 14 healthy, normal to overweight men and women | High fructose (20% TE) diet | Low fructose (<3% TE), high starch diet | Metabolic variables including $HbA_{1c}$, glycaemic responses, serum lactate, lipids | 28 days per treatment | 100 g/day *v* 14 g/day of fructose† |

CHO=carbohydrates; TE=total energy intake; $HbA_{1c}$=glycated haemoglobin.

*Treatments were not given in a randomised order.

†Carbohydrates differed only in the proportions of starch and fructose.

**Table 5| Summary of prospective cohort studies examining association between free sugars exposures and adiposity in adults**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Bes-Rastrollo (2010),[59] Spain, SUN Project | 10 162 Spanish university graduates; convenience sampling | 4.4 | SSB intake separated into thirds; FFQ | Weight change (kg) (continuous), weight gain ≥3 kg (OR), incident obesity (HR); self reported | Adjusted for age, alcohol intake, baseline BMI, dietary intake, physical activity, sex, sitting, smoking, total energy intake, TV viewing | Consumption of SSB was associated with greater weight gain in the group with the highest intake relative to the group with the lowest. However, there was no association with risk of developing obesity | +/0 |
| Colditz (1990),[76] USA, Nurses' Health Study | 31 940 married registered female nurses aged 30-55 years; consecutive sampling | 4 | Sucrose (g/day); FFQ | Weight gain (kg) (continuous); self reported | Adjusted for age, baseline BMI, total energy intake | Sucrose intake not significantly associated with weight gain at follow-up | 0 |
| Dhingra (2007),[60] USA, Framingham Offspring Study | 4028 middle aged adults, mean age 51-56 years, whose parents were in the Framingham Heart Study; random sampling used in original Framingham Heart Study cohort | 4 | SSSD (0, <1, ≥1, ≥2 servings/day); questionnaire | Incident obesity (BMI ≥30) and incident high WC (men ≥102 cm, women ≥88 cm) (OR); measured | Adjusted for age, baseline BMI/WC, dietary intake, glycaemic index, physical activity, sex, smoking, total energy intake | Compared with no SSSD intake, SSSD consumption was associated with incident obesity (1 serving/day: OR 1.21 (95% CI 0.90 to 1.62); ≥1: 1.31 (1.02 to 1.68); ≥2: 1.50 (1.06 to 2.11)). SSSD consumption was significantly associated with developing a high WC across the intake categories (1: 1.25 (1.02 to 1.54); ≥1: 1.40 (1.08 to 1.83); ≥2: 1.30 (1.09 to 1.56)) | + |
| Drapeau (2004),[61] Canada, Québec Family Study | 248 adults aged 18-65 years, living within 80 km radius of Québec; convenience sampling | 5.9 | Self perceived change (increase, maintenance, or decrease) in the intake of sugar, sweet foods, and SSSD; questionnaire | Change in weight (kg), BF (%), sum of 6 skinfolds (cm), WC (cm) (all continuous); measured | Adjusted for age, body weight indicators at baseline, change in dietary intake, change in physical activity | In unadjusted ANCOVA analyses, participants reporting an increase in intake of sugar/sweet foods had a significantly higher increase in WC and sum of 6 skinfolds than those reporting a decrease (P<0.05), while no differences were seen for weight change or BF (%). No differences were seen for any body weight indicators after an increase in SSSD consumption. In adjusted regression analyses, an increase in consumption of sugar/sweet foods was significantly associated with an increased in WC (0.16 cm) and sum of 6 skinfolds (1.62 cm; both P=0.03) | +/0 |
| French (1994),[62] USA, Healthy Worker Project | 1639 working men (mean age 39.1 years, SD 9.8) and 1913 women (mean age 37.3 years, SD 0.7) participating in an intervention study of smoking cessation and obesity prevention; convenience sampling of worksites, random sampling of workers | 2 | Sweet foods and SSSD (servings/week); FFQ | Weight change (in pounds) (continuous); measured | Adjusted for dieting behaviour at baseline and follow-up, baseline intake of sweets/SSSD, baseline weight, education, intervention group, marital status, occupation, smoking, worksite; stratified by sex | Consumption of sweet foods was associated with weight gain over time in both men and women. Each serving/week of sweet foods at baseline was associated with an weight increase of 0.28 pounds (0.13 kg, SE 0.04 kg) and 0.19 pounds (0.09 kg, 0.04 kg) in women and men, respectively, over the 2 years (both P≤0.02). No significant association was seen between SSSD intake and weight gain | + |
| Halkjaer (2004),[63] Denmark, MONICA | 2275 Danish adults aged 30, 40, 50, or 60 years; random sampling | 6 | Intake of sweet foods divided into fifths; FFQ | Change in WC (cm) (continuous); measured | Adjusted for age, alcohol intake, baseline BMI and current BMI, baseline hip circumference, baseline WC, change in dietary intake, education, physical activity, smoking; stratified by sex | No relation was seen between sweet foods consumption and WC | 0 |

