

Does an increased intake of added sugar affect appetite in overweight or obese adults, when compared with lower intakes? A systematic review of the literature

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Abstract

Changes in added sugar intake have been associated with corresponding changes in body weight. Potential mechanisms, particularly the impact of added sugar intake on appetite, warrant exploration. A systematic literature review of randomised controlled trials investigated the association between added sugar consumption and appetite in overweight and obese adults. A systematic search of Medline, Cochrane CENTRAL, Web of Science and CINAHL included studies that examined the relationship between added sugar intake and appetite markers, in comparison with a group with lower added sugar intake. A total of twenty-one articles describing nineteen studies were included in the review. The effect of added sugar on appetite was explored separately by reported comparisons of added sugar type and their effect to three study outcomes: energy consumption (n 20 comparisons); satiety (n 18); and appetite hormones, leptin (n 4) or ghrelin (n 7). Increased added sugar consumption did not impact subsequent energy intake (n 9), nor did it influence satiety (n 12) or ghrelin levels (n 4). Differences in the total daily energy intake were comparable with the differences in energy values of tested products (n 3). Added sugar intake was reported to increase leptin levels (n 3). This review did not find a consistent relationship between added sugar intake and appetite measures, which may be partially explained by variations in study methodologies. There is a need for randomised controlled trials examining a range of added sugar sources and doses on appetite in overweight and obese adults to better understand implications for weight gain.

Key words: Added sugar: Adults: Appetite: Overweight: Obesity

Overweight and obesity are global multi-factorial health epidemics⁽¹⁾ that are increasing in prevalence worldwide⁽²⁾. For example, in 2014–2015, 63% of Australian adults were either overweight (BMI 25.0–29.9 kg/m²) or obese (BMI \geq 30.0 kg/m²)^(3,4). Obesity is a known risk factor for many chronic diseases including CVD, type 2 diabetes, musculoskeletal disorders and cancer^(4,5).

The primary cause for overweight and obesity is a consistent positive imbalance between kJ consumed and energy expended⁽⁴⁾. A common dietary contributor is the replacement of nutrient-dense foods with energy-dense, nutrient-poor foods⁽⁶⁾, as seen in diets that are high in added sugar (AS) (>20% of total energy intake)⁽⁷⁾. AS includes sucrose, fructose, dextrose, lactose and sugar syrups such as glucose syrup⁽⁸⁾, which are introduced either during manufacturing or by the consumer during food preparation⁽⁸⁾.

A meta-analysis of randomised controlled trials and cohort studies reported a parallel relationship between AS consumption and a corresponding change in body weight under *ad libitum* conditions (gain of 0.8 kg when increasing AS or reduction of 0.75 kg when reducing AS), over an intervention period of 2 weeks or more⁽⁹⁾. One possible mechanism for

weight gain is the metabolism of AS. It has been noted that fructose, a major constituent of AS⁽¹⁰⁾, does not increase satiety when metabolised⁽¹¹⁾, which may lead to overconsumption and thereby, in part, explain the association between AS and weight gain.

To date, many studies that investigate the impact of AS consumption focus on the general population. However, it is known that in the overweight or obese population, a modest weight loss (\geq 5% initial body weight) reduces cardiovascular health risks associated with overweight and obesity⁽¹²⁾. This highlights a need to investigate dietetic strategies that may aid weight loss among overweight or obese individuals.

There is currently no known systematic literature review exploring the impact of AS consumption on appetite in overweight and obese individuals. The aim of this systematic review was to investigate whether increased AS consumption affects appetite in overweight or obese adults when compared with lower AS intakes. It is hypothesised that increased intakes of AS will affect appetite by reducing the feeling of satiety, resulting in an increased food intake.

Abbreviations: AS, added sugar; VAS, visual analogue scale.

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Methods

This systematic literature review followed the Preferred Reporting Items for Systematic Reviews and Meta-analyses statement⁽¹³⁾. The review was registered with PROSPERO, the international prospective register of systematic reviews (<http://www.crd.york.ac.uk/PROSPERO>; registration number: CRD42017057777).

