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Sugar-sweetened soft drinks and obesity: a systematic review of the evidence from observational studies and interventions

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Sugar-sweetened soft drinks (SSD) are a special target of many obesity-prevention strategies, yet critical reviews tend to be more cautious regarding the aetiological role of SSD in promoting excess body weight. Since ongoing evaluation of this issue is important, the present systematic review re-examined the evidence from epidemiological studies and interventions, up to July 2008. Database searches of Medline, Cochrane reviews, Google scholar and a hand search of cross-references identified forty-four original studies (twenty-three cross-sectional, seventeen prospective and four intervention) in adults and children, as well as six reviews. These were critically examined for methodology, results and interpretation. Approximately half the cross-sectional and prospective studies found a statistically significant association between SSD consumption and BMI, weight, adiposity or weight gain in at least one subgroup. The totality of evidence is dominated by American studies where SSD consumption tends to be higher and formulations different. Most studies suggest that the effect of SSD is small except in susceptible individuals or at high levels of intake. Methodological weaknesses mean that many studies cannot detect whether soft drinks or other aspects of diet and lifestyle have contributed to excess body weight. Progress in reaching a definitive conclusion on the role of SSD in obesity is hampered by the paucity of good-quality interventions which reliably monitor diet and lifestyle and adequately report effect sizes. Of the three long-term (>6 months) interventions, one reported a decrease in obesity prevalence but no change in mean BMI and two found a significant impact only among children already overweight at baseline. Of the six reviews, two concluded that the evidence was strong, one that an association was probable, while three described it as inconclusive, equivocal or near zero. Reasons for some discrepancies are presented.

Soft drinks: Sugar: Obesity: Systematic reviews

Introduction

A number of influential global reports assert that sugar-containing drinks play a key role in the aetiology of overweight and obesity[1,2]. However, comprehensive scientific reviews of the evidence base have tended to be more cautious, highlighting the weaknesses of many studies.

Previous reviews have variously described the evidence as 'not conclusive'[3], 'equivocal'[4], 'probable'[2] and 'strong'[5,6]. Most recently, a systematic review and meta-analysis concluded that the strength of relationship was near zero and there was evidence of a publication bias towards studies with a positive result[7].

Summary of previous reviews

The six reviews differ in scope, approach and conclusions. Pereira described the evidence implicating sugar-sweetened drinks in the aetiology of obesity as 'equivocal', criticising the unsatisfactory methodology of many experimental and prospective studies[4]. Bachman *et al.*[3] looked at four proposed mechanisms for the association between sugar-sweetened beverages and obesity, including excess energy intake and poorer satiation from liquids. They judged the evidence strongest for the excess energy intake hypothesis but found it 'not conclusive'[3]. Six of the studies that they included supported the hypothesis but an equal number did not, while those that did had methodological weaknesses such as not controlling for physical activity or using measurements for diet or body weight that had limited reliability and validity.

By contrast, in a systematic review of published studies up to May 2005, Malik *et al.* concluded that the evidence was 'strong' but conceded that research was needed 'to provide more convergence in the data' and 'to elucidate

Abbreviations: NHANES, National Health and Nutrition Examination Survey; SSD, sugar-sweetened soft drinks.

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mechanisms'[5]. Some aspects of this analysis have been criticised, notably the interpretation that the majority of studies show an overall positive association between SSB and obesity[8]. Several studies are reported by Malik *et al.* [5] as positive when only a selected sub-group had a positive result, or classified as 'positive non-significant' where coefficients are near zero and *P* values in excess of 0·2[7]. Furthermore, the results of two studies were confounded by the inclusion of diet soft drinks.

In 2003, the World Cancer Research Fund[2] began to commission a series of systematic literature reviews (SLR) of food and drinks promoting weight gain. The remit was to assess the totality of evidence; however, it was later decided to exclude both cross-sectional studies and longitudinal observational studies with less than 1 year of follow-up or in children aged under 5 years. The SLR on soft drinks was conducted in 2005 and based on a total of six epidemiological studies including a single randomised controlled trial by James *et al.* [9]. Although the expert group charged with conducting the SLR viewed the evidence as 'limited-suggestive', the panel responsible for interpretation concluded that it was 'probable'. The outcome was a recommendation to 'avoid sugary drinks'[2].

In April 2007, Vartanian *et al.* [6] published a wide-ranging systematic review and meta-analysis of the various nutrition and health effects of soft drinks, including forty-five studies with some measure of body weight or obesity as outcome. For all studies combined, the effect size (*r*) was 0·08, which is very small. However, they give more weight to the seven experimental or intervention studies whose effect size was 0·24 (medium).

In contrast to the conclusions reached by Malik *et al.* [5], Vartanian *et al.* [6] and the World Cancer Research Fund report[2], the latest meta-analysis, by Forshee *et al.* [7] concluded that the association between BMI and consumption of sugar-sweetened beverages in children was near zero. The authors also found evidence of a positive publication bias. The review of Forshee *et al.* [7] included papers published up to October 2006.

Owing to the frequency with which new studies are being published, ongoing review is imperative. This paper presents the results of a comprehensive review of the literature up to July 2008 regarding the association between sugar-containing drinks and body weight and obesity. It does not attempt to cover the literature pertaining to mechanisms or experimental investigations of sugar-sweetened soft drinks' links with energy intake and satiety. In evaluating the strengths and weaknesses of the evidence base, possible reasons for discrepant findings in reviews are highlighted and some issues needing to be addressed in future studies are identified.

Methods

Papers were identified from web-based searches in PubMed, Google Scholar and the Cochrane Library, supplemented by a hand-search of existing documentation and cross-references. Inclusion criteria were studies and reviews published in English up to July 2008 that related to consumption of sugar-containing drinks (SSD) and their association with body weight, BMI or adiposity in adults or children. The search was conducted iteratively using search terms: 'soft drinks'/'sugar-sweetened beverages'/'-soda'/'liquid sugars' with 'weight'/'body weight'/'obesity'/'adiposity'. Abstracts were screened for relevance and if they appeared to meet inclusion criteria were imported electronically into a bibliographic database (Endnote version 8; Adept Scientific, Letchworth Garden City, Herts, UK). Full papers were then obtained from the web or requested from authors.