**Table 5 (continued)**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Halkjaer (2006 and 2009),[64 65] Denmark, Danish Diet, Cancer and Health Study | 20 126 men and 22 570 women aged 50-64 years, living in greater Copenhagen or Aarhus areas; consecutive sampling | 5.3 | Jams, sugars and syrups (250 kJ/day), SSSD (250 kJ/day), and energy intake from foods with added sugar (MJ/day); FFQ | Change in WC (cm/5 years) (continuous); measured at baseline, self reported at follow-up | Adjusted for age, alcohol intake, baseline BMI and WC, energy intake from other sources, physical activity, smoking | Intake of jams, syrups, sugars, or SSSD was not associated with change in WC in women or men. In women only, energy intake from foods with added sugar at baseline was significantly associated with a 5 year increase in WC (per MJ/day: 0.39 cm increase (95% CI 0.18 to 0.60); P<0.001) | + (women only) |
| Hendriksen (2011),[66] Holland, MORGEN-EPIC | 11 111 adults aged 20-64 years, living in Amsterdam, Maastricht, or Doetinchem; random sampling | 4.9/9.9 | Sweets (418 kJ/day) and cakes (418 kJ/day) intake; FFQ | Annual weight change (g/year) (continuous); self reported | Adjusted for age, baseline weight and height, dietary intake, duration of follow-up, education, physical activity, sex, smoking, SSSD intake, total energy intake | No association between weight change and consumption of sweets or cakes was seen | 0 |
| Kvaavik (2004),[67] Norway, Oslo Youth Study | 371 Norwegian adults aged 23-27 years who participated in a school based intervention study 10 years previously; convenience sampling | 8 | Long term intake of SSSD (low consumption (<3 times/week at first and second follow-up), high consumption (≥3 times/week at first and second follow-up), or inconsistent consumption); questionnaire | Prevalent overweight (BMI ≥25) or obesity (BMI ≥30) at second follow-up (OR); self reported | Adjusted for baseline (adolescent) BMI; stratified by sex | No associations were seen between long term consumption of SSSD and overweight or obese status | 0 |
| Mozaffarian (2011),[68] USA, Nurses' Health Study I and II, Health Professionals Follow-up Study | 50 422 married registered female nurses aged 30-55 years, 47 898 registered female nurses aged 24-44 years, and 120 877 male health professionals aged 40-75 years; consecutive sampling | 4 | Increase in consumption (servings/day) of SSB, FJ, sweets, or desserts; FFQ | Weight gain (in pounds) over 4 year periods (continuous); self reported | Adjusted for age, baseline BMI, change in alcohol intake, change in dietary intake, change in physical activity, change in smoking, change in TV viewing, sleep duration | In pooled analysis, each serving/day increase in SSB intake was associated with a 1 pound increase in weight (0.45 kg (95% CI 0.36 to 0.53); P<0.001). Each serving/day increase in FJ was associated with a 0.31 pound increase in weight (0.14 kg (0.06 to 0.21); P<0.001). Each serving/day increase in sweets or desserts was associated with a 0.41 pound increase in weight (0.19 kg, 0.07 to 0.30; P<0.001) | + |
| Nooyens (2005),[69] Holland, Doetinchem Cohort Study | 288 men aged 50-60 years, attending a municipal health clinic; consecutive sampling | 5 | Change in intake of SSSD (glasses/day); FFQ | Change in body weight (kg/year) and change in WC (cm/year) (both continuous); measured | Adjusted for age, alcohol intake, dietary intake, occupation, physical activity, retirement status, smoking, total energy intake | In unadjusted analyses, an increase in SSSD intake from baseline to follow-up was positively associated with change in body weight and WC (per glass/day increase, β 0.2 and 0.16, respectively; both P≤0.04). After adjustment, associations were rendered non-significant (both P≥0.05) | +/0 |
| Odegaard (2010),[70] Singapore, Singapore Chinese Health Study | 43 580 Hokkien and Cantonese speaking Singaporeans aged 45-74 years, residing in housing estates built by the government; consecutive sampling | 5.7 | Intake of SSSD and intake of fruit drinks or FJ (servings per week or per month); FFQ | Weight change (kg) (continuous); self reported | Adjusted for age, alcohol intake, BMI, dialect, dietary intake, education, person years, sex, smoking, year of interview | Participants in the highest category of SSSD consumption (>2 servings/week) had a significant increase in weight (0.53 kg) compared with those who did not consume SSSD or reported only monthly consumption (P<0.001). There was no association between intake of fruit drinks/FJ and change in mean weight between baseline and follow-up. A test for interaction between SSSD intake and weight gain over time was significant (P=0.007) | + (SSSD), 0 (FJ) |

**Table 5 (continued)**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Palmer (2008),[71] USA, Black Women's Health Study | 43 960 African American women aged 21-69 years; convenience sampling | 6 | Change in intake of SSSD and change in intake of fruit drinks (servings/day); FFQ | Weight gain (kg) (continuous); self reported | Adjusted for baseline age and BMI, dietary intake and change in dietary intake, education, family history of diabetes, physical activity and change in physical activity, smoking and change in smoking | Compared with women who reduced intake of SSSD (≥1 to ≤1 servings/day), those who increased intake (≤1 to ≥1) gained significantly more weight over the 6 year follow-up (4.1 kg (SD 0.22) *v* 6.8 kg (0.28); P<0.01). No significant associations were noted between weight gain and change in fruit drink intake | + (SSSD)<br>0 (FJ) |
| Parker (1997),[72] USA, Pawtucket Heart Health Program | 465 adults aged 18-64 years, participating in a community based programme for cardiovascular disease prevention; random sampling | 4 | Intake of sugar (sucrose; g/day) and sweets (servings/week); FFQ | Weight change (kg) (continuous); measured | Adjusted for age, BMI, physical activity, smoking, total energy intake | Intake of sucrose and sweets at baseline was not associated with change in weight at follow-up | 0 |
| Schulz (2002),[73] Germany, EPIC-Potsdam Cohort | 17 396 adults aged 19-70 years, enrolled in EPIC in Potsdam; convenience sampling | 2.2 | 100 g/day increments in intake of sweets, cakes, and biscuits; SSSD; and desserts. FFQ | Weight change (OR; kg/year; large gain (≥2), small gain (1-2), stable (gain or loss of 1), small loss (1-2), large loss (≥2); self reported | Adjusted for age, baseline weight and height, change in dietary intake, education, life and health contentment, drugs use, prevalent diabetes, prevalent stroke, weight cycling history; stratified by sex | Per 100 g/day higher increment in intake of sweets at baseline, men were more likely to have a large gain or small loss in weight than remain stable (OR 1.48 and 1.43, respectively; both P<0.05). In men, SSSD intake at baseline was positively associated with large weight gain (OR 1.03), small weight loss (1.02) and large weight loss (1.03; all P<0.05). Per 100 g/day higher increment in intake of sweets or cakes/biscuits at baseline, women were less likely to have a large loss in weight than remain stable (0.67 and 0.88, respectively; both P<0.05). In women, SSSD intake at baseline was positively associated with large weight loss (1.02, P<0.05) | + and – |
| Schulze (2004),[74] USA, Nurses' Health Study II | 51 603 registered female nurses aged 24-44 years; consecutive sampling | 4 | Change in consumption of SSSD, fruit drinks, FJ (from ≤1 time/week to ≥1 time/day, ≥1 time/day to ≤1 time/week, consistently ≤1 time/week, consistently ≥1 time/day); FFQ | Weight change (kg) and BMI change (both continuous); self reported | Adjusted for baseline values of age, alcohol intake, BMI, dietary intake, oral contraceptive use, physical activity, postmenopausal hormone use, and smoking, and changes in all variables over time | Women who increased consumption of SSSD gained more weight and reported a higher BMI at follow-up than those who decreased consumption or maintained a high or low intake (all P<0.001). Similarly, women who increased consumption of fruit drink/FJ gained more weight than those who decreased consumption (P<0.001) | + (only in women who increased intake) |