Searches

A systematic search was conducted across four databases (all years to 7 April 2017): Medline, Cochrane CENTRAL, Web of Science and CINAHL. Search terms and truncations included ('overweight' OR 'obese*' OR 'obesity') AND ('added sugar*' OR 'sugar*' OR 'free sugar*' OR 'sucrose' OR 'refined sugar*' OR 'fructose' OR 'dextrose') AND ('appetite' OR 'hunger' OR 'food intake' OR 'satiety' OR 'satiat*' OR 'leptin' OR 'Ob protein' OR 'Ob gene' OR 'ghrelin' OR 'GHRL' OR 'Ppghrelin').

To be included in this review, studies were limited to randomised controlled trials and cohort studies, published in English. Studies were required to meet the following inclusion criteria: (1) conducted in overweight or obese human adults (BMI ≥ 25 kg/m²); (2) assess associations between oral AS intake and appetite, with reference to food intake (including food intake measured at a subsequent meal or total intake including the AS treatment), self-reported satiety through a visual analogue scale (VAS), or appetite hormones (leptin or ghrelin); (3) report AS intake in comparison with a comparator group of lower AS content. In addition, the following exclusion criteria were applied: (1) samples of pregnant or breast-feeding women, (2) published as conference abstracts only and (3) studies conducted in animals or children.

Article screening

One review author (K. T.) conducted the literature search and assessed potential studies for inclusion. Inclusion of articles not clearly meeting the inclusion or exclusion criteria was discussed with two additional review authors (E. N. and K. C.), until consensus was reached.

Articles were initially screened based on title and abstract. Full-text articles were retrieved if an abstract was unavailable or provided insufficient information to determine inclusion in this review. These were then assessed for eligibility using the inclusion criteria. Where multiple articles reported results from the same study, results were merged in the summary table, to avoid duplication of the study population.

Data extraction

The following data were extracted from each study: citation, details of the study population (sample size, age, sex, BMD), intervention duration, intervention details, including comparison group and measured outcomes of interest (Table 1).

Quality assessment

Study quality was assessed using the Academy of Nutrition and Dietetics Quality Criteria Checklist⁽¹⁴⁾ (online Supplementary material I). This checklist is a component of the *Evidence Analysis Manual* developed by the Academy of Nutrition and Dietetics to support systematic literature reviews in nutrition and dietetics. The checklist considers a number of aspects of study design that may impact on quality including participant selection, blinding, appropriateness of statistical analyses and risk of bias from funding sources. Studies were also classified according to the Australian National Health and Medical Research Council (NHMRC) level of evidence ranking⁽¹⁵⁾.

Results

A total of 2557 articles were identified using the search parameters. After removal of duplicates, articles were assessed for eligibility (n 1724). Following application of the inclusion and exclusion criteria, twenty-one articles describing nineteen studies were included in this review (Fig. 1). A total of two articles^(16,17) reported on a subgroup from a study already included in the review⁽¹⁸⁾ and were therefore combined in the summary table.

A review of each article according to the quality criteria checklist⁽¹³⁾ rated the quality of nineteen of the twenty-one studies as positive. A total of two studies were rated neutral as participant selection was not described^(19,20) (online Supplementary material I). Based on the NHMRC level of evidence⁽¹⁵⁾, all except three studies^(21–23) were randomised controlled trials (level II). A total of two studies^(21,22) did not state whether group allocation was randomised and, therefore, were considered to be pseudo-randomised controlled trials (level III-1). While prospective cohort study designs were also considered for this review, no cohort studies met the overall inclusion criteria.

Included studies evaluated the effects of AS consumption through food (n 5), beverages (n 12) or a combination of foods and beverages (n 2) over a period of time ranging from 1 d^(20,24–32) to 6 months⁽³³⁾. Characteristics of included studies are displayed in Table 1. Participants' mean BMI ranged from 26.1⁽³⁰⁾ to 41.1 kg/m²⁽³¹⁾ and the mean age ranged from 22⁽³⁴⁾ to 57 years⁽²⁷⁾. A total of three studies analysed only males^(19,27,28), eight only females^(22–24,30–32,35,36) and the remainder involved both sexes^(16–18,20,21,25,26,29,33,34).