To represent the totality of evidence relating to consumption of soft drinks in a non-laboratory setting, all designs were included (cross-sectional, prospective, and interventions and randomised controlled trial). SSD were defined as all cold beverages containing added sugars, whether carbonated or still, including soda pop (but not diet soda) and fruit squash and drinks with a fruit component less than 100 % pure fruit juice, hot beverages and diet drinks were not included, although studies were included if they assessed these as well as SSD. Studies were excluded if they were animal studies, not published in English, gave no data on consumption of soft drinks or sweetened beverages, provided no anthropometric data, or were short-term experiments or mechanistic studies. Four studies were discarded at this stage because they did not disaggregate SSD data from other sources of sugar[10,11], other snack foods[12] or other interventions[13]. Essential details of studies meeting the criteria were extracted into a spreadsheet and classified by design (cross-sectional, longitudinal or intervention study). Study quality was not formally assessed because this was considered too subjective and there is a lack of consensus on criteria for different study designs. Instead the strengths and weaknesses of papers are discussed where these have a direct bearing on the results. Tables 1–5 summarise these studies and retain the terminology used by the papers (for example, SSD, sweetened beverages, soda, fruit drinks).

Results

Of a total of forty-four original studies, forty were observational (twenty-three cross-sectional and seventeen prospective) and four were interventions. Three of the prospective studies also provided cross-sectional data at baseline[14–16].

Cross-sectional studies

Results were equivocal, with less than half of the studies with cross-sectional data (*n* 12/27) showing a significant positive association between SSD and BMI or overweight in at least one group (Table 1). Three American prospective studies showed an association at baseline, although in two of these[14,15], results were only significant among females. Several studies using US National Health and Nutrition Examination Survey (NHANES) data were based on the 24 h recall method for assessing diet, while the large study by Berkey *et al.* [15] used a semi-quantitative FFQ to assess diet over the previous year. The UK and Irish studies used the 'gold standard' 7 d weighed dietary record [17,18].

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Table 1. Cross-sectional studies showing a positive association between sugar-sweetened soft drinks (SSD) consumption and BMI or obesity

					Body weight, BMI or BMI *z*-score			
Study	Country	Age group (years)	Subjects (*n*)	Diet method	Positive association ($P < 0{\cdot}05$)	Positive but NS	No association or negative	Results
McCarthy *et al.* (2006)[18]	North and South Ireland	18–64	1379	7 d estimated food diary	'High energy beverages'			Many foods associated with higher BMI. Consuming high- *v.* low-energy beverages: OR of obesity 3·9
Gibson & Neate (2007)[17]	UK	4–18	1688	7 d weighed record	High SSD (quintile 5)	SSD (overall)		Only top quintile SSD (mean 870 kJ/d) associated with overweight (OR 1·67; $P = 0{\cdot}03$ *v.* quintile 1). Other sources of energy and physical activity showed stronger associations with BMI
Liebman *et al.* (2003)[26]	USA	18–99	1827	24 h	Soda			Did not distinguish between regular and diet soda. Contrast >one soda pop/week *v.* < one/week ($P < 0{\cdot}05$): 32 *v.* 20 % obese in women; 27 *v.* 17 % in men over 50 years
Warner *et al.* (2006)[25]	USA (Mexican-Americans)	2	385	FFQ past 7 d (twenty items)	Soda			One or more serving of soda *v.* none associated with odds of obesity of 3·39 ($P = 0{\cdot}02$) (soda did not include fruit drinks)
French *et al.* (1994) (prospective study with additional cross-sectional analysis)[14]	USA	Adults	3552	FFQ	Soda (women)	Soda (men)		Women consuming one serving/week were 0·47 lbs heavier than non-consumers ($P = 0{\cdot}03$). Men were 0·33 lbs heavier ($P = 0{\cdot}13$)
Novotny *et al.* (2004)[23]	Hawaii	9–14	323	3 d semi-weighed record	Soda			Body weight difference equivalent to 1·7 kg per 350 ml can of soda) in multiple regression ($P = 0{\cdot}01$). Did not include fruit drinks
Ariza *et al.* (2004)[24]	USA (Hispanic)	5–6	80	Interview questioned frequency of SSD	SSD			Overweight children were more likely to consume sweetened beverages (powdered drinks, soda pop) daily (67 *v.* 39 %; $P = 0{\cdot}03$)
Gillis & Bar-Or (2003)[22]	Canada	4–16	181	24 h + FFQ	SSD			Obese consumed more soda/SSD (seven *v.* five servings/week; $P < 0{\cdot}05$ for both sexes combined but NS in girls)
Ludwig *et al.* (2001) (prospective study with additional cross-sectional analysis)[16]	USA	12	548	Youth FFQ (past month)	SSD			Baseline SSD servings positively associated with change in BMI (mean 0·18 kg/m^2 per serving; $P = 0{\cdot}02$)
Berkey *et al.* (2004) (prospective study with additional cross-sectional analysis)[15]	USA	9–14	16 679	FFQ semi-quantitative. Typical intake over past year and change	SSD (girls)			Baseline BMI +0·06 kg/m^2 per serving of SSD in girls ($P = 0{\cdot}04$). SSD associated with higher total energy intakes (216 kcal/serving)
Troiano *et al.* (2000)[19]	USA	2–19	10 371	24 h recall	SSD energy			Except for females aged 12–19 years, overweight children in all age groups had soft drink intakes about 2 % higher (as a percentage of energy) than normal-weight children
Nicklas *et al.* (2003)[20]	USA	10	1562	24 h recall	Sweetened beverages ($P < 0{\cdot}001$)			Consumption of sweetened beverages (58 % soft drinks, 20 % fruit-flavour drinks, 19 % tea and 3 % coffee) was positively associated with overweight status ($P < 0{\cdot}001$) (OR 1·33)