ANCOVA=analysis of covariance; BF=body fat; FFQ=food frequency questionnaire; FJ=100% fruit juice; HR=hazard ratio; OR=odds ratio; SE=standard error; SD=standard deviation; SSSD=sugar sweetened soft drinks; SSB=sugar sweetened beverages (including cordials, energy drinks, fruit drinks, iced tea, soft drinks); TV=television; WC=waist circumference.

*Most adjusted results are reported unless otherwise stated.

†Higher sugar intake positively associated with weight gain (+), not associated with weight gain (0), and negatively associated with weight gain (–).

**Table 6| Characteristics of intervention studies measuring the effect of advice to reduce intakes free sugars on change in BMI in children and adolescents**

| Study | Methods | Participants | Diet | | Outcomes | Study duration | Dose |
|---|---|---|---|---|---|---|---|
| | | | Intervention | Control | | | |
| Davis 2009[46] | Randomised controlled trial | 68 Latino adolescents | Nutrition education on carbohydrate modification with targets of <10% total energy from added sugars and >14 g/1000 kcal dietary fibre | Usual diet | Glycaemic responses, insulin sensitivity, body composition, change in dietary intakes and activity levels | 16 weeks | Reduction in added sugar intake of 10 g/day *v* 2 g/day |
| Ebbeling 2006[27] | Randomised controlled intervention study | 103 adolescents aged 13-18 years consuming at least one serving/day of sugar sweetened beverages | Weekly home deliveries of non-caloric beverages, amounting to 4 servings per participant/day and two servings/day for additional household members, with behavioural counselling by telephone to encourage displacement of sugar sweetened beverages | Continued usual habits of beverage consumption | Change in BMI from baseline to follow-up, change in energy intake from carbonated beverages, consumption of non-caloric beverages | 25 weeks | Reduction in added sugar intake of 75 g/day *v* 12 g/day |
| James 2004[29] | Cluster randomised controlled trial | 644 primary school children aged 7-11 years from 6 schools and 29 classes in UK | Nutrition education to reduce consumption of carbonated beverages | No nutrition education | Change in BMI z score, change in soft drink consumption | 12 months | Reduction in 0.1 glasses/day of sugar sweetened carbonated drinks *v* no reduction |
| Paineau 2008[33] | Randomised controlled trial | 1013 children from 54 elementary schools in Paris, 1013 parents | Nutrition education to reduce fat and sugar intake and increase intake of complex carbohydrates | Nutrition education to reduce fat intake and increase intake of complex carbohydrates | Changes in anthropometry including BMI, BMI z score, changes in dietary intakes in children and parents | 8 months | Children: reduction of 10 g/day of sugar *v* 5.5 g/day |
| Sichieri 2009[40] | Randomised, controlled, cluster school based intervention | 1140 Brazilian children in the 4th grade (age 9-11 years), living in urban areas and of low socioeconomic status | Behavioural intervention to reduce intake of sugar sweetened beverages delivered in the classroom in 10×1 h sessions | 2×1 h general health sessions and printed advice regarding healthy diets | Beverage consumption, weight change, BMI | About 8 months | Reduction in carbonated beverages of 69 mL/day *v* 13 mL/day |