Studies evaluated energy consumption either through the amount of energy consumed post-treatment at a subsequent meal^(20,25–32) or as total daily energy intake, including test products^(16,17,19,22,23,33–35). Consumption of AS was reported either as prescribed doses, ranging from approximately 10⁽³²⁾ to 125 g⁽¹⁹⁾, or as a set portion of dietary energy, with individual intakes varying^(16–18,21,33). A total of four studies^(21,26–28) reported more than one type of AS source, therefore all subsequent results are presented separately by comparisons between AS type. For example, one study explored the effect of both glucose and lactose on appetite⁽²⁸⁾, which are examined separately in this review. A range of AS sources were reported, including sucrose^(16–18,20,22,23,31,32,36) (n 7 comparisons),



Table 1. Summary table of studies included in the systematic literature review on added sugar and appetite

Citation	NHMRC level of evidence, AND study quality	Intervention duration, sample size (for review)	BMI (kg/m ²), age (years), sex	Sugar type	Comparison group	Medium used	Effect: energy intake	Effect: satiety (visual analogue scale)	Effect: appetite hormone (leptin and ghrelin)
Bowen <i>et al.</i> (2006) ⁽²⁸⁾	II, P8	4 d (4 × 1 d), 19	32.1 (SEM 3.7), 53.3 (SEM 6.1), M	1. Glucose (56 g, 1025 kJ) 2. Lactose (56 g, 1025 kJ)	1. Whey (55 g, 1069 kJ) 2. Casein (55 g, 1090 kJ)	Beverage*	Highest (493 kJ, $P < 0.05$) after glucose (buffet, excluding treatments). No differences ($P > 0.05$) between lactose, whey and casein	Highest ($P < 0.05$) overall appetite after glucose (120–180 min). No differences ($P > 0.05$) in independent hunger or satiation measures	Ghrelin: all declined similarly (0–60 min). In the following rise, glucose became higher ($P < 0.05$) than lactose, whey and casein (120–180 min)
Bowen <i>et al.</i> (2007) ⁽²⁷⁾	II, P10	4 d (4 × 1 d), 28	32.5 (SEM 0.6), 57 (SEM 1.6), M	1. Fructose (65 g, 1097 kJ) 2. Glucose (65 g, 1097 kJ)	1. Whey (55 g, 1147 kJ) 2. Whey (27 g) and fructose (33 g, 1122 kJ)	Beverage (400 ml)	No differences ($P > 0.05$) (buffet, excluding treatments)	Lower fullness ($P < 0.05$) after fructose compared with whey and glucose (90–240 min)	Ghrelin: all declined similarly (0–60 min). In the following rise, glucose became higher ($P < 0.05$) than whey (180–240 min). No differences ($P > 0.05$) between fructose and whey
Dove <i>et al.</i> (2009) ⁽²⁵⁾	II, P9	2 d (2 × 1 d), 34	32.4 (SEM 3.4), 55.1 (SEM 12.5), M (13) F (21)	Fruit drink (63 g sugar, 1062 kJ)	Skimmed milk (36 g lactose, 1062 kJ)	Beverage (600 ml)	Highest after fruit drink (226 kJ, $P < 0.05$) (sandwich platter, excluding treatments)	Lowest satiety ($P < 0.05$) after fruit drink (240 min)	NR
Drewnowski <i>et al.</i> (1994) ⁽³¹⁾	II, P8	4 d (4 × 1 d), 12†	41.1 (SEM 6.2), 34.4 (SEM 7.6), F	Sucrose (50 g, 2929 kJ)	1. Maltodextrin and aspartame (2929 kJ) 2. Aspartame (1255 kJ) 3. Water (1255 kJ)	Food: white cheese (400 g)	No differences ($P > 0.05$) (buffet, excluding treatments)	‡No differences ($P > 0.05$) between sucrose and maltodextrin/aspartame. Lower hunger ($P < 0.05$) after sucrose compared with aspartame and water (90–120 min)	NR
Furchner-Evanson <i>et al.</i> (2010) ⁽³⁰⁾	II, P9	4 d (4 × 1 d), 19	26.1 (SEM 0.8), 39.2 (SEM 0.7), F	1. Dried plum (38 g sugar, 996 kJ (238 kcal)) 2. Low-fat cookie (33 g sugar, 996 kJ (238 kcal))	1. White bread (6 g sugar, 996 kJ (238 kcal)) 2. Water	Food*	No differences ($P > 0.05$) between bread, plum and cookie (yogurt and granola, excluding treatments)	No differences ($P > 0.05$) between white bread, dried plum or cookie	Ghrelin: all (except water) reduced (15–60 min), with no differences ($P > 0.05$) in the following rise (120 min)
Hollis <i>et al.</i> (2009) ⁽³⁴⁾	II, P10	12 weeks, 76	Range: 25.0–29.9, 18–50, M, F§	Grape drink (82 g sugar, 1464 kJ)	No-treatment	Beverage (480 ml/d)	No differences ($P > 0.05$) (3 d food record, including treatments)	No differences ($P > 0.05$)	NR
Kasim-Karakas <i>et al.</i> (2009) ⁽³⁵⁾	II, P10	2 months, 24	I: 35.4 (SEM 1.8) C: 38.9 (SEM 1.6) Age: 28 (SEM 3), F	Simple sugar (glucose and maltose, 1004 kJ (240 kcal))	Whey protein isolate (1004 kJ (240 kcal))	Beverage*	Energy restriction prescribed. No differences ($P > 0.05$) (7 d food record, including treatments)	NR	Leptin: no differences ($P > 0.05$)
Kasim-Karakas <i>et al.</i> (2007) ⁽²⁴⁾	II, P9	2 d (2 × 1 d), 28	35.9 (SEM 1.2), 26 (SEM 2), F	Glucose (75 g)	Whey protein isolate (75 g)	Beverage*	NR	NR	Ghrelin: all declined similarly (60 min). In the following rise, glucose became higher ($P < 0.05$) than protein (180–300 min)
Maersk <i>et al.</i> (2012) ⁽²⁹⁾	II, P8	4 d (4 × 1 d), 24	31.4 (SEM 3.1), 33.5 (SEM 9.2), M (12) F (12)	Cola (53 g sugar, 900 kJ)	1. Milk (23.5 g CHO, 950 kJ) 2. Diet cola (aspartame-sweetened, 7.5 kJ) 3. Water	Beverage (500 ml)	No differences ($P > 0.05$) (buffet, excluding treatments)	No differences ($P > 0.05$) between cola, water or diet cola	Ghrelin: all (except water) declined (0–60 min). No differences in following rise ($P > 0.05$) (180–240 min)