Table 2. Cross-sectional studies showing a non-significant or null association between sugar-sweetened soft drinks (SSD) consumption and BMI or obesity

Study	Country	Age group (years)	Subjects (*n*)	Diet method	Body weight, BMI or BMI *z*-score: Positive association ($P < 0·05$)	Body weight, BMI or BMI *z*-score: Positive but NS	Body weight, BMI or BMI *z*-score: No association or negative	Results
Andersen *et al.* (2005)[35]	Norway	8–14	3139	4 d food diary			SSD	No association between overweight status and i ntake of sweetened soft drinks or % energy from sugars. Inverse association with sweets
Bandini *et al.* (1999)[34]	USA	12–18	43				Soda	No difference in percentage of energy from soda between obese (5·9 %) and non-obese (6·0 %)
Forshee & Storey (2003)[30]	USA	6–19	3311	CSFII (2 × 24 h)		Diet soda (girls)	Soda	No association with regular soda ($P > 0·2$); slight positive association with diet soda in girls
Forshee *et al.* (2004)[31]	USA	12–16	2216	24 h + FFQ (NHANES III)			Soda and fruit ades	SSD (soda) serving of 370 g associated with +0·26 kg/m^2 BMI in girls and 0·11 kg/m^2 in boys; NS ($P > 0·3$).
Giammattei *et al.* (2003)[27]	USA	11–13	385	Self-administered lifestyle and habits questionnaire	Diet soda	SSD		NS positive correlation for regular soda (r 0·10; $P = 0·08$). Positive association with diet soda (r 0·19; $P = 0·01$)
Gibson (1998)[36]	UK	1–4	1546	4 d weighed record			SSD	No association between BMI and amount or proportion of energy from soft drinks ($P > 0·50$)
Janssen *et al.* (2005)[28]	Thirty-four countries	10–16	137 000	FFQ			SSD	Association with SSD NS in thirty countries, positive in two, negative in two
O'Connor *et al.* (2006)[32]	USA	2–5	1160	24 h			SSD	No association with the amount of total beverages, milk, 100 % fruit juice, fruit drink, or soda consumed
Rajeshwari *et al.* (2005)[21]	USA	10	1548	24 h			SSD	No linear relationship between sweetened-beverage consumption and BMI. BMI increased from 1970 s to 1990 s across all SSD consumption groups. Energy did not
Rodriguez-Artalejo *et al.* (2003)[37]	Spain	6–7	1112	FFQ			SSD	No association between SSD and BMI
Roseman *et al.* (2007)[33]	USA	11–14	4049	7 d recall			Soft drinks	No significant association between soft drink consumption and students' weight status. Definition not given. No *P* values
Silveira *et al.* (2006)[38]	Brazil	14–19	172	4 d diary, FFQ, lifestyle questionnaire			SSD	Daily soft drink consumption was not associated with overweight (OR 0·69 (95 % CI 0·27, 1·72); $P = 0·377$; 13 % of cases *v.* 18 % of controls)
Sun & Empie (2007)[40]	USA	20–74	38 000	Combined CSFII and NHANES			SSD	No substantive differences in BMI and obesity occurrence between frequent and infrequent users of SSD. Only in NHANES 1999–2002, obese adults consumed 72 g/d more (31 kcal) than the mean for non-obese adults
Veugelers & Fitzgerald (2005)[39]	Canada	10–11	4298	FFQ; soft drink sales in school			SSD	Availability of soft drinks at schools was not associated with risk of overweight (OR 0·99), although consumption was higher in schools that sold soft drinks (4·0 *v.* 3·6 cans per week; $P = 0·01$)

CSFII, Continuing Survey of Food Intake by Individuals; NHANES, National Health and Nutrition Examination Survey.

Table 3. Longitudinal studies showing a positive association between sugar-sweetened soft drinks (SSD) consumption and BMI or obesity

Study	Country	Age group (years)	Subjects (*n*)	Follow-up	Diet method	Body weight			Results
						Positive association ($P < 0.05$)	Positive but NS	No association or negative	
Nooyens *et al.* (2005)[43]	Netherlands	50–65, Men	288	5 years	FFQ (EPIC)	SSD			Unadjusted model: change in BMI = 0.2 kg/m^2 per year per serving per d; waist 0.16 cm/year per serving per d (in adjusted model BMI change = 0.12 kg/year per serving per d; waist 0.06 cm/year)
Welsh *et al.* (2005)[42]	USA	2 and 3	10 904	1 year	FFQ	SSD (overweight)	SSD (normal weight)		Positive association between SSD and overweight only in children overweight (>85th percentile BMI) at baseline. OR about 2 for > 1 drink/d *v.* < 1 drink/d (referent)
Berkey *et al.* (2004)[15]	USA	9–14	>12 000	2 years	FFQ	SSD (boys)	SSD (girls)		Consumption of sugar-added beverages was associated with small BMI gains during the corresponding year (boys: +0.03 kg/m^2 per daily serving, $P = 0.04$; girls: +0.02 kg/m^2, $P = 0.096$). Boys who increased consumption of sugar-added beverages from the prior year experienced weight gain (+0.04 kg/m^2 per additional daily serving; $P = 0.01$)
Bes-Rastrollo *et al.* (2006)[41]	Spain	Adults	7194	2 years	FFQ	SSD (in previous weight-gainers)		SSD (in weight-stable)	Among participants who had gained >3 kg in the 5 years before baseline, adjusted odds of subsequent weight gain in quintile 5 *v.* quintile 1 of sugar-sweetened soft drink consumption was 1.6 ($P = 0.02$)
Ludwig *et al.* (2001)[16]	USA	12	548	19 months	Youth FFQ (past month)	SSD			BMI increased 0.24 kg/m^2 per serving ($P = 0.03$) (adjusted for baseline BMI, demographic, diet and lifestyle). Frequency of obesity based on thirty-seven cases of incident obesity (OR 1.60 (95 % CI 1.14, 2.24); $P = 0.02$)
Phillips *et al.* (2004)[46]	USA	8–12, Girls	196 enrolled (178 after exclusion of 18)	7 years	FFQ (past year)	SSD			Percentage energy from soda associated with higher BMI *z*-score (quartile 3 (>1.5 %) and quartile 4 (>3.2 %) had BMI *z*-score +0.17 compared with quartile 1)
Schulze *et al.* (2004)[47]	USA	Adult women	51 000	8 years	–	SSD			Weight gain in 1007 women increasing SSD consumption from <1/week to >1/d was about 3 kg (over 4 years) more than in those 1020 women who decreased SSD by the same amount from 1995 to 1999. Weight gain also about 3 kg in those who were consistent high or low consumers
Striegel-Moore *et al.* (2006)[44]	USA	9–10, Girls	3371	9 years	3 d records annually	SSD			Of all beverages, increasing soda consumption predicted the greatest increase of BMI

EPIC, European Prospective Investigation into Cancer and Nutrition.