**Table 7| Summary of prospective cohort studies examining associations between free sugars exposures and adiposity in children**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Berkey (2004),[75] USA, Growing Up Today Study | 1175 adolescents aged 9-14 years, whose mothers were in the Nurses' Health Study II; consecutive sampling used in original Nurses' Health Study II cohort | 1 | SSB intake (serving/day) and increase in SSB intake (1 serving/day increase); FFQ | Change in BMI (continuous); self reported | Adjusted for age, dietary intake, ethnicity, growth, physical activity, previous BMI z score, screen time, Tanner stage, total energy intake; stratified by sex | In boys, significant associations between SSB consumption at baseline and 1 year weight gain, and between increasing SSB consumption and 1 years weight gain were seen in adjusted analyses (both P≤0.038). After adjusting for total energy intake, effect sizes in boys and girls were reduced, and associations were rendered non-significant | + |
| Blum (2005),[83] USA | 164 children aged 9.3 years (SD 1) attending elementary school; convenience sampling | 2 | SSB intake (ounces/day); 24 h recall | Change in BMI z score (continuous); measured | Adjusted for baseline BMI z score, intake of diet soda at follow-up | Regression analysis of BMI z score at follow-up showed no association with SSB consumption (data not reported) | 0 |
| Butte (2009),[84] USA, Viva la Familia Study | 789 Hispanic adolescents aged 9-14 years, at least 1 overweight child in family; convenience sampling of families | 1 | Energy intake (%) from sucrose, and from added sugar; 24 h recall | Weight gain (kg) (continuous); measured | Adjusted for age, BMI status sex, Tanner stage | Energy intake from sucrose and added sugars was not found to be associated with weight gain | 0 |
| Dubois (2007),[94] Canada, Longitudinal Study of Child Development in Québec | 380 children aged 2.5 years; random sampling | 2 | SSB intake between meals at age 2.5, 3.5, and 4.5 years, categorised as non-consumers, regular consumers, or other (including children who changed consumption); FFQ | Overweight BMI (≥95th percentile) at age 4.5 years (OR); measured | Adjusted for birth weight, dietary intake, maternal smoking, number of obese parents, physical activity, SES, sex, total energy intake | Regular consumers were 2.36 times more likely to be obese at age 4.5 years than non-consumers (95% CI 1.03 to 5.39; P≤0.05) | + |
| Faith (2006),[81] USA | 825 children aged 1-5 years enrolled in the Special Nutrition Program for Women, Infants and Children in New York State; consecutive sampling | 2 | FJ intake (servings/day) ; questionnaire | Change in BMI z score (continuous); measured | Adjusted for baseline weight-for-height z score and weight status, dietary intake, parental feeding behaviour; stratified by being at risk of overweight (weight-for-height z score ≥85th percentile) and not being at risk of overweight | At follow-up, each serving/day of FJ intake at baseline was associated with a 0.005 increase in BMI z score (P<0.01) in pooled analysis, a 0.009 increase for people at risk of overweight (P<0.01), and a non-significant increase of 0.003 for those not at risk of overweight | + |
| Fiorito (2009),[86] USA | 166 girls aged 5 years at baseline; convenience sampling | 10 | SSB intake (<1, ≥1 to <2, or ≥2 servings/day, and as a continuous variable); 24 h recall | BF (%), WC (cm), BMI percentile, overweight status (% overweight in each SSB intake group) (all continuous); measured | Adjusted for maternal BMI, parental education, SSB intake at 15 years, total energy intake (all adjusted for separately) | SSB intake at age 5 years was a significant predictor of adiposity at each 2 year time point during ages 5-15 years in unadjusted ANOVA analyses. Greater consumption of SSB at 5 years was associated with a higher WC at 15 years, after adjusting for intake of SSB and energy at 15 years and family income at 15 years (all P<0.05), but association was non-significant after adjusting for maternal BMI and parental education | + |
| Haerens (2010),[77] Belgium, Longitudinal Eating and Activity Study | 585 children aged 10 years attending elementary school; random selection of schools, consecutive sampling of children | 4 | Intake (servings/week) of SSSD and sweets; FFQ | Change in BMI z score (continuous); self reported | Adjusted for dietary intake and change in dietary intake, SES, sex, time since baseline | No associations seen between consumption of SSSD/sweets and BMI z score | 0 |

**Table 7 (continued)**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Herbst (2011),[87] Germany, DONALD Study | 216 infants aged 1 year, born to German citizens; convenience sampling | 6 | Energy intake (%) from total added sugar, added sugar in beverages and sweets, added sugar from other sources, plus changes in intake between 1-2 years; 3 day diet record | BMI z score and BF (%) (both continuous); measured | Adjusted for birth weight, BMI at birth, breastfeeding, dietary intake, gestational age (weeks), paternal education, maternal overweight, sex | Higher % energy intakes from total added sugars and added sugar in beverages and sweets at age 1 year were negatively associated with BMI z score at age 7 years. BMI z score increase per 1% energy from total added sugar: β –0.116 (SE 0.057, P=0.04; from added sugar in beverages and sweets: –0.250 (0.103, P=0.02). No associations were observed between % energy intakes from sugars and % BF | — |
| Johnson (2007),[98] UK, Avon Longitudinal Study of Parents and Children, Children in Focus | 521 children aged 5 years or 682 aged 7 years born in the final 6 month of the AVON study; consecutive population sampling used to obtain AVON cohort, random sampling within the cohort | 2/4 | Intake (servings/day) of SSB or FJ at 5 or 7 years; 3 day diet record | Fat mass index ($kg/m^{5.8}$)‡ and change in fat mass (kg) (both continuous); measured | Adjusted for baseline BMI, dietary intake, height, parental BMI and SES, sex, TV viewing | After adjusting for sex and height, fat mass index‡ at age 9 years correlated with SSB intake at 5 and 7 years (Pearson's correlation coefficient P values 0.06 and 0.03, respectively). In multivariate regression analysis, fat mass at 9 years was negatively associated with FJ intake at 5 years (–0.55 kg/daily serving, 95% CI –1.08 to –0.02) | + (SSB), – (FJ) |
| Libuda (2008),[88] Germany, DONALD Study | 235 children aged 9–13 years, born to German citizens; convenience sampling | 5 | Energy (MJ) derived from SSB, FJ, energetic beverages (SSB and FJ); and change in intake; 3 day diet record | Change in BMI z score (continuous) and % change in BF (continuous); measured | Adjusted for age, birth weight, energy intake from other sources and change in energy intake from other sources, maternal education and BMI, time, years of adolescence; stratified by sex | 5 year changes in BMI z score and BF (%) were not related to baseline intake of SSB, FJ, or combination of both. Change in BMI z score was positively associated with concurrent change in energetic beverage intake in girls (β 0.07, P=0.01) and with concurrent change in FJ intake in girls (β 0.096; P=0.013) | 0/+ |
| Lim (2009),[97] USA, Detroit Dental Health Project | 365 children aged 3-5 years from areas in Detroit with the highest proportion of low income households; random sampling | 2 | SSB (all, fruit drinks and SSSD) intake (ounces/day) and change in intake (decrease, increase, no change) FFQ | Change in BMI z score (continuous) and incident overweight (BMI ≥85th percentile) (OR); measured | Adjusted for baseline BMI, caregiver's BMI, SES, total energy intake | Odds of becoming overweight increased by 4% (95% CI 1% to 7%; P<0.05) per ounce of fruit drink consumed per day at baseline, 4% (1% to 6%; P<0.05) per ounce of all SSB consumed per day at baseline, and 4% per ounce of SSSD consumed per day at baseline, although non-significantly (95% CI –1% to 10%; P>0.05). No significant association found between change in beverage intakes and incidence of overweight (data not reported). No significant association found between change in beverage intakes and change in BMI z score | + |
| Ludwig (2001),[95] USA, Planet Health | 548 multiethnic children aged 11-12 years participating in an intervention study as controls; convenience sampling of schools, random assignment of schools to intervention/control, consecutive sampling of children | 1.6 | SSB intake (servings/day) and change in intake (increase of 1 serving/day); FFQ | Incident obesity (BMI and triceps skinfold ≥85th percentile) (OR) and change in BMI (continuous); measured | Adjusted for age, baseline BMI and triceps skinfold, dietary intake and change in dietary intake, ethnicity, physical activity, school indicator variables, sex, total energy intake, TV viewing, and change in TV viewing | For each serving of SSB consumed/day at baseline, BMI increased by 0.18 (95% CI 0.09 to 0.27; P=0.02), and for each SSB serve/d increase from baseline, BMI increased by 0.24 (0.10 to 0.39; P=0.03). Incident obesity was not associated with baseline SSB intake (OR 1.48, 95% CI 0.63 to 3.47; P=0.27), but was associated with an increase in SSB intake, with a 60% higher risk of developing obesity for each serving/day increase (95% CI 14–124%; P=0.02). | + |