Table 1. Continued

Citation	NHMRC level of evidence, AND study quality	Intervention duration, sample size (for review)	BMI (kg/m ²), age (years), sex	Sugar type	Comparison group	Medium used	Effect: energy intake	Effect: satiety (visual analogue scale)	Effect: appetite hormone (leptin and ghrelin)
Mazlan <i>et al.</i> (2006) ⁽¹⁹⁾	II, O8	21 d (3 × 7 d), 6†	27.7 (SEM 1.6), 46.7 (SEM 10.8), M	1. One tub (62.63 g sugar II, 1.5 MJ) 2. Two tubs (125.26 g sugar II, 3 MJ)	No parfait	Food: parfait (275 g/tub)	Increased ($P < 0.001$) from 0 to 3 MJ (2.1 MJ trials (7-d food record, including treatments)	No differences ($P > 0.05$)	NR
Overduin <i>et al.</i> (2016) ⁽²⁰⁾	II, O8	3 d (3 × 1 d), 10†	34.2 (SEM 3.6), 34.6 (range: 24.9–46.7), M (5) F (5)	Sucrose (39.5 g, 2000 kJ)	1. Erythritol (1471 kJ, 7.9 g sucrose) 2. Erythritol (2000 kJ, 10.7 g sucrose)	Food: semi-solid custard	No differences ($P > 0.05$) (buffet, excluding treatments)	No differences ($P > 0.05$)	NR
(a) Raben <i>et al.</i> (2002) ⁽¹⁸⁾ AND (b) Raben <i>et al.</i> (2011) ⁽¹⁶⁾ , AND (c) Sørensen <i>et al.</i> (2014) ⁽¹⁷⁾	(a) II, P10 (b) II, P9 (c) II, P10	(a) 10 weeks, 41 (b) 10 weeks, 23 (c) 1 d at week 10, 22‡	(a) M (6) F (35) I: 28.0 (SEM 0.5), 33.3 (SEM 2.0) C: 27.6 (SEM 0.5), 37.1 (SEM 2.2) (b) M (4) F (19) I: 28.7 (SEM 0.7), 35.3 (SEM 2.8) C: 27.6 (SEM 0.8), 35.5 (SEM 3.6) (c) M (4) F (18) I: 28.7 (SEM 2.3), 35.3 (SEM 9.1) C: 27.3 (SEM 2.5), 35.2 (SEM 12.4)	Sucrose (2 g/kg body weight)	Sweetener (2 g/kg body weight)	Food (20%) and beverage (80%)*	(b) Sucrose increased intake over 10 weeks (1.6 MJ/d, $P < 0.01$) (7 d food record, includes treatments) (c) Highest (3.3 MJ, $P < 0.05$) by sucrose (<i>ad libitum</i> diet in chamber, includes treatments)	(a) No differences ($P > 0.05$). (c) Sucrose was less full ($P < 0.05$) with higher ratings of prospective food consumption ($P < 0.05$) in the periods after lunch and dinner	(b) Fasting leptin: higher ($P < 0.01$) levels in sucrose compared with control (week 10). Responses consistent with changes in body weight
Reid <i>et al.</i> (2014) ⁽²³⁾	III-1, P10	4 weeks, 41	I: 32.9 (SEM 1.8), 35.1 (SEM 9.9), F C: 32.7 (SEM 2.2), 34.6 (SEM 8.5), F	Sucrose (105 g, 1800 kJ)	Aspartame (170 kJ)	Beverage (1 litre/d)	No differences ($P > 0.05$) (7 d food record, includes treatments)	No differences ($P > 0.05$)	NR
Reid <i>et al.</i> (2010) ⁽²²⁾	III-1**, P9	4 weeks, 53	I: 27.2 (SEM 2.1), 34.5 (SEM 11.0), F C: 27.8 (SEM 1.8), 32.9 (SEM 8.8), F	Sucrose (105 g, 1800 kJ)	Aspartame (170 kJ)	Beverage (1 litre/d)	No differences ($P > 0.05$) (7 d food record, includes treatments)	NR	NR