Table 4. Longitudinal studies showing a non-significant or null association between sugar-sweetened soft drinks (SSD) consumption and BMI or obesity

Study	Country	Age group (years)	Subjects (*n*)	Follow-up	Diet method	Body weight			Results
						Positive association ($P < 0{\cdot}05$)	Positive but NS	No association or negative	
Libuda *et al.* (2007)[48]	Germany	9–18	244	5 years	3 d WDR		SSD (girls)	SSD (boys)	SSD had weaker association than fruit juice. Change in total 'energetic beverages' was significantly associated with change in BMI but not percentage body fat. Girls: change = 0·055 BMI standard deviations/MJ SSD ($P = 0{\cdot}08$)
Johnson *et al.* (2007)[52]	UK	5–7, 9	1203	2 years	3 d NWDR at age 5 and 7 years	Diet soft drinks*		SSD*	No association between SSD intake at age 5 or 7 years and BMI at age 9 years. Inverse association between SSD and fat mass. Small positive association with low-energy drinks
Blum *et al.* (2005)[53]	USA	Elementary school children	166	2 years	24 h recall			SSD	No association between SSD and year 2 BMI *z*-score ($\beta = -0{\cdot}003$, se 0·004; $P > 0{\cdot}05$). Only diet drinks positively associated with BMI
Kvaavik *et al.* (2005)[55]	Norway	15, 33	485	8 years	Questionnaires			SSD	No differences in BMI, overweight or obesity in 1999 between long-term high and low consumers of SSD
Mundt *et al.* (2006)[51]	Canada	8–19	208	5 years	24 h recall repeated			SSD*	SSD was not related to fat mass development or physical activity
Newby *et al.* (2004)[54]	USA	2–5	1345	6–12 months				SSD	No association between any type of beverage (soda, diet soda, juice, milk) and change in BMI in multivariate adjusted models
Schulz *et al.* (2002)[50]	Germany (EPIC)	Adults	17 369	2 years	FFQ			Soft drinks combined	Soft drinks included water and juice. Mixed results for men (higher 'soft drink' intake at baseline was very weakly associated with both losses and gains) and weakly negative association for women (slightly greater risk of large weight losses; OR 1·02 (95 % CI 1·00, 1·03))
French *et al.* (1994)[14]	USA	Adults	3552	2 years	FFQ		Soda ($P > 0{\cdot}2$)		Small NS association between soda intake and change in weight over 2 years per serving (12 oz) (women 0·08 lbs ($P = 0{\cdot}39$); men 0·11 lbs ($P = 0{\cdot}22$))
Mrdjenovic & Levitsky (2003)[49]		6–13	30 (final sample 21)	4–8 weeks			SSD (>16 oz)		No significant difference in weight gain in high SSD (>16 oz/d) *v.* lower SSD (6–16 oz) (1·12 *v.* 0·32–0·48 kg; $P = 0{\cdot}4$)

WDR, weighed dietary records; NWDR, non-weighed dietary records; EPIC, European Prospective Investigation into Cancer and Nutrition.
* Fat mass.

Table 5. Intervention studies of sugar-sweetened soft drinks (SSD) consumption and BMI or obesity

Study	Country	Age (years)	Subjects (*n*)	Intervention and duration	Diet method	Body weight: Positive association ($P < 0·05$)	Body weight: Positive but NS	Body weight: NS or negative	Results
Sichieri *et al.* (2008)[57]	Brazil	9–12	1140	School-based intervention to reduce consumption of SSD over 1 year	24 h recall at baseline and follow-up	Overweight girls (*n* about < 80) (unadjusted model: BMI −0·01; $P = 0·009$)	Boys and girls overweight at baseline (adjusted for age: BMI −0·007; $P = 0·11$)	Total sample	SSD consumption reduced by 69 ml/d in intervention *v.* 13 ml in controls (difference 56 ml; $P = 0·03$). No significant difference in BMI (both groups gained). Unadjusted BMI in girls overweight at baseline (15 %) −0·4 kg/m^2 in intervention *v.* −0·2 kg/m^2 in controls ($P = 0·009$). Adjusted model: BMI −0·007 ($P = 0·11$)
Ebbeling *et al.* (2006)[56]	USA	13–18	113	Home delivery of diet soft drinks to replace normal consumption for 6 months	2 × 24 h diet recall and activity diary at baseline and follow-up	SSB (high baseline BMI)		SSB (overall)	82 % reduction in SSB in intervention group, no change in controls (250 kcal energy difference). BMI decreased on average by 0·26 kg/m^2 for every serving per d of SSB that was displaced. For top tertile BMI at baseline, BMI change = −0·75 kg/m^2 ($P = 0·03$). Change in total group BMI −0·14 units (NS)
James *et al.* (2004)[9]	UK	7–11	644	School-based lessons (Ditch the Fizz) given five times over 1 year	Beverage diaries (non-validated)	Soda (all types)			Soda consumption lowered in intervention group by 0·7 servings over 3 d. Prevalence of obesity was 7·7 % lower in intervention group but no difference in mean BMI
Raben *et al.* (2002)[60]	Denmark	Adults	41 (21 intervention, 20 controls)	10 weeks of high-sucrose diet (> 60 % from SSD) or aspartame-containing drinks	7 d records and 7 d diaries at 0, 5 and 10 weeks, eating behaviour questionnaire at week 10	High-sugar diet (28 % sucrose, of which drinks provided about 60 %)			High-sugar diet (28 % energy from sucrose) provided an extra 3·4 MJ energy but there was some compensation (1·6 MJ difference in total energy). Body-weight gain in sucrose group = 1·6 kg, controls lost 1 kg. Weight gain in intervention group was half the predicted amount based on energy intake