**Table 7 (continued)**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Nissinen (2009),[82] Finland, Cardiovascular Risk in Young Finns Study | 2139 children aged 3, 6, 9, 12, 15, and 18 years; random sampling | 21 | Intakes of SSSD or sweets in childhood (per 10 units/month), increase, decrease, or no change in SSSD/sweets intake from childhood to adulthood; questionnaire | Adult BMI (continuous) and adult overweight status (BMI ≥25) (OR); measured | Adjusted for adult education, age, overweight status in childhood, physical activity, smoking; stratified by sex | The predicted difference in adult BMI per 10-unit consumption difference of SSSD or sweets in childhood did not reach statistical significance for males or females. An increase in the frequency of SSSD consumption from childhood to adulthood was associated with a higher BMI in adult females (β 0.45, SE 0.12; P<0.001). Adult male overweight status was not associated with change in intake of SSSD or sweets from childhood to adulthood. Adult female overweight status was not associated with change in intake of sweets, however, compared to females who maintained a low intake of SSSD from childhood to adulthood, those who increased intake were 1.9 times more likely to be overweight as adults (95% CI 1.38 to 2.61) | 0/+ |
| Phillips (2004),[89] USA, Massachusetts Institute of Technology Growth and Development Study | 132 premenarcheal girls aged 8-12 years attending public schools in Massachusetts; convenience sampling | About 7 | Energy intake (%) from energy dense snacks (SSSD, candy/chocolate, baked goods, ice cream), divided into groups according to intake level; FFQ | BMI z score and BF (%) 4 years after onset of menarche (both continuous); measured | Adjusted for age at menarche, dietary intake, parental overweight | Of 5 categories of energy dense snacks, only SSSD intake at baseline was significantly related to BMI z score 4 years after menarche, although the effect size was small (compared with the group with the lowest intake, β 0.172 for the group with the second highest intake, and β 0.178 for the group with the highest intake; both P<0.001). No associations were seen between intake of energy dense snack and BF (%) | + |
| Skinner (2001),[90] USA | 72 white children aged 2 years, born to parents with middle to high SES; convenience sampling | 4 | Longitudinal intake of FJ (ounces/day; mean across 7 sets of measurements); 24 h recall and 3 day diet record | BMI and ponderal index (kg/m$^3$) (both continuous); measured | Adjusted for age, baseline BMI or ponderal index, longitudinal total energy intake, parental BMI, sex | Longitudinal FJ intake was not significantly associated with BMI (β −0.057; P=0.099), but was negatively associated with ponderal index (β −0.065; P=0.05) | 0/– |
| Stoof (2011),[91] Holland, Amsterdam Growth and Health Longitudinal Study | 238 adolescents aged 13 years, attending secondary schools of middle to high SES in and around Amsterdam; convenience sampling | 24-30 | Intake of SSB (servings/day); diet history | BMI, BF (%) and trunk fat (%) (all continuous); measured | Adjusted for age, baseline BMI, developmental age at baseline, physical activity, total energy intake | For men, each additional daily serving of SSB at age 13 years was associated with greater BF (%) in adulthood (β 1.14, 95% CI (0.04 to 2.23); P=0.04). In women, each additional daily serving of SSB at 13 years was associated with greater trunk fat in adulthood (β 1.62 (0.14 to 3.10); P=0.03). No relation between SSB consumption at age 13 years and BMI in adulthood found in either sex | + |
| Striegel-Moore (2006),[92] USA, National Heart, Lung and Blood Institute Growth and Health Study | 2371 non-Hispanic black and white girls aged 9-10 years, attending schools in Richmond, Hamilton County, and Maryland, from families enrolled in a health maintenance organisation in the Washington, DC area, and girl scouts in the Washington, DC area; convenience sampling | 10 | Intake of SSSD, FJ, fruit drinks (100 g/day); 3 day diet record | Concurrent change in BMI (continuous); measured | Adjusted for ethnicity, intake of other beverages, study site and visit, total energy intake, within-individual correlation of repeated measures | Beverage intake was measured at baseline and at most annual follow-up visits (visits 1-5, 7, 8, and 10). Concurrent change in BMI was positively associated with intake of SSSD per 100 g/day (β 0.011 (SE 0.005); P<0.050). BMI was not found to be associated with intake of FJ or fruit drinks (both P≥0.05) | + (SSB), – (FJ) |