Rezvani <i>et al.</i> (2013) ⁽²¹⁾	III-1**, P10	10 weeks, 32	M (16) F (16) Glucose: M: 29.3 (SEM 1.1), 54 (SEM 3) F: 29.4 (SEM 1.3), 56 (SEM 2) Fructose: M: 28.4 (SEM 0.7), 52 (SEM 4) F: 30.3 (SEM 1.0), 53 (SEM 2)	1. Glucose (25% EER, 30% EER complex CHO) 2. Fructose (25% EER, 30% EER complex CHO)	No-beverage, complex CHO baseline (55% EER)	Beverage (25% EER)*	NR	NR	Fasting leptin: higher levels ($P < 0.05$) in fructose and glucose (10 weeks) compared with baseline. Responses consistent with changes in body weight
Saris <i>et al.</i> (2000) ⁽³³⁾	II, P9	6 months, 316	30.4 (SEM 2.7), 39 (SEM 9), M (155) F (161)	Fat-reduced, high-simple CHO diet (26% EEI simple CHO)	1. Complex CHO diet (17% EEI simple CHO) 2. Habitual diet (20% EEI simple CHO)	All food and beverage*	No differences ($P > 0.05$) between high simple CHO and habitual diet (7 d weighed food record, includes CHO)	NR	NR
Sunwit <i>et al.</i> (1997) ⁽³⁶⁾	II, P9	6 weeks, 42	I: 35.9 (SEM 4.8), 40.6 (SEM 8.2), F	Low-fat, high-sucrose diet (121 g sucrose)	Low-fat, low-sucrose diet (6 g sucrose)	Food*	NR	No differences ($P > 0.05$)	NR

Effect of added sugar intake on appetite

Table 1. Continued

Citation	NHMRC level of evidence, AND study quality	Intervention duration, sample size (for review)	BMI (kg/m ²), age (years), sex	Sugar type	Comparison group	Medium used	Effect: energy intake	Effect: satiety (visual analogue scale)	Effect: appetite hormone (leptin and ghrelin)
Vozzo <i>et al.</i> (2002) ⁽²⁶⁾	II, P9	3 d (3 × 1 d), 20	C: 34.9 (SEM 4.4), 40.3 (SEM 7.3), F Range: 25.3–37.7, 44–71, M (12) F (8)	1. Glucose (75 g) 2. Fructose (75 g)	Flavouring with water	Beverage (300 ml)	Lowest ($P < 0.05$) after glucose (–908 kJ) and fructose (–901 kJ) (buffet, excluding treatments)	Highest fullness ($P < 0.05$) and lowest hunger ($P < 0.05$) after glucose and fructose (30–60 min)	NR
Wissing <i>et al.</i> (2015) ⁽²⁶⁾	II, P10	6 d (6 × 1 d), 55	27.4 (SEM 2.4), 29 (SEM 8), F	1. Sucrose (10 g, 157 kJ) 2. Sucrose (20 g, 314 kJ) 3. Sucrose (50 g, 785 kJ)	1. Water (8 kJ) 2. Protein (10 g, 178 kJ) 3. Protein (20 g, 348 kJ)	Beverage (500 ml)	No differences ($P > 0.05$) (buffet, excluding treatments)	No differences ($P > 0.05$)	NR

NHMRC, National Health and Medical Research Council; AND, Academy of Nutrition and Dietetics; M, male; F, female; NR, not reported; I, intervention; C, control; CHO, carbohydrate; EER, estimated energy requirement; EEI, estimated energy intake.