Studies showing a positive association with BMI in children. The majority of studies on children and youth derive from the USA, where consumption of SSD is typically about twice that of UK and Europe (8–10 % of energy *v.* 4–5 %). The largest of these was the prospective study by Berkey *et al.* of 16 679 children, which found a significant association between baseline SSD consumption and BMI in girls and a non-significant association among boys[15]. The prospective study of Ludwig *et al.* also found an association between baseline consumption of sugary drinks and change in BMI over 19 months (mean 0·18 units for each daily serving (95 % CI 0·09, 0·27); $P = 0·02$)[16]. Troiano *et al.* [19] using USA NHANES data spanning the 20 years up to 1994 reported a higher consumption of energy from soft drinks among overweight youth (2–19 years) compared with non-overweight youth in each age group. However, lack of evidence of an increase in energy intake suggested that physical inactivity was a major factor[19]. In the Bogalusa study of 10-year-olds in Louisiana (*n* 1594) Nicklas *et al.* reported that consumption of sweetened beverages (a definition which included tea and coffee) was associated with risk of overweight, but so were numerous other eating patterns[20]. They noted that the percentage of variance explained by any of the eating patterns was very small and results varied by ethnic group and sex. A subsequent trend analysis of this same dataset concluded that there was no linear association between BMI and SSD because BMI increased from the 1970 s to the 1990 s across all SSD consumption groups[21]. In a small case–control study of 181 Canadian children (ninety obese and ninety-one matched controls), obese children reported a higher frequency of consuming regular soda (seven *v.* five times per week; $P < 0·05$) but also consumed more energy, fat, meat, chips and grains as well as more food away from home[22]. However, one criticism of this study is that the non-obese controls were self-selected volunteers who may have been from more health-conscious families.

Positive associations of SSD with weight status may be more likely in populations with high intakes, including some ethnic groups. A Hawaiian study among 9- to 14-year-olds (*n* 323) who were high consumers of soda (750 g/d) found each serving to be associated with an additional 1·7 kg body weight ($P = 0·01$) while milk was inversely associated with weight[23]. This study used a 3 d record of diet and adjusted for all main confounders such as puberty, physical activity, energy intake and ethnicity. Evidence from two other studies is weaker. In a pilot study among Hispanic 5- to 6-year-olds[24] the overweight children were more likely to consume sweetened beverages daily (67 *v.* 39 %; $P = 0·03$) but only eighty out of 250 mothers completed an interview and few other components of diet were assessed. Among Mexican-American infants whose mothers were recruited in pregnancy, the adjusted odds of being overweight at 2 years were three times higher for those who consumed soda daily compared with none[25]. One weakness of this study is that fruit drinks, which are popular in this young age group, were not assessed.

Last, an analysis of UK data from a national survey of young people aged 4–18 years found a weak association between the risk of overweight and SSD consumption that only reached statistical significance for the top quintile (mean 870 kJ/d from SSD; OR 1·67 (95 % CI 1·04, 2·66); $P = 0·03$)[17]. However, other sources of energy intake and physical activity had a stronger impact in regression models. Importantly, this study also incorporated adjustments for under-reporting and dieting.

Studies showing a positive association with BMI in adults. There were three studies in adult populations showing a significant positive association between SSD and obesity. Like some studies in children, many of these suggest that the association is more complex, with clustering of lifestyle and environmental factors in certain groups. In the largest study, a prospective investigation of 3552 American adults in the Healthy Worker Project, women consuming one serving per week of soda were 0·21 kg heavier at baseline than non-consumers ($P = 0·03$), but men consuming one serving *v.* none were not significantly heavier (0·15 kg; $P = 0·13$)[14]. In a study of rural communities in the USA, Liebman *et al.* [26] found that subjects who drank one or more servings of soda pop per week were more likely to be overweight or obese than those who drank less, while other sweetened beverages such as Kool Aid and fruit juices showed no relation with overweight. However, they also found that overweight or obese adults were more likely to order supersize portions, watch TV and to eat while doing other activities[26]. Second, as this study did not distinguish between regular and diet versions of soda, there may be some overestimation due to reverse causality (diet drinks being chosen by overweight people). Among Irish adults, McCarthy *et al.* found that consuming high-energy beverages rather than low-energy beverages was associated with a higher likelihood of being obese but so was consuming larger portions of many foods, such as chips, savouries and fat spreads[18]. Importantly, this study was controlled for a number of different confounders including energy intake:BMR, age, sex, education and total food consumed.

Studies showing a non-significant positive association with BMI. One small study of 11- to 13-year-olds in California showed a weak positive association between SSD and BMI that failed to reach significance ($P = 0·08$)[27]. The review of Malik *et al.* [5] classifies this study as 'significant positive'. However, this is based on results for diet and regular soda combined, although it is evident that diet soda drives the positive association (r 0·19; $P < 0·001$), whereas the association with regular soda was non-significant (r 0·10; $P = 0·08$) (Table 2).