**Table 7 (continued)**

| First author (year), country, study name | Population and recruitment | Follow-up (years) | Exposure and assessment method | Outcome and assessment method | Covariates and stratification | Findings* | +/0/–† |
|---|---|---|---|---|---|---|---|
| Vanselow (2009),[78] USA, Project EAT | 2294 adolescents aged 11-15 years, attending 31 ethnically and socioeconomically diverse schools in Minneapolis or St Paul; convenience sampling | 5 | Servings/week of SSSD, fruit drinks, orange juice, apple juice; FFQ | Change in BMI (continuous); self reported | Adjusted for age, physical activity at baseline and follow-up, baseline BMI, cohort, ethnicity, other beverage intake, SES, sex, TV viewing at follow-up | No association found between consumption of SSSD, fruit drinks, orange or apple juice at baseline and BMI at follow-up | 0 |
| Viner (2006),[79] UK, 1970 British Birth Cohort | 4461 adolescents aged 16 years, born in one week in 1970 in England, Northern Ireland, Scotland, and Wales; consecutive sampling | 14 | 0, 1, or ≥2 servings of SSSD on previous day; questionnaire | Change in BMI z score at age 16-30 years (continuous); self reported | Adjusted for baseline BMI z score, height at baseline and follow-up, SES, sex | Compared with people who consumed no SSSD on the day before baseline, those who reported consuming ≥2 servings had a significantly greater change in BMI z score over the 14 year follow-up (β 0.13 (95% CI 0.01 to 0.26); P<0.04) | + |
| Weijs (2011),[80] Holland | 120 infants aged 4-13 months; convenience sampling | 8 | Intake of total sugar and intake of beverage sugar (% of total energy, g/day);<br>2 day diet record | Overweight status at age 8 years (≥1 increased in BMI z score) (OR); self reported | Adjusted for animal protein intake, baseline age, baseline body weight, breastfeeding, SES, sex | In unadjusted analyses, sugar intake at baseline (% of total energy and g/day) and beverage sugar intake at baseline (g/day) did not differ between people not overweight and those overweight at follow-up. However, beverage sugar intake (as % of total energy) differed between the groups (P=0.04). For intake of beverage sugar per 1% of total energy intake, adjusted OR for overweight at age 8 years was 1.13 (95% CI 1.03 to 1.24) | + |
| Welsh (2005),[96] USA, Pediatric Nutrition Surveillance System and Missouri Demonstration Project | 10 904 children aged 2-3 years enrolled in the Special Nutrition Program for Women, Infants, and Children in Missouri; consecutive sampling | 1 | SSB intake (servings/day: 0 to <1, 1 to <2, 2 to <3, ≥3); FFQ | Overweight status at follow-up (BMI ≥95th percentile) (OR); measured | Adjusted for age, birth weight, dietary intake, ethnicity, sex, total energy intake; stratified by being normal or under weight at baseline (BMI <85th percentile), being at risk of overweight at baseline (BMI 85th to <95th percentile), or being overweight at baseline (BMI ≥95th percentile) | Children who were at risk for overweight at baseline and consumed ≥1 SSB/day were 1.8-2.0 times more likely to become overweight than children who consumed <1 SSB/day. Children who were overweight at baseline and consumed ≥1 SSB/day were1.8-2.1 times as likely to remain overweight than children who consumed <1 SSB/day. There was a positive but non-significant relation between SSB consumption and development of overweight in children who were normal or under weight at baseline | + |
| Williams (2008),[93] USA, Healthy Start Project | 519 children aged 3-4 years, attending 1 of 9 selected preschools in upstate New York; convenience sampling | 4 | Sucrose intake (g/day); direct observation and 24 h recall | BMI at 1 year follow-up (continuous); measured | Adjusted for dietary intake at baseline and follow-up, baseline BMI, ethnicity, sex, total energy intake | Sucrose intake at baseline was inversely associated with BMI at follow-up (β –0.10, P<0.05) | — |

ANOVA=analysis of variance; BF=body fat; FFQ=food frequency questionnaire; FJ=100% fruit juice; OR=odds ratio; SD=standard deviation; SE=standard error; SES=socioeconomic status; SSSD=sugar sweetened soft drinks; SSB=sugar sweetened beverages (including cordials, energy drinks, fruit drinks, iced tea, soft drinks); TV=television; WC waist circumference.

*Most adjusted results are reported unless otherwise stated.

†Higher sugar intake positively associated with weight gain (+), not associated with weight gain (0), and negatively associated with weight gain (–).

‡Fat mass index is usually defined as body fat mass (kg)/height ($m^2$); in Johnson et al[98] it is kg/$m^{5.8}$.