* Exact volume of test beverage or food was not reported.

† Study included another intervention group which was not relevant to this review, only the relevant group was included in the summary table and review.

‡ Reports on hunger, satiety and desire to eat were pooled with lean individuals due to lack of difference in responses.

§ Number of males and females was not specified in the research paper.

|| Sugar content estimated from percentage of energy reported in test product.

¶ One subject excluded due to not completing the second respiratory chamber stay.

** Articles where authors did not specify whether allocation was randomised, studies were allocated to III-1.

glucose^(21,24,26–28,35) (n 6), fructose^(21,26,27) (n 3), lactose⁽²⁸⁾ (n 1), fruit drink^(25,34) (n 2), cola⁽²⁹⁾ (n 1), parfait⁽¹⁹⁾ (n 1), dried plum⁽³⁰⁾ (n 1) and cookies⁽³⁰⁾ (n 1). In all, one study examined a whole-of-diet approach, reporting participants' total intake of AS from a range of sources⁽³³⁾. The effect of AS on appetite was explored through three study outcomes: energy consumption^(16–20,22,23,25–35) (n 16), satiety^(17–20,23,25–32,34,36) (n 14) and appetite hormones (leptin^(16,21,35) and ghrelin^(24,27–30)) (n 8).

Following AS consumption, significant reductions in subsequent energy intake, compared with controls, were reported in two comparisons⁽²⁶⁾, nine found no difference in energy intake^(20,27–32) and two reported a significant increase in energy intake^(25,28). When total daily energy intake was examined, including the AS source, four comparisons found no difference in energy intake^(22,23,34,35) and three reported a significant increase in energy intake^(16,17,19,33). Consumption of AS significantly increased satiety in two comparisons⁽²⁶⁾, whilst twelve found no differences in reported satiety^(19,20,23,27–32,34,36) and four reported significantly reduced satiety^(17,25,27,28). In response to AS consumption, one comparison reported no change in leptin levels⁽³⁵⁾, whereas three reported significantly increased leptin levels^(16,21). Studies exploring the impact of AS on ghrelin reported an immediate drop in ghrelin levels (measured at 60 min post AS consumption), followed by a later rise (120–180 min)^(24,27–30). Findings related to this rise in ghrelin levels varied, with four comparisons finding no difference between AS and control at 120^(28,30)–180 min^(27,29), whilst three reported a significantly higher ghrelin measure after AS consumption, compared with the control group at 120⁽²⁸⁾–180 min^(24,27).

Discussion

This systematic review found inconsistent associations between AS intake and appetite in overweight and obese adults. Measures of appetite were examined through *ad libitum* energy intake, satiety (VAS) and appetite hormones (leptin and ghrelin). These measures have previously shown good intra-individual reproducibility and validity as measures of appetite^(37–40).

Changes in *ad libitum* energy consumption in response to AS intake were examined in twenty comparisons. A total of thirteen comparisons^(20,25–32) examined single-day influences of AS at a following meal. Over half (n 9) of these comparisons found that AS consumption had no influence on subsequent energy intake^(20,27–32). The lack of change in energy intake after AS consumption, despite differences in energy content of the pre-load^(20,27–29,31,32), aligns with previous findings of an incomplete compensation of energy intake following AS consumption in studies of both short and longer durations^(41,42). A total of three long-duration studies (7 d to 6 months) reported an increased total daily energy intake when AS was compared with a control of lower energy value^(16,17,19,33). A 2-month study⁽³⁵⁾ reported no differences in total daily energy intake when AS was compared with an isoenergetic control. In all, two single-day comparisons reported a reduced energy intake following AS ingestion⁽²⁶⁾. However, when accounting for differences in energy values between the AS and control products, these