Studies showing no association with BMI. Thirteen studies showed no association between SSD and BMI, of which twelve were in children (Table 2). The largest was a synthesis of data from thirty-four European countries covering more than 137 000 school-aged children[28]. The authors reported no association overall between overweight status and soft drinks; OR in adjusted logistic regressions were null (not significantly different from 1) in thirty studies, positive (>1) in two and negative (<1) in two. Most of the studies used a FFQ and assessed overweight from reported BMI using age- and sex-specific cut-offs[29]. A further eleven studies in children, including six from the

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USA[30–34] and five from other countries (Norway[35], UK[36], Spain[37], Brazil[38] and Canada[39]), likewise report no association between SSD and overweight. Four of these were new studies not included in previous reviews. Interestingly, both of the studies by Forshee and colleagues[30,31] are classified by Malik *et al.* as suggestive positive[5]. However, in the first study of 3311 young people aged 6–19 years old participating in the Continuing Survey of Food Intake by Individuals (CSFII), there was a positive association with diet soda but not regular soda ($P > 0{\cdot}2$)[30], while in the second, an analysis of NHANES III using two diet methods, the interpretation of Malik *et al.* [5] is based on results for the 24 h recall (slightly positive but $P > 0{\cdot}36$) whereas the FFQ data gave results that were slightly negative. In any event both were described by the original author as near zero[31].

In agreement with this conclusion, a summary analysis has recently been published of NHANES and CSFII data ($n > 34\,000$). This found 'no substantive differences in BMI and obesity occurrence between frequent and infrequent users of SSD'[40]. Thus the majority of cross-sectional studies do not support a positive association of BMI with SSD.

Longitudinal studies

Of the seventeen longitudinal studies included, half (eight) showed a significant positive association between SSD consumption and weight or weight gain in at least one subgroup (Table 3). However, four of these showed results which were non-significant in another subgroup[15,41,42], or lost significance on multivariate adjustment for confounders[43].

Longitudinal studies showing a positive association with BMI in children. Five studies were in children (including one in preschool children). In the largest, of more than 12 000 children aged 9–14 years old followed up for 2 years[15], consumption of SSD was associated with small (self-reported) BMI gains during the corresponding year, although this was statistically significant only among boys (boys: $+0{\cdot}03$ kg/m^2 per daily serving, $P = 0{\cdot}04$; girls: $+0{\cdot}02$ kg/m^2, $P = 0{\cdot}096$). Boys who increased consumption of sugar-sweetened beverages from the prior year experienced weight gain ($+0{\cdot}04$ kg/m^2 per additional daily serving; $P = 0{\cdot}01$). The authors acknowledge that the magnitude of the actual differences was modest. Thus a boy consuming three servings per d over 10 years was predicted to gain 0·9 BMI units more than if he consumed none.

In another large American study, among young preschool children from low-income households, consumption of one or more sugar-containing drinks per d (*v.* none) was associated with increased risk (OR about 2) of being overweight 1 year later[42]. However, this was only statistically significant among children heavier at baseline (BMI > 85th percentile). One weakness of this study is that neither parental obesity nor breast-feeding was adjusted for in regressions although both are highly important risk factors for overweight in this age group.

The prospective study by Ludwig *et al.* [16], although small, has shown the strongest association so far and been particularly influential in driving public policy. Among 548 children (12 years old) in the Planet Health project they reported a significant association between weight gain and SSD consumption (both at baseline and over time), estimating that a single serving was associated with a gain in BMI of 0·24 kg/m^2 over 19 months ($P = 0{\cdot}03$) and a 60 % increase in odds of obesity[16]. The estimates were adjusted for baseline BMI, demographics, diet and lifestyle. The odds of obesity, however, were based on only thirty-seven cases of incident obesity. Although otherwise of good quality, this study has been criticised for using raw BMI rather than BMI *z*-score, which is the preferred measure in children, and for not assessing drinks intake in those who decreased their BMI[4].

Two further American studies following girls though puberty found that soda consumption significantly predicted increase in BMI. The largest (n 3371) using the well-respected National Heart, Lung, and Blood Institute Growth and Health Study employing 3 d food diaries repeated annually, found that, of all beverages, increasing soda consumption predicted the greatest increase of BMI[44]. However, the investigators did not adjust for physical activity, which has been shown to be a major predictor of weight gain in this cohort[45]. The smaller study by Phillips *et al.* used the FFQ to study 178 girls for 7 years and found that the percentage of energy from soda was associated with higher BMI *z*-score, though not with higher body fat[46].

Longitudinal studies showing a positive association with BMI in adults. The largest study included more than 51 000 women nurses[47]. Weight gain over a 4-year period was highest among women who increased their SSD consumption (from one or fewer drinks per week to seven or more per week) and smallest among women who decreased their intake just as dramatically (difference about 3 kg over 4 years). Interestingly, those with stable consumption patterns (either low or high) did not differ in weight gain (which was also about 3 kg).

Similarly, a Mediterranean cohort study in 7194 men and women followed over 28 months found a subgroup effect, with a significant positive association with weight gain only among adults who had been gaining > 3 kg weight in the 5 years before the study. The adjusted OR for weight gain in the top quintile *v.* lowest quintile of SSD was 1·6 (95 % CI 1·2, 2·1; *P* for trend = 0·02). This association was absent in the participants who had not gained weight in the 5-year period before baseline[41].

Last, a small Dutch study of 288 middle-aged men studied for 5 years found that one serving of SSD daily was associated with a BMI gain of 0·2 kg/m^2 per year, although this reduced to 0·12 kg/m^2 in the multivariate adjusted model ($P > 0{\cdot}05$)[43]. By comparison the weight gain associated with retiring from an active job was 0·42 kg/m^2.

Longitudinal studies showing a non-significant positive association with BMI in children or adults. Six studies reported a non-significant positive result in at least one group. These include the result among girls in the study by Berkey *et al.* [15] ($P = 0{\cdot}096$), the result among normal-weight preschoolers in the study by Welsh *et al.* [42] and that using the final adjusted model in the Dutch study[43]. In addition, the study by French *et al.*, which had found a

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small positive association among women at baseline, failed to find this prospectively ($P = 0{\cdot}39$ for women; $P = 0{\cdot}22$ for men)[14] (Table 4). Although Malik *et al.* [5] classified this study as positive but non-significant, the coefficients were virtually zero (one 12 oz serving (per week) was associated with a weight change of a mere 40 g for women or 55 g for men over 2 years).