# Figures

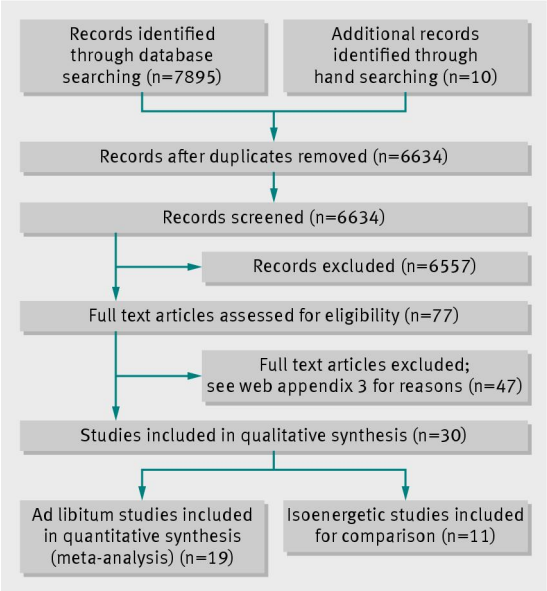



**Fig 1** PRISMA flow diagram for randomised controlled trials

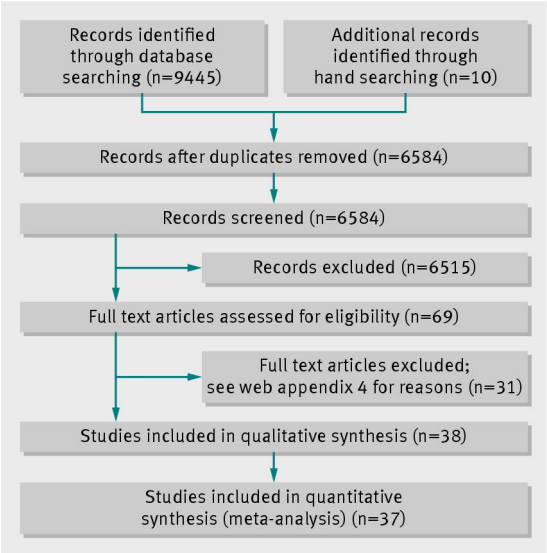



**Fig 2** PRISMA flow diagram for cohort studies

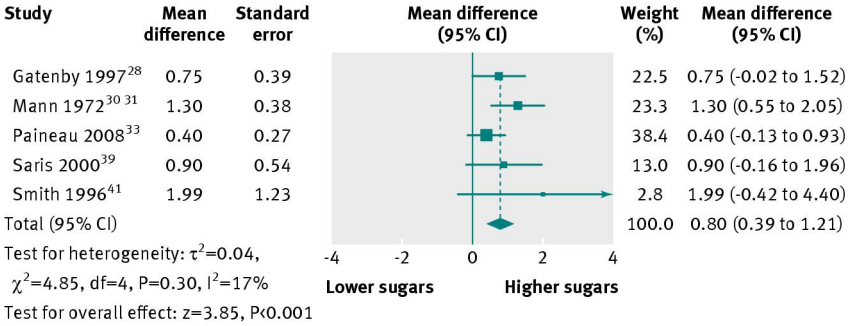



**Fig 3** Effect of reducing intake of free sugars on measures of body fatness in adults. Pooled effects for difference in body weight (kg) shown for studies comparing reduced intakes (lower sugars) with usual or increased intakes (higher sugars). Overall effect shows increased body weight after intervention in the higher sugars groups. Data are expressed as weighted mean difference (95% confidence interval), using generic inverse variance models with random effects

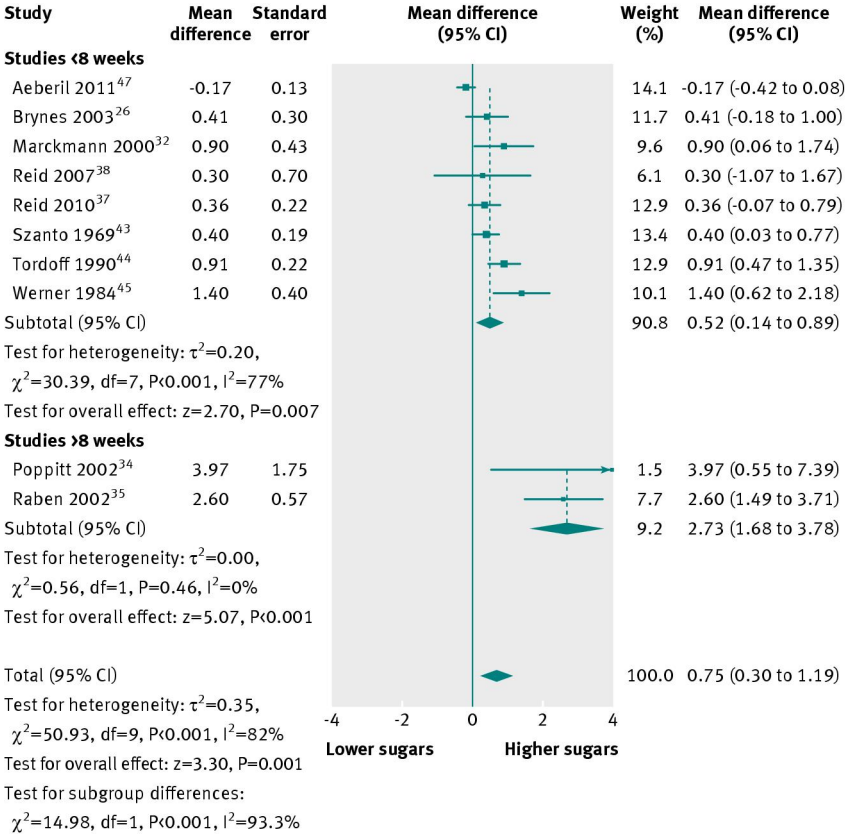
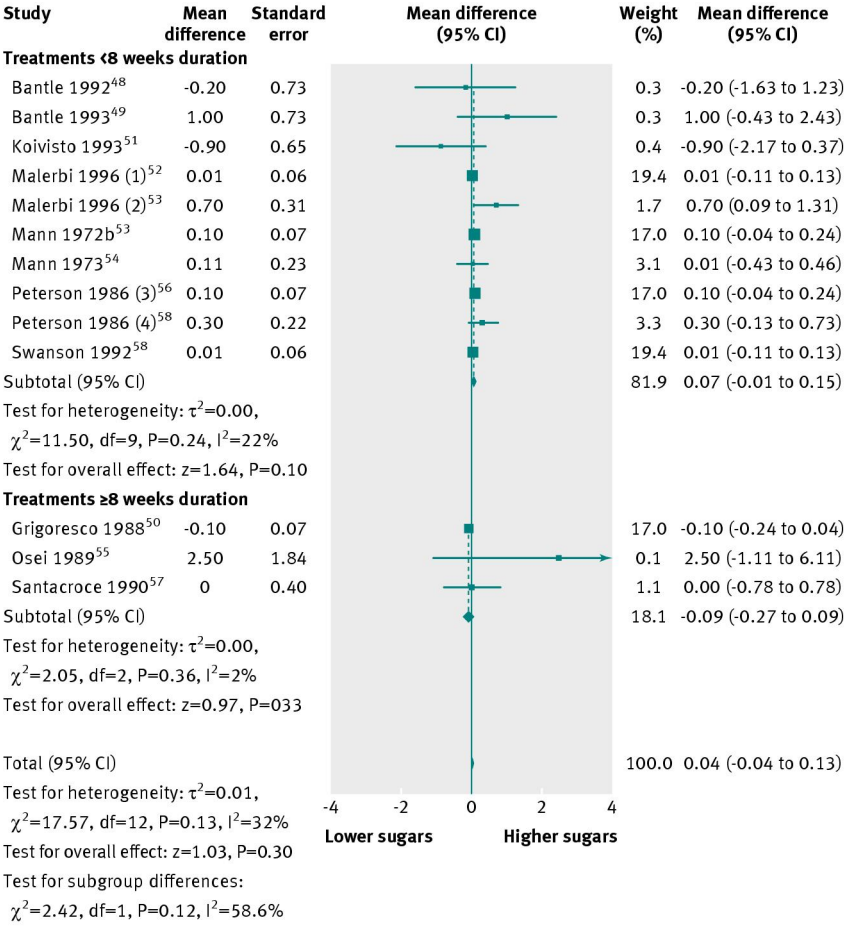