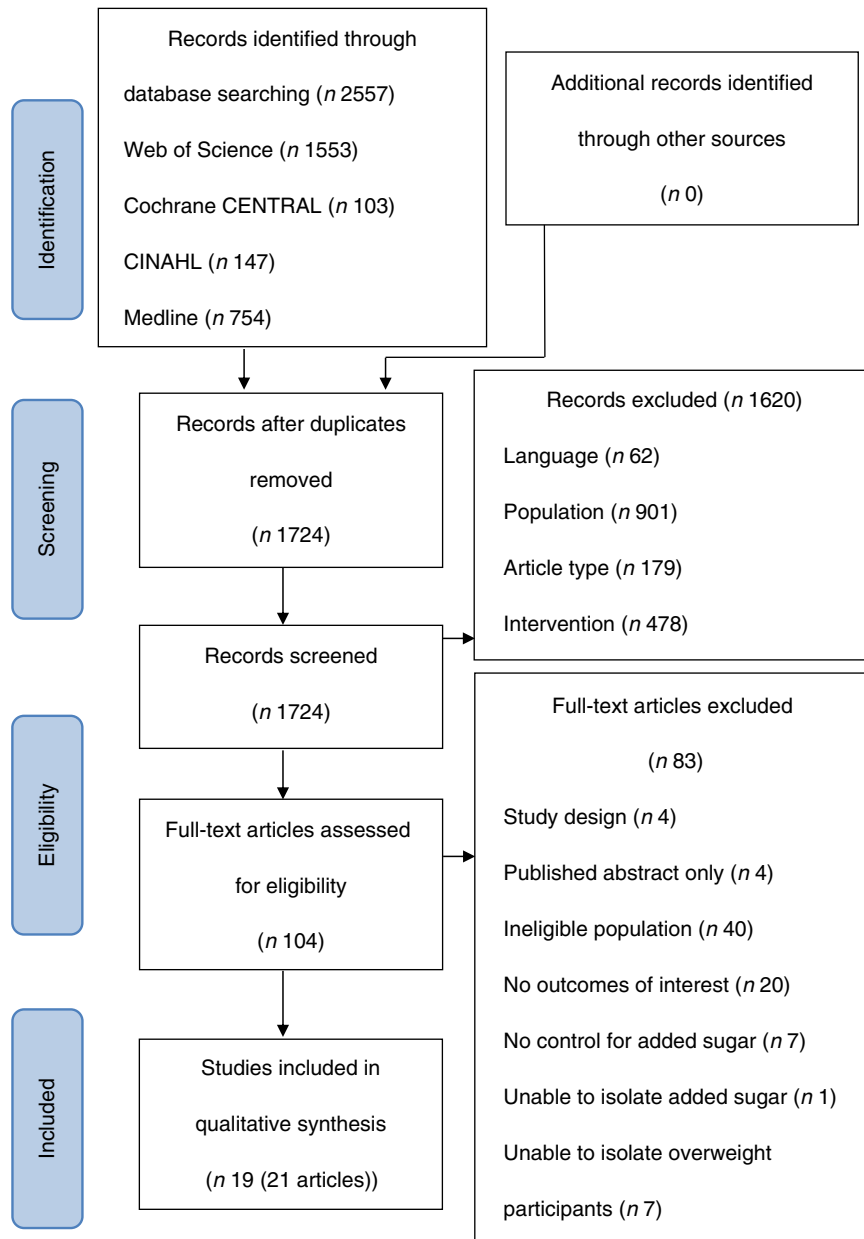


Fig. 1. Preferred Reporting Items for Systematic Reviews and Meta-analyses flow diagram⁽¹³⁾ of the number of studies extracted for review.

analyses found that the significant reduction in energy intake at the following meal only equated to 40% of the energy provided by the AS beverage, consequently resulting in a higher total energy intake across the day⁽²⁶⁾. Despite differing findings, most comparisons^(16,17,19,20,26–29,31–33,35) suggested that if energy intake over the day did not compensate for that provided by the AS, total daily energy intake would increase⁽⁴³⁾. This could explain the relationship between AS and body weight reported in *ad libitum* diets⁽⁹⁾.

Energy intake can be influenced by a diverse array of environmental, cultural, behavioural and economic factors^(4,44). Therefore, when analysing appetite, energy intake is most reliable when combined with another appetite measure such as a VAS⁽⁴⁵⁾. In this review, changes in VAS responses were similar to changes in reported energy consumption ($n\ 14$)^(17,20,23,25–32,34).