A recently published study among 244 adolescents (Dortmund Nutritional and Anthropometric Longitudinally Designed (DONALD) cohort), although small, is noteworthy for its strong design (3 d weighed dietary record, measured BMI and 5-year follow-up) and robust statistical analysis (repeated measures, change in BMI on change in intake)[48]. Results showed no association with change in BMI *z*-score, either cross-sectionally or prospectively among boys, but a weak and non-significant association among girls ($P = 0{\cdot}08$), equivalent to an increment of 0·055 BMI SD over 5 years per MJ of SSD (per d). There was a stronger association with fruit juice consumption than with SSD with the result that the combination 'energetic beverages' was statistically significant in girls ($P = 0{\cdot}01$). Finally, one very small study of thirty children followed for 4–8 weeks at summer camp found that those consuming large amounts of SSD (>16 oz/d) gained more weight (1·12 kg) than those who consumed less (0·32–0·48 kg)[49], but the study had insufficient power ($P = 0{\cdot}4$) and there were other methodological weaknesses, such as recorded weights for only twenty-one children.

Longitudinal studies showing no association with BMI. In the largest study, German adults in the European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam cohort (*n* 17 369) were followed up over 2 years to assess which food groups were associated with weight gain or weight loss, compared with stable weight[50] (Table 4). 'Soft drinks' included all types including water, SSD, diet drinks and fruit juice. The authors used polytomous logistic regression to examine factors associated with weight gain or weight loss and models were adjusted for a large number of confounders, including physical activity, dieting and baseline BMI. Results were inconclusive for men (higher 'soft drink' intake at baseline was very weakly associated with both losses and gains) and weakly negative for women (slightly greater of risk of large weight losses (OR 1·02; 95 % CI 1·00, 1·03).

Three recent longitudinal studies not included in previous reviews are worth special notice because they assessed fatness, not merely weight or BMI. No association with percentage body fat was found in the DONALD study[48] mentioned above, but the authors suggest that physical activity and timing of pubertal changes may have masked this. The second study, by Mundt *et al.*, tracked body fatness among normal Canadian children and adolescents and found no relationship between fat mass development and consumption of sugar drinks[51]. Models included physical activity and adjusted for physical maturation, which is important because soft drink consumption and fat mass both tend to rise with age. Finally, a cohort study of 1203 children in Avon, UK (Avon Longitudinal Study of Parents and Children (ALSPAC) cohort) did not find any significant association between consumption of sugared soft drinks at age 5 or 7 years and fatness at age 9 years[52]. This study employed a reliable dietary method and examined body fatness (by dual-energy X-ray absorptiometry) as well as body weight. There was a small positive association with consumption of low energy soft drinks, which were possibly being used as a weight-reduction strategy. A similar positive association with diet drinks but not SSD was found in a study of 166 elementary school children studied for 2 years by Blum *et al.* [53].

Among 1345 preschool children aged 2–5 years assessed over 6–12 months in Dakota, USA, Newby *et al.* [54] found no association between change in BMI and consumption of any types of drinks (fruit juice, fruit drinks, milk, soda and diet soda) in multivariate models. Intakes of fruit juice and milk were high in this sample but low intakes and limited variation of soda and fruit drinks in this study may have limited the ability to detect an association with BMI or weight[54].

Finally, in a Norwegian study of 485 older adolescents tracked into adulthood, Kvaavik *et al.* found no difference in BMI, overweight or obesity at age 33 years between those who were long-term (previous 8 years) high consumers or low consumers of SSD[55]. Long-term high SSD consumption was also associated with lifestyle differences including less physical activity, more smoking and higher energy intakes that could cloud associations in this and other studies.

In summary, about half of the longitudinal studies show a significant positive result but the effect appears small. Problems remain in assessing the independent effect of SSD, due to potential confounding from other diet and lifestyle factors.

Intervention studies

Randomised intervention trials constitute the highest level of evidence for a hypothesis, but these are relatively few (Table 5). Of the four interventions identified, only two were strictly of SSD[56,57]. The first was a pilot study, which randomly assigned 103 adolescents aged 13–18 years who regularly consumed SSD to either intervention or control groups. The intervention group received home delivery of non-energy beverages for 6 months, during which they decreased consumption of SSD by 82 %, while controls did not change. This resulted in a net difference in BMI between groups of $-0{\cdot}14$ kg/m^2 (NS). However, baseline BMI was a significant modifier and among those children who were in the highest third of BMI at baseline the net effect on BMI was $-0{\cdot}75$ kg/m^2 ($P = 0{\cdot}03$). The authors commented that the greater weight loss with increasing baseline BMI was not simply because of a greater decrease in energy intake from SSD and speculated that some individuals are inherently more susceptible than others to the adverse effects of SSD. The more recent and larger, school-based study of 1140 Brazilian children aged 9–12 years achieved only a small reduction in consumption and produced no difference in weight gain between the intervention and control groups over one school year. However, there was a small effect in those who were already overweight at baseline (about 0·2 kg/m^2), which was statistically significant among girls ($P = 0{\cdot}009$)[57].

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In England, a randomised cluster intervention by James *et al.* in six primary schools to reduce consumption of fizzy drinks via education sessions was associated with a 7·7 % difference in the prevalence of obesity between the intervention and control groups at 1 year[9]. However, there was no change in mean BMI overall and children in the intervention group still gained weight despite reducing their consumption of fizzy drinks. In a critique of this study, French *et al.* suggested that methodological limitations leave unanswered questions[58]. Only about half of the children returned the beverage diaries and no information was given on other drinks or food or on the reliability or validity of the beverage data. Moreover, this study focused on carbonated drinks exclusively. It is perhaps surprising that such a non-intensive five-session intervention achieved any impact at all, and less surprising that at the 2-year follow-up there was no residual difference between the groups[59].