**Fig 4** Effect of increasing free sugars on measures of body fatness in adults. Pooled effects for difference in body weight (kg) shown for studies comparing increased intake (higher sugars) with usual intake (lower sugars). Overall effect shows increased body weight after intervention in the higher sugars groups. Data are expressed as weighted mean difference (95% confidence interval), using generic inverse variance models with random effects




(1) Fructose *v* starch, (2) Sucrose *v* starch, (3) Patients with type 1 diabetes, (4) Patients with type 2 diabetes

**Fig 5** Isoenergetic exchanges of free sugars with other carbohydrates or other macronutrient sources. Pooled effects for difference in body weight (kg) for studies comparing isoenergetic exchange of free sugars (higher sugars) with other carbohydrates (lower sugars). Data are expressed as weighted mean difference (95% confidence interval), using generic inverse variance models with random effects

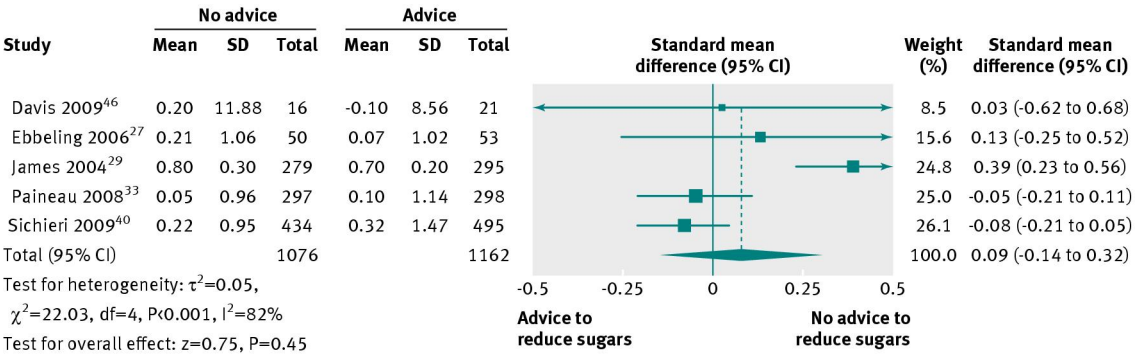
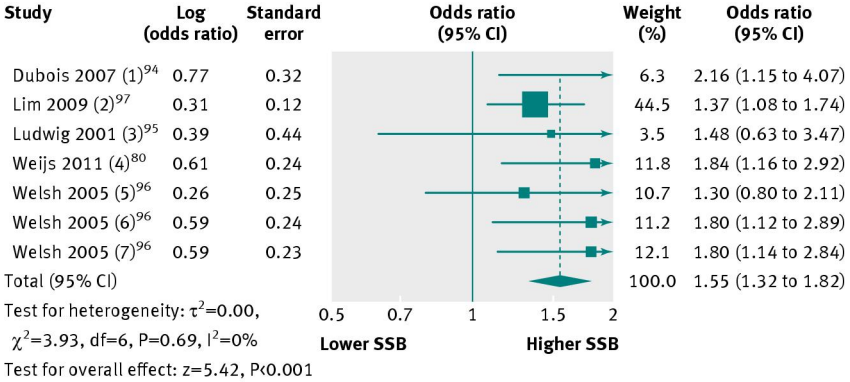



**Fig 6** Effect of reducing free sugars on measures of body fatness in children. Pooled effects for standardised mean difference in body mass index for studies comparing advice to reduce intake of free sugars with no advice regarding free sugars. Data are expressed as weighted, standardised mean difference (95% confidence interval), using generic inverse variance models with random effects




(1) OR for incident obesity in frequent versus infrequent consumers of SSB between meals
(2) OR for incident overweight per daily serve SSB (8 oz)
(3) OR for incident obesity per daily serve SSB
(4) OR for incident overweight per approximate daily serve SSB (5% energy from beverage sugar)
(5) OR for incident overweight in normal weight children who consumed >1 serve/d SSB versus <1 serve SSB/d
(6) OR for remaining overweight in overweight children who consumed >1 serve/d SSB versus <1 serve SSB/d
(7) OR for incident overweight in children at risk of overweight who consumed >1 serve/d SSB versus <1 serve SSB/d

**Fig 7** Association between free sugars intakes and measures of body fatness in children. Pooled estimates for odd ratios for incident overweight or obesity in children consuming one or more servings of sugar sweetened beverages per day at baseline compared with children who consumed none or very little at baseline. Overall estimate shows higher odds of overweight or obesity at follow-up in those who consumed one or more servings of sugar sweetened beverages at baseline. Data are expressed as odds ratio (95% confidence interval), using generic inverse variance models with random effects

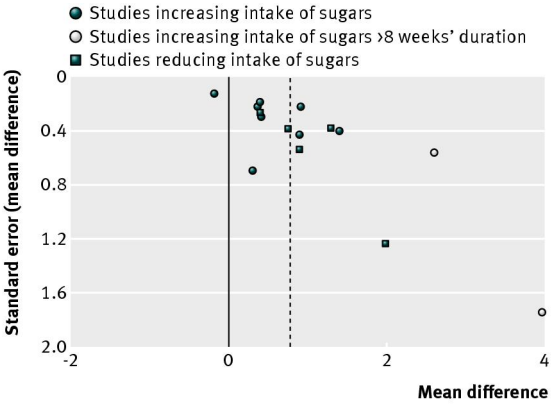



**Fig 8** Funnel plot of randomised ad libitum trials in adults