Only three comparisons^(18,19,27) reported inconsistent findings between energy intake and the VAS scores.

A single-day study⁽²⁷⁾ reported that fructose consumption resulted in lower feelings of fullness (satiety) than was observed following consumption of a whey-control beverage of similar energy content. This finding may be explained by an incomplete fructose digestion at high fructose doses⁽⁴⁶⁾. However, these findings need to be further examined. Two longer duration comparisons ($\geq 7\ d$)^(16,18,19) reported that AS consumption increased energy intake with no associated change in satiety. This inconsistency could be explained by an identified sensitivity issue of the VAS in longer-term studies⁽³⁷⁾. Despite the majority of comparisons between energy intake and VAS being consistent, the three identified discrepancies highlight the need to incorporate objective appetite measures, such as appetite

hormones, when examining the relationship between AS and appetite⁽³⁷⁾.

Leptin is a hormonal response to food intake, thereby providing a physiological objective measure of appetite. Leptin, produced by adipose cells, inhibits hunger signals in the central nervous system, ensuring long-term regulation of energy balance⁽⁴⁰⁾. A total of three comparisons found a significant increase in leptin levels after 10 weeks of AS consumption^(16,21), whereas one shorter duration comparison (2 months) found no difference in leptin levels in response to AS intake⁽³⁵⁾. The differing leptin findings could be explained by a positive relationship between leptin concentrations and body fat stores⁽⁴⁷⁾. Studies in this review identified that after adjusting for changes in body weight, the relationship between leptin levels and AS consumption was no longer significant^(16,21). This explains the inconsistency in responses to AS intake in findings of self-reported satiety, energy intake and leptin levels when body weight was not accounted for.

Unlike leptin, ghrelin is associated with hunger ratings in individuals of all weight categories⁽⁴⁰⁾. Research suggests that ghrelin acts as a physiological meal initiator through a preprandial rise and postprandial fall of plasma ghrelin levels⁽⁴⁸⁾. This ghrelin response aligns with all seven ghrelin comparisons of AS in this review^(24,27–30), with AS consumption resulting in an immediate drop in ghrelin levels (60 min), followed by a later rise (120–180 min)^(24,27–30). Findings relating to the rise differed between studies. Only three comparisons^(24,27,28) reported a significantly higher final ghrelin measure after AS consumption, compared with the control. Each used glucose as the AS source, whereas the four comparisons that had no significant ghrelin response used lactose, fructose, dried plum, cookies or cola as the source of AS. These results may indicate that different sources of AS are digested differently, contradicting current research that compared AS sources and their influence on ghrelin^(49,50). It should be noted that intakes of AS tended to be higher in the comparisons that reached significance (56⁽²⁸⁾–75 g⁽²⁴⁾) compared with those that did not (33⁽³⁰⁾–65 g⁽²⁷⁾). These observations could suggest that ghrelin may respond to carbohydrate intake in a dose-dependent manner, as previously reported^(50,51). Inconsistency in ghrelin findings indicates that this area requires further research.

Although the present review followed a systematic process to provide an insight into the effect of AS intake on appetite in overweight or obese adults, when compared with lower intakes, there are some limitations. The results are limited by the substantial variation between studies, including the large range in doses, duration, control comparator and type of AS, possibly explaining the inconsistent results and impeding the ability to establish a dose effect. As a result of the variability between studies, it was not considered appropriate to pool the results in a meta-analysis. Many studies included in this review did not compare AS with an isoenergetic control^(19,20,22,23,26–29,31–34,36), which may have confounded results, as suggested previously^(9,43). The variety of mediums included could have influenced appetite, with liquid meals previously reported to be less satiating than solid meals, independent of energy density^(52,53). Similarly, the variation in study duration, which

included both acute and longer duration studies, may have resulted in some of the inconsistencies observed.

Conclusion

This review did not find a consistent relationship between AS intake and appetite measures, which may be partially explained by variation in study methodologies. The inconsistent results highlight a need for further randomised controlled trials that explore the impact of differing types of AS sources (including those replicating real-life consumption of AS) and different doses of AS on appetite in overweight and obese adults, to assist with targeting dietary messages for weight management.

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Supplementary material

For supplementary material/s referred to in this article, please visit <https://doi.org/10.1017/S0007114518003239>

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