Finally, the shortest-duration study meeting the present review's criteria was a 10-week parallel trial investigating the impact of a diet very high in sucrose (3·4 MJ supplemental energy per d) on *ad libitum* food intake and weight gain in overweight adults, compared with controls given artificially sweetened foods and drinks. The supplementation was achieved 60 % through sugar-sweetened beverages[60]. Energy intake in the sucrose group increased by 1·6 MJ (i.e. about 50 % compensation was observed) and body weight increased in the sucrose group (by 1·6 kg) and fell in the sweetener group (by 1·0 kg) (difference between groups $P < 0{\cdot}001$). Although this clearly shows the potential of excess energy intake from sugar to result in weight gain, actual weight gain was half that predicted from intake. Furthermore, the level of sucrose used in the intervention was extreme (28 % of energy or approximately twice normal adult intake) and therefore the generalisability of these results is questionable.

Conclusions

Despite the large number of studies on this topic, the inconsistencies of definition, design, statistical treatment and interpretation make it difficult to draw definitive conclusions as to whether sugar-sweetened beverages are significantly implicated in weight gain. Particular areas of weakness in the evidence base are highlighted below:

(1) Insufficient long-term interventions;
(2) Differing definitions of SSD and terminology;
(3) Differing units for serving size and frequency;
(4) Unreliable methods for dietary assessment;
(5) Narrow focus on SSD with inadequate assessment of other diet components or energy;
(6) Weight and height self-reported, not measured;
(7) Poor or no measurement of physical activity;
(8) Inadequate exploration of confounders or effect modifiers in analysis (for example, baseline BMI, ethnicity, baseline diet, misreporting);
(9) Inconsistent evidence between subgroups;
(10) Underpowered studies, no conclusions can be drawn;
(11) Possibility of publication bias towards positive studies.

Cross-sectional studies were the most numerous but have the weakest design in the conventional hierarchy and are prone to confounding and reverse causality. However, they can offer advantages, such as more reliable methods, large sample size and generalisability and, if adjusted for covariates and confounders, may yield results consistent with cohort studies. For example, three longitudinal studies yielded positive associations of similar magnitude when analysed cross-sectionally[14–16] and twelve out of twenty-seven cross-sectional studies in the present review found a positive association in at least one group.

Longitudinal studies are more powerful than cross-sectional studies, being able to relate change in weight to dietary factors, and often to diet change (change on change). However, they are also prone to confounding from concurrent changes in other aspects of diet and lifestyle and can suffer from attrition bias (drop-outs). Less than half the longitudinal studies reviewed (eight out of seventeen) showed a significant positive effect in at least one subgroup, and four of these had non-significant or null effects in other subgroups. Six studies showed no association in any group. The largest effects tended to be seen in the smaller studies, while larger cohort studies had much more modest effects.

Intervention studies provide the strongest form of evidence and have the potential to infer causality. However, they are also the most costly to conduct. In practice it is often difficult to ensure comparability of groups at baseline, compliance in the intervention group, non-contamination of the control group and adequate monitoring of diet and lifestyle during the trial. Three studies in the present review provide limited evidence that avoidance of sugar-containing soft drinks or substitution with other lower-energy beverages may help prevent further weight gain in overweight individuals[56,57,60]. By contrast, little effect was observed in normal-weight individuals. This may be partly explained by lack of power due to small sample size, but also to the small reductions in SSD achieved. Substitution effects (for example, replacement of SSD with fruit juice) may also be a factor in explaining the disappointing outcomes but many studies did not adequately assess consumption of other food and drink.

Previous reviews vary in their conclusions as a result of different inclusion criteria and classification methods. Half regard the evidence as less than persuasive; indeed the total weight of evidence can only be considered in favour of the hypothesis if non-significant studies are counted as positive. At the same time it must be acknowledged that 'absence of evidence is not evidence of absence'[61] and there are methodological reasons that may weaken the ability of studies to demonstrate an effect. In particular, low sample size and measurement error increase the likelihood of type II error (failure to detect an effect where one exists). Dietary methods need to be able to quantify consumption over several days if individual records are to be used.

Variation between studies in how confounding factors are treated also weakens the ability to compare results. Statistical approaches that separate 'energy from soft drinks' and 'energy from other sources' may provide more insights than simple adjustment for total energy in regressions. A surprising number of studies did not

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distinguish between diet and regular versions of soft drinks and thus potentially overestimated positive effects (diet versions being more strongly associated with excess weight). Spurious positive associations could also arise if SSD consumption is correlated with sedentary behaviours, while the reverse could occur if active people consumed more soft drinks. Thus assessment of energy intake and preferably of energy expenditure or physical activity is important in defining the mediators of weight change. It may also help to assess or adjust for confounding resulting from under-reporting.

Finally, there is the issue of publication bias, which may be in either direction. It has been suggested that industry-funded studies tend to show smaller effects[6], but these are comparatively few. On the other hand it is well recognised that publication bias normally works in the opposite direction (null studies remaining unpublished), which results in overestimation of positive effects[7,62].

In conclusion, SSD are by nature a source of energy but there is little evidence from epidemiological studies that they are more obesogenic than any other source of energy. Assertions that SSD are a disproportionate cause of excess body weight and/or that their avoidance would be effective in preventing weight gain are, in my opinion, not well substantiated by the science. The totality of evidence is dominated by American studies that may be less applicable to the European context where consumption is substantially lower and composition or formulation may differ (high-fructose corn syrup *v.* sucrose, proportion of diet *v.* non-diet, etc). Most studies suggest that the effect of SSD on body weight is small except in susceptible individuals or at high levels of intake. Thus effects quoted in terms of a 12 oz serving size daily or per MJ of energy exceed those achieved in practice. Moreover, methodological weaknesses mean that many studies cannot detect whether SSD or other aspects of diet and lifestyle have contributed to excess body weight or weight change Meta-analysis provides a way to quantify effect sizes but this requires authors to provide sufficient statistical detail on outcomes and exposures, in subgroups as well as the total sample, and on the effects of adjusting for confounding variables. In particular, more intervention studies are required, especially among over-weight consumers of SSD, but these should use reliable measurements of diet and physical activity and have adequate length of follow-up. New trials are due to report soon; therefore ongoing review of this area is imperative. Since obesity is a complex issue whose aetiology involves genetics, diet and lifestyle, it is vital to maintain an integrated perspective on all the influences (and inter-actions) relevant to weight gain.

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